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Annex 14

Summary profiles of chemicals with information on use, production, emission, monitoring and legal status

Annex 14 : Summary profiles of chemicals with information on use, production, Emission, monitoring and legal status

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Summary

High concern
Chlordane, chlordecone, mirex, triphenyltin and toxaphene are used as fungicides and moluscicides on food crops and as insecticides (e.g. ants), are persistent and bioaccumulative and found in biota (fish=food) and human mother milk. Exposure through food is very likely because the substances are persistent and found in food. Toxaphene is already forbidden (as plant protection product) to be used in the EU but is found to be transported by long boundary transport through air and is still found in biota and mother milk. Chlordane is also forbidden in the EU and the USA but still found in mother milk and wild life biota. These substances are prioritised as high concern.

Lindane and HCB are fungicides and insecticides used on seed and soil before culturing although HCB also may be used on food crops. However in the EU HCB is severely restricted. HCB is persistent and lindane inherently biodegradable. Both substances bioaccumulate. Both substances are found widely in the environment and in fish (food) and mother milk. Lindane is also found in human tissues. Because this indicates exposure through food and mother milk, both substances are prioritised as having high concern.

Linuron is used on food crops. Human exposure may be expected by food but because these substances are not persistent, exposure is less likely. The substance is not bioaccumulative. The substance is prioritised as having high concern for exposure.
It should be checked whether there is indeed no or hardly any exposure.

Acetochlor and alachlor are herbicides and fungicides used on food crops. Human exposure may be expected by food but because these substances are not persistent, exposure is less likely. All these substances are not bioaccumulative. Only alachlor is measured in the environment (in water systems). These substances are prioritised as high concern.

Maneb, thiram, metam natrium and zineb are herbicide and fungicides used on food crops. Metam natrium is also used as a fungicide used on soil before culturing. Human exposure may be expected by food but because these substances are metabolised quickly to metabolites (ETU and MITC), exposure is less likely. All these substances are not bioaccumulative. Of maneb, metam natrium, thiram and zineb the metabolites ETU and MITC are measured in the environment (water systems). These substances are prioritised as high concern.

Vinclozolin is a fungicide used on food crops. Human exposure may be expected by food because vinclozolin is inherently biodegradable. Vinclozolin is not bioaccumulative. Vinclozolin is measured in the environment (in water systems). This substance is prioritised as high concern.

Atrazine is a herbicide used alongside roads and also used on food crops. Exposure is expected though food and direct exposure by contact of playing children with soil and plants alongside roads. Atrazine is persistent and not bioaccumulative and is found in the environment primarily in water systems. Atrazine is prioritised as having high concern.

Tri-n-propyltin is prioritised as high concern because practically no information is available.

Tributyltin is used as molluscicides in antifouling paints and as wood preservatives, disinfectants and biocides for cooling systems. Exposure may occur indirectly to accumulation in food (fish). Tributyltin is persistent and highly bioaccumulative and therefore presents high concern for wild life. TBT is prioritised as high concern.

BBP, DBP and DEHP are used as plasticizer and softeners in e.g. toys and packaging material (for food). DBP and BBP are also used in cosmetics and carpet backings. DBP is furthermore used in dental impression materials and DEHP in tubes and bags used for blood transfusion. Exposure to children is expected through toys and food (substance leaching from packaging material). Other

exposure may occur through dental fillings and through blood transfusion by which another vulnerable group (patients) are exposed. All substances do bioaccumulate. BBP is not persistent, DBP low to medium persistent and DEHP is persistent. All substances are found in the environment primarily in water systems and wild life biota. Based on their exposure thses substances are prioritised as high concern.

Bisphenol A is used as a resin in dental fillings to coat teeth especially childrens teeth and in packaging material (coating in food cans from which it has been proven to leach). Exposure occurs through food and dental fillings, which may be especially for children. The substance is persistent but does not bioaccumulate and is found primarily in water systems. The substance is classified as having high concern for exposure.

Styrene is used in closed systems in paints, paper, pulp, as polymer in polystyrene for hobbies, crafts and toys and as packaging material in food containers (from which it has been shown to leach). It is also used as a flavoring agent. Exposure may occur primarily through food (flavoring, packaging) and toys. Although styrene is readily biodegradable and does not bioaccumulate based on the possible exposure and because it is found in food and water, styrene is prioritised as high concern. Because there no recent information it may be that the use of styrene may have changes so that the mentioned exposures may not occur any more. This has to be researched.

DDT is a insecticide used against sickness. Although it is forbidden in the EU, the USA and Japan, it is still used in some countries. Because it is very persistent, bioaccumulative and still widely found in the environment there is a high concern for exposure.

PCBs have been used in the past in electrical equipment but are not severely restricted and banned. PCBs are are still available through existing products, at the production site and the waste stage. PCBs are still found widely in the environment and are found in food (fish) and mother milk. PCBs are persistent and do bioaccumulate. Because of the exposure in food and mother milk PCBs are classified as having high concern of exposure.

PCDDs are formed during combustion of waste and during several industrial processes. Exposure is expected through emission at production and at waste stage. The substances are found in food (fish, meat, and dairy products) and mother milk. PCDDs are persistent and highly accumulating. The substances are classified as having high concern for exposure.

PBB are used as flame-retardants. Exposure is only expected through the production site, the waste stage and food (fish). PBBs are persistent and most are bioaccumulated and biomagnified. PBBs are found in biota (fish). The substances are classified as having high concern.

Resorcinol is used in the manufacture of dyes, pharmaceuticals, tanning and cosmetics. Exposure may occur through use as a pharmaceutical or cosmetic on skin and by (wood or cigarette) smoke inhalation. Resorcinol is readily biodegradable and not bioaccumulated. The substance is only found in effluents and cigarette smoke. The substance is prioritised as high concern.

3,4-Dichloroaniline is used as an intermediate in closed systems but is also formed as a metabolite of linuron and diuron, which are used on food crops. Exposure is expected indirectly through food. 3,4-Dichloroaniline is persistent but does not bioaccumulate and is found in the environment in water systems. The substance is classified as having high concern for exposure.

Medium concern

Nitrofen is a herbicide used on food crops. Human exposure may be expected by food but because this substance is restricted in the EU, exposure is less likely. Nitrofen is inherently biodegradable and bioaccumulative and not measured in the environment (in water systems). This substance is prioritised as medium concern.

Amitrole is a herbicide used alongside roads. Exposure is possible through contact of playing children with soil and plants alongside roads. But because the substance is not persistent and not bioaccumulative exposure is not likely. Amitrole is found in the environment primarily in water systems. The substance is prioritised as medium concern.

4-Tert. octylphenol and nonylphenol are used as raw materials for detergents, emulsifiers, in paints, anti-oxidants, pesticides, in PVC and as spermicide in contraceptive foams. The substances are also degradation products of APEOs. Exposure is expected through release from polystyrene and PVC (e.g. in baby bottles). The substances are inherently biodegradable and expected to bioaccumulate. The substances are found in the environment in water systems and biota. The substances are classified as having medium concern.

Low concern

Nitrotoluene is used as an intermediate in the varnish industry, pharmaceuticals and fragrants. It is used in closed system, which indicates no exposure. Although it is inherently biodegradable it is not bioaccumulated. Nitrotoluene is prioritised as low concern.

Tetrabutyltin is used as an intermediate in the production of other organotins and exposure is not likely. The substance is persistent and not found in the environment and therefore prioritised as low concern.

Acetochlor

The substance was selected to be evaluated in the expert meeting because it is a HPV chemical which is produced in more than 1000 tonnes/year.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 3 and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1 Physico chemical properties of acetochlor

Water solubility	223 mg/l at 23 C (Worthing 1987)
Vapour pressure	Negligible (Worthing 1987)
Log Kow	3.03 experimental (Syr, 1996)

Acetochlor is moderately soluble in water.
The EC formulation is stable > 2 years at room temperature. The main loss from soils is by microbial degradation (Worthing, 1987).
Acetochlor is not persistent and degrades rapidly in soil with an average DT50 of 4-5 weeks, entirely by microbial degradation. Due to low vapour pressure limited volatisation is expected and if present in air it is expected to degrade rapidly (calc. DT50 2.6 days). Studies in mammals and fish demonstrate no bioaccumulation (CEFIC, 1999)

Use, Exposure and emissions
The majority of the formulated material is exported to meet product demands in non-EU territories. The quantity of aectochlor used in the EU is < 1000 tonnes (CEFIC, 1999).
Acetochlor is a selective pre-emergence or pre-plant herbicide controlling annual grasses, certain annual broad-leaved weeds and yellow nutsedge in soybeans. It is absorbed by the shoots (less so by the roots) of germinating plants and inhibits protein synthesis in susceptible plants (Worthing, 1987).

Vulnerable use and vulnerable groups
Because acetochlor is used as a herbicide on food crops this could mean a certain risk. However acetochlor is not persistent. Acetochlor could also present a risk to agricultural workers applying the herbicide. Assumed is that these workers take the neccessary precautions using the substance.

Environmental concentrations
There are no measurements of acetochlor in the environment.

Legal status
No information available on the legal status of acetochlor.

Conclusion
Acetochlor is used on food crops. Human exposure may be expected by food but because these substances are not persistent, exposure is less likely. The substance is not bioaccumulative. The substance is prioritised as having high concern for exposure.

It should be checked whether there is indeed no or hardly any exposure.

References
Syr 1996. Syracuse corporation estimate

Worthing, (1987). The pesticide manual. A World Compendium. 8th Edition.

Alachlor

The substance was selected to be evaluated in the expert meeting because it is a HPV chemical which is produced in more than 1000 tonnes/year.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 2 and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1 Physico chemical properties of alachlor

Water solubility	148 mg/l (fra97) (ARS, 1995 in fra97) 240 mg/l (4 RIWA, 1998)
Vapour pressure	2.9 mPa at 25C (Worthing 1987)
Henry coefficient	0.0021 Pa.m3/mole at 25 ºC (ARS, 1995 in fra97)
Log Koc	2.09 (ARS, 1995 in fra97)
Log Kow	2.9(ARS, 1995 in fra97)\ 3.09 (4,3 RIWA, 1998)

Alachlor is moderately soluble in water. Based on the log Kow slight bioaccumulation is expected.
The degradation in sediment and plants takes place by the splitt off of the chloride atom. Thereafter further degradation to aniline derivates takes place. After 4 to 5 weeks in sediment the greatest part of alachlor is degraded (fra97). The biodegradation half-lives are 27 days (ARS, 1995 in fra97), 23 days aerobically in soil on 0-0.6 m at 20ºC, 100 days anaerobically in soil on 0-0.6 m at 20 ºC, 4-5 weeks in soil and plants due to rapid splitt off of the chloro atom and degradation of derivatives (Verschueren, 1996 in fra97). The DT50 in water is > 30 days and in soil 22 days (4 RIWA, 1998). Its' persistence in soil ranges from about 1 to 3 months and it is degraded by hydrolysis, photolysis and microorganisms to a large number of metabolites containing functional groups of dihydroindoles, tetrahydroquinones, acetanilides, and anilides (gr99).
The release of alachlor in the environment occurs particularly as a result of its application in the field. In soil, alachlor is transformed to its metabolites primarily by biodegradation. The half-life of alachlor disappeared from soil is about 15 days, although very little mineralisation has been observed.
Alachlor is rapidly metabolised in plants (Worthing 1987). It persists in soil 42-70 days depending on the conditions. The loss being by microbial metabolism (Worthing 1987).
Alachlor is highly to moderately mobile in soil and the mobilization decreases with an increase in organic carbon and clay content in soil. In water both photolysis and biodegradation are important for the loss of alachlor, although the role of photolysis becomes important in shallow clean water, particularly in the presence of sensitizers.
The bioconcentration of alachlor is aquatic organisms is not important (gr99).
Alachlor is eliminated in a sewage treatment plant by an active coal filtration. In which > 90% is eliminated (long execution time 2000 l/g) (1 RIWA, 1998).

Use, Exposure and emissions
The use of alachlor is forbidden in the Netherlands since the 90s (gre96).
Alachlor is used as a herbicide in cabbage, corn and winter cabbage cultures (perkow and ploss 1996 in bruhn 1998). Alachlor is a commonly used herbicide for preemergence and early postemergence control of many annual broad-leaved weeds and most grasses. It is used on crops such as corn, peas, soybeans, peanuts, beans, cotton, milo and sunflowers. It is usually sold in the form of emulsifiable concentrates, microscopic capsules and granules for application to the soil or weeds where the above types of crops are grown (gr99).

Vulnerable use and vulnerable groups
Because alachlor is used as a herbicide on food crops this could mean a certain risk. However alachlor is rapidly degraded and quickly metabolised in plants. Alachlor could also present a risk to agricultural workers applying the herbicide. Assumed is that these workers take the neccessary precautions using the substance.

Environmental concentrations
In the Fraunhofer report alachlor is measured in water with a median concentration of 0.0525 µg/l (mean 0.0793 µg/l) based on 120 data from 25 stations (39 data were above the determination limit). Furthermore it is measured at several sites in the Netherlands.

Table 2 Occurrence in the environment of alachlor

Compartment	Year	Time	Location	Concentration average (max.)	Unit	Reference (source)
Water	1992		Lakes and rivers	0.05 (0.14)	µg/l	Gre96 (RIZA nota 92/96 in fra97)
Water	1992		North-sea coast	0.04 (0.07)	µg/l	Gre96 (RIZA nota 92/96 in fra97)
Water	1992		Wadden-sea coast	0.04 (0.1)	µg/l	Gre96 (RIZA nota 92/96 in fra97)
Water			Rhine	0.02-0.04	µg/l	6 RIWA, 1998
Water			Rhine	0.1-<1	µg/l	22 RIWA, 1998
Water			Meuse	0.1-1	µg/l	22 RIWA, 1998
Water			Boezemwater	0.02-0.04	µg/l	6 RIWA, 1998
Water			Stagnant waters	0.02-0.04	µg/l	6 RIWA, 1998
Water			Ijsselmeer	0.1-1	µg/l	22 RIWA, 1998
Water			Haringvliet	0.1-1	µg/l	22 RIWA, 1998
Ground water			Rhine bank	0.04	µg/l	6 RIWA, 1998
Drinking water				Not found in drinking water		22 RIWA, 1998

d.l= detection limit

Legal status
Alachlor is listed on the OSPAR candidate list and Priority pesticides list under Directive 91/414/EEC (and specified under Council Regulation 3600/92)

Conclusion
Alachlor is used in food crops which may result in human exposure. Human exposure may be expected by food but because this substance is not persistent, exposure is less likely. Alachlor is measured in the environment (in water systems). The substance is prioritised as high concern.

The extent of the exposure should be checked.

References
Bruhn, T., et al, (1998), Umweltforschungsplan des bundesministeriums für umwelt, naturschutz und reaktorsicherheit. Einstufung von Schadstoffen als endokrin wirksame Substanzen.
Forschungsbericht 216 02 001/08.
DHC99: Dutch Health Council (1999). Endocrine-disrupters in the Netherlands.
Fra97 Franse & de Voogt, (1997). Oestrogene verbindingen in het Nederlands milieu, MTC report.
Gre96: Greve, (1996). (Dutch Health Council). Hormoon-verstorende stoffen in Nederland. Gebruik, emissie, milieuconcentraties en fysisch/chemische karakteristieken
RIWA (1998), Xeno-oestrogenen en drinkwater(bronnen).
Worthing, (1987)The pesticide manual. A World Compendium. 8th Edition.

Atrazine

The substance was selected to be evaluated in the expert meeting because it is a HPV chemical which is produced in more than 1000 tonnes/year.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 2 and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1 Physico chemical properties of atrazine

Water solubility	30-35 mg/l (gre96) 30 (4 RIWA, 1998)
Vapour pressure	4E-5 Pa (Ordelman, 1993b)
Henry coefficient	0.00029 Pa.m3/mole (gre96)
Log Koc	2.1 (gre96)
Log Kow	2.6 (gre96) 2.5 (4 RIWA, 1998) 2.49 (1 RIWA, 1998)

Atrazine is moderately soluble in water.
The half-life in water is 40 to 750 days (gre96).
The s-triazine ring of atrazine is fairly resistant to degradation. 2-Chloro-4-ethyl-amino-6-amino-s-triazine, 2-chloro-4-amino-6-isopropylamino-s-triazine, 2-hydroxy-4-ethylamino-6-isopropyl-amino-s-triazine and 2-hydroxyl-4-ethylamino-6-amino-s-triazine have been identified as microbial trans-formation products of atrazine. Chemical degradation of atrazine may be more important environmentally than biodegradation. Atrazine may hydrolyse fairly rapid in either acidic or basic environments, yet is fairly resistant to hydrolysis at neutral pHs. Furthermore the rate of hydrolysis was found to drastically increase upon small additions of humic materials, indicating atrazine hydrolysis could be catalyzed (828 in DHC99). Atrazine is hydrolysed with a half-life of 742 days in fresh water and biodegraded in >40 days. In combination the half-life in water is 60 days (Ordelman 1993b). The half-life in sediment 85 days (Ordelman, 1993b).
Photochemical metabolisation of triazines may also occur. As end-product of the metabolisation of atrazine 2,4,6-trihydroxy-1,3,5-triazine is found. In water and sludge-systems several metabolisation products of atrazine are found: desisopropyl-atrazin, desethyl-atrazin, desisopropyl-hydroxy-atrazin en 2-hydroxy-4,6-desethyl-1,3,5-triazine. Desisopropyl-atrazin and desethyl-atrazin have been found in ground water (Ordelman, 1993b).
The DT50 in soil is 50 days and in water 86 days (4 RIWA, 1998). According to Cefic material DT50 in soil is 16-77 days in laboratory and field studies, resp. (CEFIC, 1999).

Biodegradation of triazines may go through several routes: dechlorination to a hydroxy compound (atrazine), removal of a alkyl group and removal of a amino group. Removal of the alkyl group is the first step in the microbial metabolisation of s-triazines. This results for atrazine in the formation of desethyl-atrazin (2-amino-4-chlor-6-isopropylamino-s-triazine) and desisopropyl-atrazine (e ethylamino-4-chloro-6-amino-s-triazine). This product is mostly formed by soil organisms (Adams and Thurman, 1991 in Ordelman 1993b) but may also be formed in water systems (Ordelman, 1993b).

The BCF for atrazine is 10-83 for algae, 2-15 for molluscs, 3-10 for fish and 40 in sludge (Bol, et al, 1992 in Ordelman, 1993b). This means no bioaccumulation potential. For atrazine biomagnification is not an important process. Two food chains have been studied (algae> daphnias and daphnias> catfish). The contribution of food to the accumulation of atrazine was for both food chains not higher than 10%. This means that uptake from water is the most important route (Ellgehausen , 1980 in Ordelman,

1993b). Based on CEFIC material atrazine has a low bioaccumulation potential: 28 day BCF is 7.7 to 15 for Lepomis macrochirus (CEFIC, 1999).

Use, Exposure and emissions

Atrazine is not produced in the Netherlands. In Europe atrazine is produced in the UK in Hauxton and in Switzerland (Monthey) which is probably the biggest producer. Its production site is on the Rhône. Furthermore it is also formulated in other countries (Ordelman 1993b).

192 tonnes atrazine was used in the Netherlands in 1985, 188 tonnes in 1988, 189 tonnes in 1991 and 192 tonnes in 1994 (Ordelman et al , 1993). The emission is 2.25 tonnes/year in 1994 (gre96) and 3.6 tonnes/year in 1988. Atrazine is produced in more than 50 tonnes/year in Italy (Industria prodotti chimica SpA, Oxon). The total use in Europe is 6000 tonnes/year (RIWA, 1998). Production in the US alone is some 100 million pounds annually (882 in DHC99).

It is used as a selective herbicide for weed control in agriculture. Atrazine may be released into the environment through effluents at manufacturing sites and at points of application where it is employed as a herbicide (DHC99). The most probable exposure would be occupational exposure, which may occur through dermal contact or inhalation at places where atrazine is produced or used as a herbicide (828 in DHC99).

Atrazine is used as a hercide in corn- and asparagus cultures, below fruitcultures and vinegards. Furthermore it is used on roads and on uncultured land in combination with amitrol, bromacil and dalapon (Perkow and ploss 1996 in Bruhn 1998). Atrazine is a member of the s-triazine herbicides and is the most widely used herbicide in the world (811 in DHC99). Atrazine is a systemic active herbicide, which is mostly taken in by the roots of the plant. It is concentrated in the meristemic tissue of the plant. It inhibits photosynthesis and accelerates the metabolisation of nitrates to nitrites and ammonia. It has a long working period and is used in corn cultures.

Atrazine is deposited through atmospheric deposition of 170 tonnes in the Rhine in the total basin in 1991 of which 5.3 tonnes within the Netherlands. Atrazine is also supplied from abroad by the rivers Rhine and Meuse and is measured in these rivers (Ordelman 1993b).

Vulnerable use and vulnerable groups

Because atrazine is used as a herbicide on food crops this could mean a certain risk. Exposure may also occur during application and production. Assumed is that workers take the neccessary precautions using the substance. The use on roads is the most vulnerable exposure because children and other vulnerable groups may be exposed in this way.

Environmental concentrations

The EEA groundwater report states that the substance is found in at least six countries (including eastern European countries) in groundwater concentrations above 0.1 µg/l (38 in Fraunhofer report). The Scremotox-study finds for the total North Sea that atrazine is within the priority ranks 1-20 out of the 52 ranked substances (37 in fraunhofer, 1999).

Atrazine is found in marine and freshwater in 1992 and 1993, in rain water in 1988 and 1993 and in shallow ground water (Ordelman, 1996).

Table 2: Occurrence in the environment of atrazine

Compartment	Year	Time	Location	Concentration average (max.)	Unit	Reference (source)
Water	1993		Lakes and rivers	0.1 (0.7)	µg/l	Gre96
Water			North-sea coast	0.08 (1.1)	µg/l	Gre96
Water			Wadden-sea	0.04 (0.2)	µg/l	Gre96
Water			Rhine	0.1-<1	µg/l	22 Riwa, 1998
Water			Rhine	0.01-0.2	µg/l	Riza, Riwa, Kiwa in Riwa, 1998
Water			Meuse	0.1-<1	µg/l	22 Riwa, 1998
Water			Meuse	0.01-1.8	µg/l	Riwa, 1998

Compartment	Year	Time	Location	Concentration average (max.)	Unit	Reference (source)
Water			Meuse	0.44	µg/l	88 Riwa, 1998
Water			Drentsche Aa	0.03	µg/l	87 Riwa, 1998
Water			Groote Beek	0.62	µg/l	87 Riwa, 1998
Water			Twente kanaal	0.33	µg/l	87 Riwa, 1998
Water			Z-willemsvaart	0.38	µg/l	87 Riwa, 1998
Water			Almelo's kanaal	0.4	µg/l	87 Riwa, 1998
Water			Ettenlandsch kanaal	0.08	µg/l	87 Riwa, 1998
Water			Bergumermeer	0.19	µg/l	87 Riwa, 1998
Water			Ijsselmeer	0.05	µg/l	88 Riwa, 1998
Water			Ijsselmeer	0.1-<1	µg/l	22 Riwa, 1998
Water			Haringvliet	0.1-<1	µg/l	22 Riwa, 1998
Water			Info-spec	0.01-0.6	µg/l	Riwa, 1998
Water			Rhine Lobith	0.12 (av) 0.2 (max)	µg/l	SIVEGOM, 1990 in Ordelman, 1993b
Water			Rhine Lobith	0.12 (av) 0.3 (max)	µg/l	SIVEGOM, 1991 in Ordelman, 1993b
Water			Rhine Lobith	0.13 (av) 0.25 (max)	µg/l	RIWA 1990 in Ordelman, 1993b
Water			Rhine Lobith	0.09 (av) 0.2 (max)	µg/l	RIWA 1991 in Ordelman, 1993b
Water			Rhine Lobith	0.08 (av) 0.19 (max)	µg/l	Forschungsvorhaben, 1990 in Ordelman, 1993b
Water			Rhine Lobith	0.08 (av) 0.21 (max)	µg/l	Forschungsvorhaben 1991 in Ordelman, 1993b
Water			Meuse, Eijsden	0.28 (av) 0.7 (max)	µg/l	SIVEGOM, 1990 in Ordelman, 1993b
Water			Meuse, Eijsden	0.25 (av) 0.6 (max)	µg/l	SIVEGOM, 1991 in Ordelman, 1993b
Water			Meuse, Eijsden	0.33 (av) 1.0 (max)	µg/l	RIWA, 1990 in Ordelman, 1993b
Water			Meuse, Eijsden	0.43 (av) 1.8 (max)	µg/l	RIWA, 1991 in Ordelman, 1993b
Water			Meuse, Keijzersveer	0.35 (av) 1.0 (max)	µg/l	RIWA, 1990 in Ordelman, 1993b
Water			Nieuwe Waterweg, Maassluis	0.09 (av) 0.1 (max)	µg/l	Van Steenwijk, 1992 in Ordelman, 1993b
Water			Westerschelde, Schaar v ouden Doel	0.15 (av) 0.21 (max)	µg/l	Van Steenwijk 1992 in Ordelman, 1993b
Water			Ijsselmeer, Andijk	0.26 (av) 0.7 (max)	µg/l	RIWA, 1990 in Ordelman, 1993b
Water			Ijsselmeer, Andijk	0.38 (av) 0.9 (max)	µg/l	RIWA, 1991 in Ordelman, 1993b
Water			Aa Oost-Brabant	2.9 (av) 14 (max)	µg/l	Waterschap De Aa, 1993 in Ordelman, 1993b
Ground water			Corn	0.19 (av) 0.58 (max)	µg/l	Verdam, 198 in Ordelman, 1993b
Ground water			Corn	0.07 (av) 0.3 (max)	µg/l	Lagas, 1989 in Ordelman, 1993b
Ground water			Corn	0.06 (av) 0.3 (max)	µg/l	Lagas, 1990 in Ordelman, 1993b
Ground water			Corn	1.77 (av) 7.4 (max)	µg/l	Lagas, 1991 in Ordelman, 1993b
Ground water			Potatoes/beet/grain/ corn	0.05 (av) 0.14 (max)	µg/l	Cornelese en van Maaren, 1992 in Ordelman, 1993b
Rain water			North America	1.5 (max)	µg/l	Hopman, 1990 in

Compartment	Year	Time	Location	Concentration average (max.)	Unit	Reference (source)
						Ordelman, 1993b
Rain water			Switzerland	0.001 (dl)-0.6	µg/l	Buser, 1990 in Ordelman, 1993b
Rain water			Italie	Up to 1.99 (in 10 of 146 samples)	µg/l	Trevisan, 1993 in Ordelman, 1993b
Rain water	1991		Germany	Found on 4 locations	-	Geissler and Scholer, 1993 in Ordelman, 1993b
Rain water	1991		Netherlands, Eibergen	0.39 (av) 0.74 (max)	µg/l	RIVM (Van Zoonen), 1991 in Ordelman, 1993b
Rain water	1991		Netherlands, Huibergen	0.08 (av) 0.16 (max)	µg/l	RIVM (Van Zoonen), 1991 in Ordelman, 1993b
Rain water	1991		Netherlands, Vredepeel	0.05 (av) 0.06 (max)	µg/l	RIVM (Van Zoonen), 1991 in Ordelman, 1993b
Rain water	1991		Netherlands, Kloosterburen	0.07 (av) 0.16 (max)	µg/l	RIVM (Van Zoonen), 1991 in Ordelman, 1993b
Rain water	1991		Netherlands, Leiduin	0.09 (av) 0.14 (max)	µg/l	RIVM (Van Zoonen), 1991 in Ordelman, 1993b
Rain water	1991		Netherlands, Wieringerwerf	0.09 (av) 0.19 (max)	µg/l	RIVM (Van Zoonen), 1991 in Ordelman, 1993b
Rain water	1993		Netherlands, Flevoland	0.31 (av) 0.09 (max)	µg/l	HS Fleverwaard, 1993 in Ordelman, 1993b
Sediment	-		Mark en Dintel Netherlands	<0.1 (dl)	µg/l	HS West-Brabant, 1989 in Ordelman, 1993b

d.l= detection limit

Legal status

Atrazine is on list 2 of Council Directive 76/46/EEC, Annex 1A of the Third North Sea Conference, the OSPAR candidate list, the HELCOM priority list, the Priority pesticides list under Directive 91/414/EEC (and specified under Council Regulation 3600/92). Furthermore the substance is referred to in Directive 79/119/EEC including restricted substances (shortlist derived from DG ENV);

Conclusion

Atrazine is a herbicide used alongside roads and also used on food crops. Exposure is expected though food and direct exposure by contact of playing children with soil and plants alongside roads. Atrazine is persistent and not bioaccumulative and is found in the environment primarily in water systems. Atrazine is prioritised as having high concern.

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Butylbenzylphthalate (BBP)

The substance was selected to be evaluated in the expert meeting because it is a HPV chemical which is produced in more than 1000 tonnes/year.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 3 and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1 Physico chemical properties of butylbenzylphthalate (BBP)

Water solubility	2.7 mg/l (Iuclid, 1996) 29 mg/l (gre96) (teunissen 96b in fra97) 4 mg/l (ritsema 89 in fra97) 3 mg/l (30 riwa, 1998)
Vapour pressure	5.0E-06 Pa 25 degC (Staple, 1997)
Henry coefficient	5.8E-04 Pa.m3/mol (Staples, 1997) 0.13 Pa.m3/mol (gre96) (teunissen 96b in fra97)
Log Koc	4.3 (gre96) calc. (teunissen 96b in fra97) 4.7 calc. (ritsema 89 in fra97)
Log Kow	4.9-5.2 (gre96) (teunissen 96b in fra97) 4 (ritsema 89 in fra97) 4.78 (30 riwa, 1998)

BBP is poorly soluble in water with a very low volatily. BBP is a low persistent to medium persistent chemical. The biodegradation half-lifes are 24-168 days in soil, 6-60 days in air, 24-168 days in surface water and 48-4320 days in ground water (howard, 1991 in fra97). The DT50 in water (hydrolysis) is 28 days (30 riwa, 1998). The expected removal from a sewage treatment plant is > 90% with active coal (riwa, 1998). The degradation in the sewage treatment plant is 99% in 1.5 day (riwa, 1998).
The log Koc indicates a strong sorption.

The log Kow indicates a possible bioaccumulation (fra97). However, BBP is bioconcentrated in organisms at low trophic levels (mussels) but are metabolized and excreted by fish, birds and mammals, primarily as phthalic acid and the mono-ester (temanord96 in sepa98). BBP is not considered to bioaccumulate in food chains and both are biodegradable (sepa98).

Limit value in water (Grenswaarde/MTR) is 9 µg/l (gre96)

Use, Exposure and emissions
BBP is produced 10000-50000 tonnes/year in the EU (CEFIC 163 (p3)) and used as a softener and a plasticizer (CEFIC 163 (u3)). DBP and BBP emissions amount to 500-800 DI tonnes/year (DBP and BBP) (EKOrappport 1995 in DHC99).

BBP is manufactured by the reaction of the monobutyl ester of phthalic acid with benzyl chloride (Skinner, 1992). In the USA, Monsanto Company is the sole manufacturer of BBP (Anon., 1996). In Europe it is produced in Germany (Bayer), Belgium (Monsanto Europe) and the UK.
Consumption of BBP in Europe is approximately 18 000-45 000 tonnes per year (Harris et al., 1997).

BBP is used mainly as a plasticizer in PVC for vinyl floor tile, vinyl foams, and carpet backing. Other polymers plasticized with BBP include cellulose plastics, polyvinyl acetate, polysulfides, polyurethane and in regenerated cellulose film for packaging. It is also used in vinyl products such as toys, synthetic

leathers, acrylic caulking, adhesive for medical devices and in the cosmetic industry as well as a dispersent and carrier for insecticides and repellents (813 in DHC99). In addition it is used as an organic intermediate, a solvent and a fixative in perfume. It is also a component of some consumer products, such as nail polish (Martin, 1996 in Gulden, 1998).

BBP is released from facilities that manufacture the substance or blend it with PVC (Howard, 1990). Releases may also occur through diffusion of BBP from PVC products.

Total on-site environmental releases of BBP reported to the Canadian National Pollutant Release Inventory by 11 facilities using BBP amounted to 3.7 tonnes in 1994, all to the atmosphere. Total transfers of BBP for off-site disposal were much higher, amounting to 33.3 tonnes in 1994, with 25.1 tonnes going to incinerators and the remainder, 8.2 tonnes, to landfill. A reported total of 3.7 tonnes of BBP was sent for recovery in 1994, 2.3 tonnes for energy recovery and 1.4 tonnes for recovery, reuse, or recycling (NPRI, 1996).

In the USA, it was estimated that manufacturing facilities released approximately 176 tonnes to the environment in 1993, with about 99% released to the atmosphere (TRI93, 1995).

BBP may be released to air through automobile emissions and from combustion of refuse (Graedel et al., 1986 in Gulden, 1998). It has also been detected in stack emissions from hazardous waste combustion facilities and from coalfired power plants in the USA (Oppelt, 1987 in Gulden, 1998). Reasonable worst-case emissions of BBP from incinerators, boilers, and industrial fumaces buming such wastes were predicted to be 3 $\mu g/m^3$ waste gas (Dempsey & Oppelt, 1993). In a study of four US coal-fired utility boiler plants, the emission rates for BBP in flue gases ranged from 2 1 0 to 3400 mg/h (Haile et al., 1984). BBP was identified, but not quantified, in extracts of municipal incinerator fly ash from the Netherlands, but it was not detected in extracts from Japan or Ontario (Eiceman et al., 1979 in Gulden, 1998).

In leachate from municipal landfills in the USA, BBP was detected, but not quantified (Brown & Donnelly, 1988 in Gulden, 1998). BBP has also been detected (detection limits not reported) in groundwater at disposal sites in the USA (Plumb, 199 l). BBP was also detected in 2 of 44 groundwater samples at a Superfund site in Michigan, USA, at estimated concentrations of 0.6 and 1.0 µg/litre (US EPA, 1996 in Gulden, 1998).

In Canada, BBP has been detected in stonn sewer effluents at concentrations up to 50 µg/litre (Hargesheimer & Lewis, 1987) and in effluents from municipal sewage treatment plants and industrial plants at concentrations up to 25 µg/litre (Munro et al., 1985; SIGMA, 1985; OMOE, 1988, 1990, 199l). BBP has also been detected in sludges from Canadian sewage treatment plants at concentrations up to 914 498 ng/g dry weight (OMOE, 1988 in Gulden, 1998).

BBP can be emitted from products containing the substance. For example, BBP has been detected in emissions from carpets (Bayer & Papanicolopoulos, 1990 in Gulden, 1998), PVC floorings (Bremer et al., 1993), and vinyl wall coverings (Etkin, 1995 in Gulden, 1998), although quantitative data were not identified.

CSTEE, 1998 collected a number of determinations of phthalates leachate from toys. From these leach values they used the highest emission rates as worst-case. The leached amounts of phthalates are calculated per area and time and for BBP the maximum emission rates are estimated at 15 µg/10 cm^2 (max. emission rate of DEHP 610 µg/10 cm^2). Assuming an exposure period of 6 hr; a product surface area, 10 cm^2; and body weight, 8 kg, the following intake dose of 1.9 µg/kg/day (DEHP 75 µg/kg/day) can be calculated (CSTEE, 1998).

Vulnerable use and vulnerable groups
Groups exposed to BBP are consumers, children and medical patients.

Environmental concentrations

Information on BBP is limited but in 25% of samples from Rhine (surface water and effluents) BBP was detected at a mean concentration of 0.078 mg/l (highest value 49 mg/l) (temanord 96 in sepa98). In the Fraunhofer report BBP is measured in sediment with a median concentration of 130 µg/l (mean 103.89 µg/l) based on 10 data from 9 stations (9 data were above the determination limit).

Table 2 Occurrence in the environment of butylbenzylphthalate (BBP)

Compartment	Year	Time	Location	Concentration	Unit	Reference (source)
Water			IJsselmeer	<0.01	µg/l	Ritsema 1989 in Fra97
Water			Rhine	0.04	µg/l	32 riwa, 1998
Water			Rhine	0.1-<1	µg/l	22 riwa, 1998
Water			Meuse	0.1-<1	µg/l	22 riwa, 1998
Water			Drentsche Aa	0.01	µg/l	87 riwa, 1998
Water			Zuid-Willems kanaal	0.01	µg/l	87 riwa, 1998
Water			Info-spec	0.1-1	µg/l	Riwa, 1998
Water				<0.06	µg/l	RIZA/RIKZ Loes in DHC99
Water				<0.01-0.1	µg/l	MTC, bel97 in DHC99
Sediment				<0.01-0.05	µg/kg	Ritsema 1989 in Fra97/MTC in DHC99
Sediment				<25-56	µg/kg ww	RIZA/RIKZ Loes in DHC99
Suspended matter	1986			0.01-0.9	Mg/kg	Bel97 in DHC99
Suspended matter				<49-705	µg/kg ww	RIZA/RIKZ Loes in DHC99
Air						
Soil						
Wildlife biota				<80-1730	µg/kg dw	RIZA/RIKZ Loes in DHC99
Humans						
Food						
Drinking water						

d.l= detection limit

Legal status

BBP is listed in Annex 1D of the Third North Sea Conference, the OSPAR candidate list and Council Regulation 793/93/EEC 1.3. priority list;

Conclusion

BBP is a substance of high concern for human exposure. The substance is used in cosmetics and toys, carpet, wallpaper and paint. Therefore also vulnerable groups like children and medical patients can be exposed to BBP.

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Bisphenol A

The substance was selected to be evaluated in the expert meeting because it is a HPV chemical which is produced in more than 1000 tonnes/year.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 1 and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1 Physical/chemical properties of Bisphenol A

Water solubility	120 mg/l (TemaNord, 1996)
Vapour pressure	27 Pa (at 1700C) (TemaNord, 1996) 5.32x10-6 Pa at 25C (TemaNord, 1996)
Henry coefficient	0.00001-0.000001 Pa.m3/mole (TemaNord, 1996)
Log Koc	2.5-3.2 (fra97)
Log Kow	2.2-3.8 (fra97)

Bisphenol A is crystalline powder (Tema Nord, 1996).
The solubility of bisphenol A in water is moderate, the volatisation very low.

The log Kow does not indicate bioaccumulation. The measured bioconcentration factor (BCF) in fish is between 5 and 68 and indicates low bioaccumulation. The calculated BCF (196) indicates medium bioaccumulation (Staples 1995 in fra97). After breeding of carps at 150 and 15 g/L for 42 days at 25C according to the OECD Guideline 305, the bioconcentration coefficient was 5.1 - 13.8 and <20 - 67.7, respectively (MITI in CEFIC197 (b1)/ Japan, 1997). Bioconcentration factors of 42 to 196 were estimated based on water solubility and the octanol/water-distribution coefficient (Gulden, 1998).
Only sparse information concerning the bioaccumulation of bisphenol A in biota has been published. The US EPA (EPA 1984, EPA/On, Doc. 408486028) estimated bioconcentration factors of bisphenol A from 15 to 366 using a model of Veith et al. (1979) and log Pow data (3.32, 3.84, 2.2 to 3.4) obtained by Korerunan & Gorekliov (1971), Lyman et al. (1982) and Eadsforth (1983), respectively (TemaNord, 1996).
The log Koc indicates a negletable sorption (fra97). The possibility of its translocation in soil is relatively small (Tema Nord, 1996).
Biomagnification is not significant because metabolism and excretion in mammals (gr99).

The half-life in sediment, surface water and ground water indicate that bisphenol A is persistent. According to the producers bisphenol A is biodegradable in water. The bisphenol that is not degraded in the water column and reaches the surface water will adsorb onto sediment. In the sediment biodegradation will take place. However bisphenol A is also potentially mobile in sediment and may therefore contaminate ground water (fra97). The biodegradation half-lifes in soil are 1 to 180 days, in air 0.74 to 7.4 days, in surface water 1-150 days, in ground water 2 to 360 days (hedset, 1993 in fra97).

Photolysis half lifes in water are 2.27 - 160 days (Tema Nord, 1996). Bisphenol A may be transformed in water by photolysis at wave lengths above 290 nm and most readily under alkaline conditions (Hanze 1994 in Tema Nord, 1996). Additionally, Peltonen et al. (1986a in TemaNord, 1996) have reported a photo-decomposition of vaporised bisphenol A, when irradiated by UV-B light, which yields reactive free radicals (TemaNord, 1996). The significance of photolysis as an important degradation process seems low as bisphenol A is expected to bind to organic materials and, therefore, may undergo sedimentation in aquatic systems (TemaNord, 1996). In air, bisphenol A may react with hydroxyl radicals with an estimated half life t1/2of 4 hours (Hanze, 1994 in TemaNord, 1996).

The degradation half-life of Bisphenol A in aquatic organisms is assumed to be 1 - 180 days under aerobic conditions, and 4 - 360 days under anaerobic conditions, respectively (Tema Nord, 1996). Some micro-organisms seem to be able to degrade bisphenol A (TemaNord, 1996). Lobos et al. (1992) isolated a gram-negative aerobic Bacillus species (strain MV1) from a sludge enrichment obtained from a waste water treatment plant associated with a plastics manufacturing facility. The bacterial strain was able to use bisphenol A as a sole carbon and energy source. 60% of the carbon contained in bisphenol A was mineralized to C02, 20% was associated with the bacterial cells and the remaining 20% was converted to soluble organic compounds (Lobos et al. 1992 in TemaNord, 1996).
It appears that a straight forward conclusion concerning the biodegradability of bisphenol A is difficult to make due to conflicting test results. Tumer & Watkinson (1986) used the "modified SCAS procedure" to test the biodegradation of bisphenol A and concluded that bisphenol A is "inherently" biodegradable. Wagner (1993) using the "EPA Shake Flask Method" found bisphenol A to be "readily biodegradable" using an unadapted inoculum. In natural waters collected adjacent to a bisphenol A chemical plant discharge Dorn et al. (1987) found a greater than 90% degradation of bisphenol A within four days and concluded that the compound is "readily biodegradable" (TemaNord, 1996).
Contrary to these results, Stone & Watkinson (1983) reported that bisphenol A is "not readily biodegradable". This conclusion was based on the results obtained in the "Closed Bottle Test" and the "Modified Sturm Test", which are standardized OECD screening tests for biodegradability. MITI (1992) found that 0% of the bisphenol A inoculated with activated sludge was degraded after a 14 day period, and concluded that bisphenol A is "not easily biodegradable". In other OECD biodegradation studies of bisphenol A biodegradation was observed to be very slow (Hanze 1994 in TemaNord, 1996). The conflicting results obtained concerning the biodegradability of bisphenol A may arise from differences in test method conditions, including differences in the ratio of the number of micro-organisms and the concentration of the test substance (Pedersen et al. 1994 in TemaNord, 1996). Furthermore, toxic inhibitory effects of the test substance towards micro-orgarusms may arise when high concentrations of the test substance are used inhibit or limit microbial degradation of the substance. Finally, variability in test result may be due to biological variation of the test systems themselves, for instance differences in number of active degraders in the inocula used (Pedersen et al. 1994 in TemaNord, 1996).
Acclimatization and redox conditions seem to play a significant role for the biodegradation of bisphenol A. In aerobic aquatic tests (non-acclimated) the half-life of bisphenol A has been reported to lie in the range 24 hours to 6 months, whereas acclimatized aerobic test showed a fast degradation of Bisphenol A with a half-life t1/2of 2.5 to 4 days. Under anaerobic conditions (non-acclimated) the half life of bisphenol A lay in the range 96 hours to 24 months (Howard et al. 1991, cited-in Hanze 1994 in TemaNord, 1996).
In summary, factors, which influence the biodegradability of bisphenol A, seem to be the microbial composition of the receiving environment and the prevailing redox conditions. In media where bisphenol A has not previously be introduced (non-acclimated/non-adapted), the degradation rate may turn out to be very slow. On the contrary, in acclimated media a complete mineralization of bisphenol A may quickly occur. Anaerobic conditions seem to retard the biodegradation of the compound (TemaNord, 1996).
Because adapted/ re-exposed inocula/media have been used in most of the tests yielding results indicating "inherent biodegradabability" or "ready biodegradability", which is not "'allowed" in ready biodegradability tests (Petersen et al. 1994 in TemaNord, 1996), the conclusions on biodegradability presented here are based on tests using unadapted inocula (TemaNord, 1996). Based on the test results obtained, it may be concluded that bisphenol A is not readily biodegradable (TemaNord, 1996).

Bisphenol A may, upon discharge to the environment, distribute between the air, water, soils, sediments and biota compartments (TemaNord, 1996). Eisenreich et al. (1981) reported that substances with a Henry's Law constant between 10^{-6} and 10^{-8} tend to partition predominately into water. Based on the moderately high water solubility, the very low vapour pressure and the low Henry's Law constant, it is concluded that bisphenol A may have a tendency to partition into water and that the rate of evaporation from soil and water will be low (TemaNord, 1996).

Hanze (1994) concluded on the basis of the Koc, of 293 to 1,524 and the high log Pow value of bisphenol A that this chemical has a high potential for absorption to organic materials (TemaNord, 1996).
In a fugacity calculation (level 1) using the "ESTHER" manual (Ken-d 4/89) the environmental distribution of bisphenol A has been estimated to be: 32% in sediments, 43% in water, 24% in soil and 3.5x10-5% in air (Hanze 1994 in TemaNord, 1996).

Based on the low vapour pressure of bisphenol A at room temperature, evaporation to and transportation in air is expected to be minimal. However, at elevated temperatures the vapour pressure of bisphenol A rises which will result in an increased volatility of the compound (TemaNord, 1996). Bisphenol A is expected to exist almost entirely in the particulate phase in the atmosphere. Loss from the atmosphere may occur by dry deposition or photolysis (Hedin & Pererüu,5 1993, Hanze 1994 in TemaNord, 1996). Hanze (1994) emphazised that molecular bisphenol A is expected to be transformed by photolysis and distribute to the atmosphere in particulate form which may diminish photochemical transformations (TemaNord, 1996).

The high affinity of bisphenol A for organic materials (based on log PO, and K"" values) indicates that mobility in soil is expected to be low (TemaNord, 1996).
Transportation of bisphenol A in the aquatic environment is considered to be the predominant pathway, for distributing the compound between environmental compartments. In the receiving water bisphenol A is expected to partition into particulate matter, sediments and biota considering the high log Pow, and Kow values of the compound. Therefore, it is expected that bisphenol A will have a low to moderate mobility in terrestrial soils and when entering aquatic environments will tend to partition into the sediment, dissolved organic matter and biota (TemaNord, 1996).

Bisphenol A is moderately lipophilic and binds more or less strongly to soil sediment and suspended particles. Abiotic degradation of bisphenol A in water is assumed to be negligible, since the molecule contains no hydrolyzable functional groups. Bisphenol A is, for the most part, biologically degraded, with a half-life of < 4 days. The primary metabolites of the microbial degradation have been identified as 4-hydroxybenzoic acid and 4-hydroxyacetophenone, which, for the most part, are further mineralized. Minor amounts of bisphenol A are hydroxylated tot 2,2-bis(4-hydroxyphenyl)-1,2-propanediol (lobos 92 in gulden 98).

If released to soil, bisphenol A is expected to have moderate to low mobility. It may biodegrade under aerobic conditions following acclimation. It may biodegrade under aerobic conditions following acclimation. If released to the atmosphere, bisphenol A is expected to exist almost entirely in the particulate phase. Bisphenol A in particulate form may be removed from the atmosphere by dry deposition or photolysis. Photodegradation products of bisphenol A vapor are phenol, 4-isopropylphenol, and a semiquinone derivative of bisphenol A (828 in DHC99).

Bisphenol A is almost completely excreted by rats within 2 days (Knaak and sullivan 96 in gulden 98). Based the log Kow, and considering the relatively rapid biodegradation, it is not expected that bisphenol A accumulates significantly in aquatic organisms (Gulden 98). Bisphenol A is excreted by rats via faeces and urine, as such and/or as glucuronides (knaak 1996 in sepa98). The degradability of bisphenol A seems to be fairly rapid but not rapid enough to be defined as readily biodegradable (temanord 1996 in sepa98).

No information has been found concerning the metabolic conversion of bisphenol A in aquatic and terrestrial invertebrates or vertebrates. In mammals (rats) bisphenol A metabolism occurs through a partial conversion into phenols, increasing their urinary content in a free and bound form. Bisphenol A is passed unaltered and in the form of glucuronides from the body in the urine and faeces (Knaak et al. 1966 cited in Sheftel 1995).
Pot97 suggest that the estrogen-like properties of Bisphenol A have not been manifested in previously conducted oral toxicity studies because of relative rapid metabolism and elimination of orally administered bisphenol A (cefic197 (ch4)).

Bisphenol A has been shown to be rapidly degraded in acclimated wastewater treatment plants (fur90 in cefic197) and in receiving waters (dot87 in cefic 197). Half lifes ranged from 1-2 days following a brief adaptation period (2-4 days) (SETAC99 in cefic 197 (cw3)). Expected removal from sewage treatment plant is >90% (riwa, 1998)

Use, Exposure and emissions

Peltonen et al. (1986ab) reported that bisphenol A is a major component of epoxy resins which are formed by a reaction between bisphenol A and epichlorohydrin. Solid epoxy resins are used in powder painting, the powder being sprayed on the metal object to be painted and a protective film subsequently formed during curing in an oven at 2000C. The temperature of the heating resistors may though reach 3500C - high enough to degrade the polymer with a subsequent contamination of the workroom air. Peltonen et al. (1986b) showed that thermal degradation of epoxy polymers started at 180-250C, with bisphenol A being a major degradation product (TemaNord, 1996).

Bisphenol A is one of the most widely produced chemicals worldwide (TemaNord, 1996).
In the EU bisphenol A is produced in approx. 650000 tonnes/year (cefic197 (p6)). The amounts of their production in EU are 100,000 - 500,000 t/year, and in Japan 260,000 t in 1994 and 240,000 t in 1996, respectively (108 in Japan). In 1995 18 tons were imported to Sweden as raw material for processes and 15 tons were imported included in chemical products (Prod.Reg.Kemi in TemaNord, 1996).
Bisphenol A is produced in Germany (Bayer AG, Krefeld 140.000m3 tonnes; Dow Germany Inc, Stade 113000 m3 tonnes) and the Netherlands (General Electric Pernis and Shell 89000 m3tonnes) (RIWA, 1998). In 1995 210,000 tonnes of bisphenol A were produced in Germany (Leisewitz97 in gulden98). The use in Germay is 137000 tonnes/year (TvBR1, 1997 in fra97). The emission is 2.6 + 9.3 tonnes/year in Germany (TvBR1, 1997 in fra97).

Bisphenol A is an intermediate in the manufacture of polymers, epoxy resins, polycarbonates, fungicides, antioxidants, dyes, phenoxy, polysulfone and certain polyester resins, flame retardant and rubber chemicals (813 in DHC99).
Other uses of bisphenol A are as a resin in plastic dental fillings and polycarbonate plastics are used to coat teeth, especially children's teeth and the packing industry as in the inside of food cans. At this last use the bisphenol may migrate to the food (fra97; 813 in DHC99).
Apart from being used primarily as an intermediate in the production of epoxy and polycarbonate resins, bisphenol A is widely used as a component in the manufacture of phenoxy resins and corrosion-resistant unsaturated polyester-styrene resins. Furthermore, bisphenol A is used as a stabilizer for plasticizers (mainly DINP & DIDP) in PVC, as a thermal stabilizer for PVC resins, as an antioxidant in rubber and plastics, as a fungicide, and as a raw material in the production of tetrabromobisphenol A and other compounds used in the manufacture of flame retardants (EPA 1984, Sheftel 1995, Hanze 1994, I. Andersson, KemI, pers. comm. In TemaNord, 1996).

Most bisphenol A is used as a monomer in the production of polycarbonates and epoxy resins. In addition, bisphenol A is used as an antioxidant in plastics and hydraulic fluids, in the production of flame and tooth-filling material (composite with bisphenol –A-glycidyl (methacrylate as matrix resin) (Leisewitz97 in gulden98).
Bisphenol A is predominantly used as a raw material of polycarbonate resin and epoxy resin. Other usages are as the raw materials of phenol resin, plastic polyester, anti-oxidation agent, stabilizer of polyvinyl chloride and engineering plastics (polysalphon, bismalade, triazine, and polyallilate) (Japan, 1997).
Bisphenol A may leach from plastic due to incomplete polymerization or breakdown of the polymer during heating, e.g. during sterilization by autoclaving (813 in DHC99).

A materials flow analysis of bisphenol A is underway (Leisewitz 97 in gulden98). According to that, the emission of bisphenol A during production and processing, through migration of the converted monomers of polycarbonate and through the recycling of fax paper is estimated to be 3 tonnes total

yearly. According to manufacturers, less than 1 tn/year of bisphenol A is emitted through air and water during production and further processing (leisewitz, 97 in gulden98). Bisphenol A can be emitted from food service items made from polycarbonates and from epoxy resin coatings in foodstuff cans. According to EU directive 90/128/EWG (synthetic materials directive), a specific migration value of 3 mg/kg is allowed for bisphenol A. Bisphenol A and bisphenol-A-glycidyl-methylacrylate can also be released from synthetic tooth-filling material (spahl 91, Olea 96 in gulden 98).

The primary sources of environmental release of bisphenol A are expected to be effluents and emissions from its manufacturing facilities and facilities which manufacture epoxy, polycarbonate, and polysulfone resins. The most probable routes of human exposure to bisphenol A are inhalation and dermal contact of workers involved in the manufacture, use, transport or packaging of this compound or use of epoxy powder paints. On the other hand, when the above mentioned resins are used for packing of foods, the possibility of their incorporation into foods is suggested (89 in Japan). Bisphenol A has been shown to leach from polycarbonate, a common plastic used for many consumer products including jugs used to bottle drinking water. Polycarbonates also are commonly used to line food cans and recent studies found bisphenol A in half the canned foods samples where the foods contained up to 80 parts per billion (0.080 ppm), an amount 27 times more than the amount reported to make breast cancer cells proliferate (813 in DHC99). Recently, liquor obtained from tinned vegetables that contained bisphenol A exhibited estrogenic effects with human cancer cells (909 in DHC99).

The consumption of bisphenol A in the Nordic countries is collocated as follows. The data represent produced and imported amounts; amounts contained in exported products having been subtracted. In Sweden in 1994 the annual consumption of bisphenol A is 87 tonnes, in Norway in 1995 30 tonnes (incl. Exported quantities as well as imported and produced amounts), in Denmark in 1995 1120 tonnes and in Finland in 1995 28 tonnes. The total annual consumption of bisphenol A within the Nordic countries lies around 260 tonnes (TemaNord, 1996).
In 1992 the amount of bisphenol A consumed in Sweden was estimated from Swedish Products Register data to be 41 t, of which 16 t was used as antioxidants in plastic materials (KemI 15/95). Hedin & Perenius (1993) estimated that the annual consumption of bisphenol A in Sweden lies in the range 30-260 tonnes (TemaNord, 1996).

In table 2 the five most common functions in terms of annual consumption of products containing bisphenol A are presented for the countries Sweden, Denmark and Norway.

Table 2: Functions of products containing bisphenol A.

Functions of products containing bisphenol A	**Annual consumption (t)**
Sweden-	
• Raw materials- plastic manufacture	~ 30
• Stabilizers	~ 27
• Raw materials for synthesis +raw imterials for ceramics &glass, filling in materials	~ 20
• Hardeners	~9
• Plasticizers	~ 0.2
Total	~ **86** (98.9%)
Denmark	
• Binding materials	~ 38
• Insulating materials	~ 19

• Hardeners	~ 19
• Paints/lacquer	~ 7
• Filling in materials	~ 6
Total	**- 89 (79.5%)**

Norway

• Hardeners for plastics	8
• Paint and harnesses (other appl.)	5
• Stabilizers	4
• Glue	3
• Binders for paint, glue, harnesses	3
Total	**23(76.7%)**

From this table it is evident that the use of bisphenol A is predominately confined to binding, stabilizing, plasticizing and hardening functions in plastic products, paints/lacquers, binding materials and filling-in materials. The five most common uses in terms of amounts consumed account for 98.9%, 79.5% and 76.7%, respectively, of the total consumption in Sweden, Denmark and Norway (TemaNord, 1996).

It appears that bisphenol A is mainly used in industrial branches such as the chemical industry, building and construction industry, the iron/metal industry, the plastics industry and service industries. In Sweden, Denmark and Norway, 98.9%, 104.5% and 83.3%, respectively, of total bisphenol A consumption can be ascribed to these industrial branches (TemaNord, 1996).

Based on the high world-wide production volume of bisphenol A, and the fact that it is used at many sites and in many types of products, it is likely that bisphenol A enters the environment in substantial quantities (EPA 1984). Both diffuse sources (products in use, rest and waste products) and point sources (accidental spills, industrial wastewater discharges) may contribute to the emission of bisphenol A to the environment (TemaNord, 1996).
Emissions of bisphenol A during production of the pure chemical are considered to be minimal because the production occurs in a closed system (EPA 1984). However, inadvertent and accidental spins may occur during manufacturing, processing, handling and distribution of the chemical (TemaNord, 1996).
Important point sources for the emission of bisphenol A to the surrounding environment may be the large volumes of waste waters from industries manufacturing epoxy--, polycarbonate- and polysulphone hardeners and from industries involved in rubber production (EPA 1984, Hanze 1994, Lobos et al. 1992). Matsumoto (1982) considered the bisphenol A detected in polluted japanese rivers to originate mainly from industrial products such as epoxy and polycarbonate resins and their degradation products (TemaNord, 1996).

No estimates of emissions of bisphenol A from point sources were found in the literature (TemaNord, 1996).

Emission of bisphenol A from products in use has been reported by a few authors. Krat et al. (1986), cited in Sheftel (1995), determined the migration of bisphenol A into water from epoxy coatings during a 7 day period to be 4 µg/l at 37C. Buczowska & Jarnuszkiewicz (1971) showed that bisphenol A and other components from epoxy resins used as corrosionresistant coatings in ship water-tanks migrated into the water if the epoxy had not been properly hardened (TemaNord, 1996).

Leakage of bisphenol A and the related compound BADGE (bisphenol Adiglycidyether) into food-simulant liquids (distilled water, 15% ethanol, 3% acetic acid, olive oil) has been reported by Philo et al. (1994). Bisphenol A underwent 40% decomposition in 1 hour at 100C and 3% acetic acid.

BADGE underwent 90-100% decomposition in all aqueous simulants and 15-25% decomposition in olive oil at 175C. Brotons et al. (1995) reported migration of bisphenol A into canned food from the lacquer coatings of the cans. Up to 33 µg bisphenol A per can were detected (Brotons et al. 1995 in TemaNord, 1996).
Krishnan et al. (1993) found that bisphenol A is released from polycarbonate flasks during autoclaving. Polycarbonate is produced by condensing monomer bisphenol A with phosgene gas to yield carbonate linkages that make up the polycarbonate polymer. The carbonate linkages are subject to hydrolytic attack at high temperatures; such degradation is accelerated in alkaline pH and retarded at pH 5 or below. Krishnan et al. (1993) reported that bisphenol A leaches out of polycarbonate flasks during autoclaving in concentrations up to 10-15 nM ~ 2.3-3.4 µg/l (TemaNord, 1996).

Based on the concentration of bisphenol A in percolates from Danish waste dump sites (30 µg/l) and. a yearly percolate estimate of approximately 1 million m3 Kjolholt et al. (1994) estimated the emission of bisphenol A from waste disposal sites in Denmark to be 30 kg yr-l (TemaNord, 1996).

Vulnerable use and vulnerable groups

At point sources and by contamination of food items, the compound may be a hazard (sepa98).
Bisphenol A is used in and proven to leach from food cans. Therefore it presents a risk to humans. It is also used in dental fillings and teeth coatings especially for children. It is unknown if it leaches from these fillings but this may present a risk to children.

Environmental concentrations

There are almost no data on concentrations of bisphenol A in the environment. In a polluted river in the Tokyo area of Japan, 0.06-1.9 mg/l were determined but no data on bisphenol A levels in aquatic systems, sediments or terrestrial systems from Europe have been found (SEPA 98). In polluted Japanese rivers concentrations of up to 9 µg bisphenol A/l have been detected (TemaNord, 1998).

No information on sea water concentrations of bisphenol A was found. No data has been found concerning levels of bisphenol A in sediments or biota from fresh water or marine environments.
Only sparse information on levels of bisphenol A in terrestrial soils has been found in the literature (TemaNord, 1996).

Bisphenol A has been found in the Bilina river in the Czech Republic, a tributary of the Elbe. Its sediment is heavily contaminated with other chemicals and the bisphenol A contamination is suspected to come from an epoxy resin producing chemical plant found on the Bilina (kurz 96 in gulden98).

Muszkat et al. (1993) carried out field studies at 3 sites in the region of Glil-Yam (Israel). In the soil surface of a citrus grove, which had been irrigated with municipal effluents for 20 years, a bisphenol A concentration of 500 ppb (0.5 mg kg-1) was detected. In control soils which had received only rainwater or groundwater irrigation the bisphenol A content was n.d. and 200 ppb (0.2 mg kg-1), respectively (TemaNord, 1996).
In terrestrial soils from Israel Bisphenol A has been detected in concentrations up to 0.5 mg/kg.

In the water of industrial sewage treatment plants of industries that produce or process polycarbonates and epoxy resins, bisphenol A is found. The amount is estimated to be 20 µg/l (TemaNord, 1996). Concentrations in effluents from industries using bisphenol A have been reported to lie in the range 80 to 4,800 µg L-1 (Friedman 1980 in EPA 1984, Dom et al. 1987, Matsumoto et al. 1977, cited in EPA 1984 in TemaNord, 1996).

Kjolholt et al. (1994) were unable to detect any Bisphenol A in flue gas, cinders, fly ash, wash water and flue gas cleaning residue from a Danish waste incinerator plant (Naestved Forbraendingsanlaeg). Neither was bisphenol A found in compost from a Compost Treatment Plant in Denmark (Frederikssund Komposteringsanlaeg) or in gas produced from the compost (TemaNord, 1996).

Concentrations in air of 0.04-0.2 µg/m3 were measured 15 m above a field in Japan (Hanze 1994 in TemaNord, 1996). Concentrations of Bisphenol A in work place air are 1.7 µg/m3 in workplace air in laminating work, 6.3 µg/m3 in workplace air beside the painting machine in industrial painting with epoxy powder, 10-20 µg/m3 in welding work (>250C) (degradation products)., 0.4 µg/m3 in workplace air at drying epoxy paint in hot ovens and <0.05 µg/m3 in machine tooling polycarbonate plastics (TemaNord, 1996).

Bisphenol A has been reported to be released from lacquered cans (up to 33 mg/can), from polycabonate flasks during autoclaving (up to 3.4 mg/l) and from dental demenet (Krishnan 1993, Brotons 1994, Olea, 1996 in sepa 98).

Table 3 Occurrence in the environment of Bisphenol A

Compartment	Year	Time	Location	Concentration	Unit	Reference (source)
Water	-	-	USA	0.2-1.9	µg/l	Fra97
Water	1993		Rhine and Meuse at Lobith and Eysden	0.1 average (0.1 max.)	µg/l	Phernambucq in fra97
Water			Rhine	<0.1	µg/l	22 riwa, 1998
Water			Japanese river in Tokyo area	0.06-1.9	Mg/l	Sepa98
Water			Meuse Eijsden	0.03-0.035 av (0.16 max)	µg/l	Riza/rikz in DHC99 (LOES voorstudie
Water	1976		Japan	Not detected in 60 samples 0.05-0.1 (dl)	ppb	Japan, 1997
Water	1975-1976		Japan Tokeo area, polluted river water	0.06-1.9	µg/l	Matsumoto, 1982 in TemaNord, 1996
Water	1974-1975		Japan, Ogasawara Islands, unpolluted water	Not detected	µg/l	Matsumoto, 1982 in TemaNord, 1996
Water			Japan polluted rivers	0.01-0.09 1-9	µg/l	Matsumoto, 1977 in TemaNord, 1996
Sediment	1976		Japan	Not detected in 50 samples 0.2-5 (dl)	ppb	Japan, 1997
Wastewater				0.1-<1	µg/l	22 riwa, 1998
Municipal waste water			City	0.260-0.54	µg/l	Riza/rikz in DHC99 (LOES voorstudie
Effluent RWZI			City	0.03-0.37	µg/l	Riza/rikz in DHC99 (LOES voorstudie
Effluent			Industries that produce or process polycarbonates and epoxy resins	20	µg/l	TemaNord, 1996
Effluent			Industries using bisphenol A	80-4800	µg/l	Friedman, 1980 and Dorn, 1987 in TemaNord, 1996
Influent			Industrial	Mostly 5-10x higher sometimes 100 x higher (>2	µg/l	Riza/rikz in DHC99 (LOES voorstudie

Compartment	Year	Time	Location	Concentration	Unit	Reference (source)
				ug/l)		
Effluent AWZI			Industrial	0.02-0.1	µg/l	Riza/rikz in DHC99 (LOES voorstudie
Air			Field in Japan	0.04-0.2	µg/ m3	Hanze, 1994 in TemaNord, 1996
Air			Workplace air	<0.05-20	µg/ m3	Hanze, 1994 in TemaNord, 1996
Soil			Israel: Glil-Yam, soil irrigated with municipal effluent	500	ppb	Muszkat 1993 in TemaNord, 1996
Wildlife biota	1976		Fish in Japan	Not detected in 10 samples 5 (dl)	ppb	Japan, 1997
Food			Release from lacquered cand	<33	Mg/ can	TemaNord, 1996
Food			Release from polycarbonate flasks	<3.4	Mg/l	TemaNord, 1996

d.l= detection limit

Legal status

Bisphenol A is referred to in Commission directive 90/128/EEC relating to plastis materials and article intended to come into contact with foodstuffs, Council directice 67/548/EEC concerning the classification, packaging and labelling of dangerous substances.

Conclusion

Bisphenol A is used as a resin in dental fillings to coat teeth especially childrens teeth and in packaging material (coating in food cans from which it has been proven to leach). Exposure occurs through food and dental fillings which may be especially for children. The substance is persistent but does not bioaccumulate and is found primarily in water systems. The substance is classified as having high concern for exposure.

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Chlordane

The substances chlordane and chlordane (cis and trans) were selected to be evaluated in the expert meeting because it are a very persistent chemicals.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 2 and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1 Physico chemical properties of chlordane

Water solubility	0.67 (shiu 1990 in fra97) 0.1 (40 riwa, 1998)
Vapour pressure	1 x 10-5 mm Hg at 25 °C (EHC34)
Henry coefficient	0.00039 Pa.m3/mole (Verschueren 96 in fra97)
Log Koc	4.78 (ARS 95 in fra97)
Log Kow	5.5 (gre96) 6 (Verschueren 96 in fra97) 6 (1 riwa, 1998)

Chlordane is a viscous, light yellow to amber-coloured liquid (EHC34).
Chlordane is insoluble in water but soluble in most organic solvents (EHC34). The solubility of chlordane in water is poorly.
The log Kow and the solubility in water indicate potential accumulation in organisms (fra97). The BCF for a goldfish is 160, for a mosquito 8260 And for an oyster 7300 (24 riwa, 1998). Limited bioaccumulation in the adipose tissue of terrestrial and aquatic organisms can take place. In general, concentration factors in mammals are less than 1 (EHC34). Grimes & Morrison (1975) examined the uptake of chlordane by 13 types of bacteria and found that although the uptake of alpha- and beta-isomers of chlordane was the same for any one species, the concentration factors (CF) differed greatly between species. The CFs ranged from a few hundred to several thousand, with 3 species giving much higher values. The highest CF was 53 000 for Caulobacter vibrioides. Caulobacter cells were found to contain 4 distinct lipid-containing materials, and this was offered as an explanation of the high CF. Sanborn et al. (1976) used unlabelled chlordane and labelled 14C-chlordane on filamentous Oedogonium alga and obtained CFs of 49 500 and 98 386. The lower figure may be due to uncertainties in determining chlordane in solution and in the alga. Moore et al. (1977), using the planktonic alga Ankistrodesmus amalloides, obtained a very much lower CF of 5560, but even this species shows accumulation potential (EHC34).

Chlordane is a persistent compound (fra97). The half-lives in soil are 283 days to 3.8 years, in air 5.2 h to 2.2 days, in surface water 283 days to 3.8 years and in ground water 566 days to 7.6 years (howard 91 in fra97).
If released to soil, chlordane may persist for long periods of time. Chlordane is expected to be generally immobile or only slightly mobile in soil; however its detection in various groundwaters indicates that movement to groundwater can occur. Chlordane can volatize significantly from soil surfaces on which it has been sprayed particularly moist soil surfaces; however, shallow incorporation into soil will greatly restrict volatile losses (828 in DHC99). Although sufficient biodegradation data are not available, is has been suggested that chlordane is very slowly biotransformed in the environment which is consistent with the long persistence periods observed under field conditions.
The components of technical chlordane are relatively insoluble in water and are readily adsorbed onto soil particles. As a result, one of the characteristics of soil residues is that they do not migrate readily through the soil profile (Canada, National Research Council, 1974; von Rumker et al., 1974). In general, not more than 15% of the residues migrate below the cultivated layer (Canada, National

Research Council, 1974). As a result, residues are not likely to become a serious contaminant of the lower soil strata or deep water sources (Canada, National Research Council, 1974). The organic matter and moisture contents of the soil can affect the volatilization of chlordane components (Stauffer, 1977 in EHC34). The organic matter causes greater adsorption and thus reduces volatilization while soil moisture increases volatilization (Stauffer, 1977). Also, liquid formulations are more volatile than granular (Atallah et al., 1979 in EHC34).
Some volatilisation into air from treated soils, and some run-off into surface waters can take place (EHC34). Chlordane is fairly persistent in soil and sediments, especially in the form of its alpha- and gamma-isomers, which are, to a certain extent, translocated into crops grown on the soil (EHC34).

If released to water, chlordane is not expected to undergo significant hydrolysis, oxidation or direct photolysis. Adsorption to sediment is expected to be a major fate process. Sensitized photolysis in the water column may be possible (828 in DHC99). One important aspect of chlordane residues is that they accumulate in sediment. The fate and behaviour of chlordane was investigated in an isolated fresh water lake, previously free from pesticide residues (Oloffs et al., 1978). The lake was treated with technical chlordane at 10 µg/litre, and sediment samples were analysed for chlordane residues 7, 24, 52, 279, and 421 days after treatment. It was found that water residue concentrations declined rapidly. After 7 days, only 46.1% of the chlordane residue remained. After 421 days, residues were still detectable, but all levels were below 0.01% of the initial concentration. It was observed that chlordane residues moved quickly to the bottom sediment and persisted there. Mean residue levels in sediment were 35.29 µg/kg wet weight after 7 days and 10.31 µg/kg after 421 days (EHC34).

If released to the atmosphere chlordane will be expected to exist predominantly in the vapor phase (828 in DHC99). Chlordane is stable to light under normal conditions.

Three conversion products of gamma-chlordane were found in white cabbage and carrots, 4 weeks after application. One of the two metabolites isolated from white cabbage (35% of the total), was given the chlordene chlorohydrin structure. The other isolated metabolite (15% of the total) was assigned the dihydroxy-beta-dihydroheptachlor structure. The third metabolite was not identified. 1,2-Dichlorochlordene, oxychlordane, and photo-alpha-chlordane, as well as the parent chlordane compounds, were found in alfalfa after treatment of the soil with chlordane (Canada, National Research Council, 1974 in EHC34).
Oxychlordane (or 1,2-dichlorochlordene epoxide) is the common metabolite derived from both alpha- and gamma-chlordane. It has been found in the fat of pigs fed either of the isomers and in the milk and cheese from cows fed alfalfa treated with technical chlordane. According to some authors, alpha- and gamma-chlordane give rise to oxychlordane via the intermediate 1,2-dichloro-chlordene (Canada, National Research Council, 1974 in EHC34).
In experimental animals, chlordane is readily absorbed via the skin and through oral ingestion, and probably also following inhalation. It is readily distributed in the body, the highest levels being found in adipose tissue, followed by the liver. The distribution was found to be similar in the rat and the rabbit. The metabolism of chlordane, which is a complex mixture, has been largely elucidated. Several metabolites have been identified and species differences have been found. Oxychlordane is the most relevant animal metabolite, being more persistant and toxic than the parent compound (EHC34). Following a single, oral dose, elimination of chlordane was almost complete after 7 days in the rat. After long-term exposure, elimination from the body was slower (EHC34).
Chlordane is used almost exclusively as a soil insecticide to control soil pests such as termites (Canada, National Research Council, 1974). Thus, residues of chlordane are mainly present in this environmental compartment. In most temperate climates, only the two chlordane isomers generally persist (Canada, National Research Council, 1974). For example, in Nova Scotia, chlordane was applied at 5 kg/ha per year to sandy loam soil for 3 years. Fifteen years later, approximately 15% of the residues remained, the alpha and gammma isomers being the major components (US EPA, 1976a,b in EHC34).
The removal from sewage treatment plant is >90% (riwa, 1998).

Use, Exposure and emissions

Technical chlordane is a mixture of more than 26 components. The most important components are cis- (alpha) and trans (gamma)-chlordane (60-75%), heptachlor, cis- and trans-nonachlor, alpha-, beta- and gamma-chlordene. The primary metabolite of chlordane is oxychlordane (who, 1984 in bruhn 1998/EHC34).

The production of technical chlordane is strictly controlled and its composition varies within narrow limits (Canada, National Research Council, 1974). Chlordane is available in the USA in five basic formulations (von Rumker et al., 1974, IARC, 1979), oil solutions containing chlordane at 2 - 200 g/litre, and emulsifiable concentrates containing chlordane at 400 - 800 g/litre (EHC34).

Chlordane has been used for more than 35 years as a broad-spectrum contact insecticide, mainly on non-agricultural crops and on animals (EHC34). Chlordane was first prepared in the 1940s by exhaustive chlorination of the cyclopentadiene-hexachlorocyclopentadiene adduct (IARC, 1979 in EHC34). Chlordane is produced commercially by reacting hexachloro-cyclopentadiene with cyclopentadiene to form chlordane, which is then chlorinated to produce chlordane (IARC, 1979 in EHC34). Chlordane was first produced commercially in the USA in 1947 (IARC, 1979 in EHC34). Production in the USA, in 1974, amounted to 9.5 million kg (IARC, 1979 in EHC34). It was banned from general use except for termite control (since 1 July 1983; EHC34) and was banned from all use in 1988 (811 in DHC99). Currently, there are no approved uses for chlordane in the US (828 in DHC99). Chlordane is not produced in Europe nor has it ever been manufactured in Japan (IARC, 1979 in EHC34). In Japan, the only permitted use of the compound is for the control of termites. It is also used against wood-boring beetles and in ant baits. Both the amounts of chlordane produced and used have decreased considerably in recent years (WHO, 1982 in EHC34)). Chlordane is not allowed in the Netherlands (gre96/fra97). Chlordane is not allowed in Europe since 1981 (RIWA). In Germany chlordane is forbidden to be used on agricultural soil since 1971 and totally since 1988 (bruhn 1998).

Chlordane is on Annex 1 and 2 of the EU council regulation 2455/92 which prohibits all plant protection products containing chlordane as an active ingredient, to be used or placed on the market. Chlordane is also on the PIC list and in EC directives 76/769/EC and 79/117/EC (ISPRA, 2000).

Chlordane is a versatile, broad spectrum, contact insecticide and is used mainly for non-agricultural purposes (primarily for the protection of structures, but also on lawn and turf, ornamental trees, and drainage ditches) (von Rumker et al., 1974). Furthermore, it is used on corn, potatoes, and livestock. In 1978, a US EPA cancellation proceeding led to a settlement on contested uses. This settlement allowed for limited usage by crop, location, amount allowed, and maximum time interval for use (EHC34). In Canada, the use of chlordane is controlled under the Pest Control Products Act and it is used for the protection of structures, ornamental plants, lawns, and various crops (Canada, National Research Council, 1974 in EHC34). Chlordane is used as a non-systemic contact and stomach insecticide with some fumigant action. It is also used as an acaricide, a pesticide and a wood preservative. In addition, it is used in termite control and as a protective treatment for underground cables (811 in DHC99).

The spraying of chlordane leads to distribution to air and atmospheric transportation. This may explain the diffuse presence of chlordane in the environment (Kramer, et al, in fra97).

The main source of exposure of the general population is through residues in food. This is not a significant problem since chlordane is not normally used on food crops, and residues in food of animal origin are usually below accepted residue levels in various countries. Under normal circumstances, chlordane intake from air and water is insignificant. Chlordane has, however, been detected in the air of buildings where the compound has been used for termite and other insect control. Under occupational exposure conditions, both inhalation and skin contact are relevant, if adequate preventive and protection measures are lacking (EHC34).

Vulnerable use and vulnerable groups
Chlordane is mainly used as an insecticide for non-agricultural purposes. However chlordane is very persistent. Chlordane could also present a risk to agricultural workers applying the herbicide and in the production plant. Assumed is that these workers take the necessary precautions using and producing the substance. The fact that chlordane is found in human mother milk indicates that chlordane presents a specific risk to a vulnerable group: children.

Environmental concentrations
The detection of chlordane in remote atmospheres (Pacific and Atlantic Ocean; the Arctic) indicates that a long range transport occurs. It has been estimated that 96% of the airborne reservoir of chlordane exists in the sorbed state which may explain why its long range transport is possible without chemicals transformation. The detection of chlordane in rain water and its observed dry deposition at various rural locations indicates that physical removal through wet and dry deposition occurs in the environment (828 in DHC99).

Entry into the atmosphere occurs mainly through aerial applications of dusts and sprays, soil erosion by the wind, and volatilization from soil and water (Canada, National Research Council, 1974 in EHC34).
Few data are available on the routes of entry or the behaviour and fate of chlordane in aquatic systems. It can be assumed that not much originates from ground water since there is little leaching of chlordane. One possible source is surface run-off, but no studies have tested the extent of this assumption. Another source is rain; however, in two studies, chlordane levels did not exceed 2 - 3 ng/litre rain water (Bevenue et al., 1972a; US EPA, 1976a,b in EHC34).

Chlordane has been found as a residue in wild life and in human mothermilk.

Table 2 Occurrence in the environment of chlordane

Compartment	Year	Time	Location	Concentration	Unit	Reference (source)
Water			USA Sacramento river	0.006	µg/l	24 riwa, 1998
Wildlife biota	Trans		Canada rivers Salamander	0-7.1 av	Ng/g ww	41 riwa, 1998
Wildlife biota	Trans		Canada rivers Salamander Sexual glands	0.9-2.3 av	Ng/g ww	41 riwa, 1998
Wildlife biota	Trans		Canada rivers Salamander Liver	1.7-2.1 av	Ng/g ww	41 riwa, 1998
Wildlife biota	Trans		Canada rivers Turtle eggs	1.0-7.4 av	Ng/g ww	41 riwa, 1998
Wildlife biota	Cis		Canada rivers Salamander	0.4-13.9 av	Ng/g ww	41 riwa, 1998
Wildlife biota	Cis		Canada rivers Salamander Sexual glands	3.6-9.0 av	Ng/g ww	41 riwa, 1998
Wildlife biota	Cis		Canada rivers Salamander Liver	7.2-10.1 av	Ng/g ww	41 riwa, 1998
Wildlife biota	cis		Canada rivers Turtle eggs	1-131.9 av	Ng/g ww	41 riwa, 1998
Humans	Alpha		Canada mothermilk	Av, <0.03 med	Ng/g	42 riwa, 1998
Humans	delta		Canada mothermilk	0.21Av, <0.9 med	Ng/g	42 riwa, 1998
Humans	Alpha		Canada mothermilk fat	Av, <0.03 med	Ng/g	42 riwa, 1998
Humans	delta		Canada mothermilk fat	Av, <0.9 med	Ng/g	42 riwa, 1998

d.l= detection limit

Legal status

Chlordane is referred to in Directive 76/769/EEC including banned substances (shortlist derived from DG ENV).

Conclusion

Chlordane is used as a insecticide on food crops, is persistent and bioaccumulative and is found in biota (fish=food) and human mother milk. Exposure through food is very likely because the substance is persistent and found in food. Chlordane is also forbidden in the EU and the USA but still found in mother milk and wild life biota. These substances are prioritised as high concern.

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Chlordecone

The substance was selected for evaluation in the expert meeting because it is a very persistent chemical.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 2 and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1 Physico chemical properties of chlordecone

Water solubility	< 1 mg/l (fra97) 7.6 mg/l (gre in DHC99) 1 - 2 mg/litre (Orndorff & Colwell, 1980b in EHC43)
Henry coefficient	2.5 E-8 Pa.m3/mole (gre in DHC99)
Vapour pressure	< 0.0000003 mm at 20 degree C (fra97) <3 x 10-7 at 25 °C (EHC43)
Log Kow	4.5 (gre in DHC99)

Chlordecone (Kepone) is a tan- to white-coloured solid (EHC43).
Chlordecone is poorly soluble in water.
DT50 is 3.8 to 46 year (gre in DHC99).
Chlordecone is an extremely stable compound and it is not expected to be degraded in the environment to any significant extent. However, there have been reports of trace amounts of monohydro chlordecone being found (Carver et al., 1978, Orndorff & Colwell, 1980b in EHC43), but the mechanism of its formation is not clear. Solar irradiation of chlordecone in the presence of ethylenediamine will result in 78% degradation after 10 days, but no study of the degradation products or their toxicity has been undertaken (Dawson, 1978 in EHC43).
Laboratory and field observations indicate that chlordecone does not volatilize to any significant extent (Dawson, 1978 in EHC43).
Chlordecone has a high affinity for soils and sediments such that, at equilibrium in the environment, residue levels in particulate matter will be 104 - 105 times that in any surrounding water (Dawson, 1978 in EHC43). Consequently, sediments act as sink for chlordecone-contaminated water and soils provide a sink for most atmospheric contamination (EHC43).
Data on the bioconcentration of chlordecone are given in Table 2. It should be noted that none of the exposures were representative of realistic environmental levels. Bioaccumulation in detritus, such as decomposing Spartina cyanosuroide, was demonstrated by Odum & Drifmeyer (1978 in EHC43). As detritus is a major energy source in aquatic environments, this could represent an important entrance for chlordecone into aquatic food webs. Both aquatic invertebrates and fish bioaccumulate chlordecone to very high levels. Depuration is slow in fish, thus residues tend to be high. Levels of chlordecone accumulated in edible fillets were almost the same as the whole body concentrations in sheepshead minnows and spot; therefore one of the largest residue reserves in contaminated fish is in the edible portion (Bahner et al., 1977 in EHC43). No data are available on the bioconcentration of chlordecone by terrestrial organisms.

Table 2 Bioaccumulation of chlordecone

Organism	Bioconc. Factor (BCF)	Exposure concentration (µg/litre)	Time	Reference
algae, unicellular	230-800	100	24 h	Walsh et al. (1977) in EHC43
Oyster (Crassostrea Virginica)	9354 9278	0.03 0.39	19 d 21 day	Bahner et al. (1977) in EHC43
grass shrimp (Palaemonetes pugio)	698 (425-933)	12-121	96 h	Schimmel & Wilson (1977) in EHC43
grass shrimp (Palaemonetes pugio)	5127	0.023	28 day	Bahner et al. (1977) in EHC43
grass shrimp (Palaemonetes pugio)	11425	0.4	28 day	Bahner et al. (1977) in EHC43
Spot (Leiostomus xanthurus)	3217	0.029	30 day	Bahner et al. (1977) in EHC43
spot (Leiostomus xanthurus)	1120	1.5	96 h	Bahner et al. (1977) in EHC43
Fathead minnow (Pimephales promelas)	16600	0.004	56 day	Huckins et al. (1982) in EHC43
Sheepshead minnow juv 21-day (Cyprinodon variegatus	1800	0.041	Life cycle test	Goodman et al. (1982) in EHC43
Sheepshead minnow, juv 42-day (Cyprinodon variegatus)	2400	0.041	Life cycle test	Goodman et al. (1982) in EHC43
Sheepshead minnow adult male (Cyprinodon variegatus)	3900	0.041	Life cycle test	Goodman et al. (1982) in EHC43
Sheepshead minnow adult female (Cyprinodon variegatus)	3700	0.041	Life cycle test	Goodman et al. (1982) in EHC43
Sheepshead minnow embryos (Cyprinodon variegatus)	2900	0.041	life cycle test	Goodman et al. (1982) in EHC43
sheepshead minnow juvenile progeny (Cyprinodon variegatus),	2400	0.041	life cycle test	Goodman et al. (1982) in EHC43

Chlordecone accumulation in an estuarine food chain (composed of green algae, oysters, mysids, grass shrimps, sheepshead minnows, and spot) occurred at concentrations as low as 0.023 µg/litre (Bahner et al., 1977 in EHC43). When oysters were fed chlordecone-contaminated algae, the maximum overall accumu-lation and transfer of chlordecone (or "food-chain potential") from water to algae and then to oysters was 2.1 (Bahner et al., 1977 in EHC43). However, the transfer potential (transfer from one trophic level to the next) from algae to oysters was only 0.007; therefore, transfer of chlordecone from algae to oyster and retention in oyster were inefficient. When spot were fed mysids that had eaten chlordecone-contaminated brine shrimp, the food-chain potential from water to brine shrimp to mysids and finally to fish ranged from 3.9 to 10.5.The transfer potential from shrimp to mysids was 0.53 and from mysids to spot, 0.85. This indicated that much of the chlordecone was being transferred through the trophic levels (EHC43)

Chlordecone is readily absorbed following ingestion by animals and human beings. It is also absorbed following inhalation and dermal exposure. It is widely distributed in the body; accumulation occurs mainly in the liver. The half-life in the body is of the order of several months and excretion is slow, mainly via the faeces (EHC43). The compound had a long biological half-life and disappeared more slowly from the liver that from other tissues. Excretion occurred mainly in the faeces, a total of 66% of

the dose being removed in the faeces and 2% in the urine in the 84 days following administration (EHC43). Excretion of chlordecone by the gastrointestinal tract, in addition to the biliary route, occurs in rats as well as human beings (Boylan et al., 1979 in EHC43).
Chlordecone presents a major hazard for aquatic ecosystems because of its stability and persistence in sediments, its bioaccumulation in food chains (EHC43). Early reports did not include any evidence of chlordecone degradation in the natural environment (Dawson, 1978; Geer, 1978 in EHC43), but, in a more recent study, microbial action has been shown to transform chlordecone into monohydro and possibly dihydrochlordecone (Orndorff & Colwell, 1980a in EHC43).

Use, Exposure and emissions
Technical grade chlordecone contains from 88.6% to 99.4% chlordecone (Blanke et al., 1977 in EHC43), 3.5 - 6.0% water (Dawson, 1978 in EHC43) and 0.1% hexachlorocyclopentadiene. It has been formulated as a wettable powder (50% chlordecone), emulsifiable concentrates, granules, and dust (Information Canada, 1973 in EHC43). Its production in the USA was discontinued in 1976; information about its production elsewhere is lacking (EHC43). However, a year later it was reported that a French company was considering the establishment of production facilities in France (Anonymous, 1978b in EHC43), but no further information on this proposal is available (EHC43).
Gilbert & Giolito (1952) first reported the synthesis of chlordecone in 1952. Commercial production in the USA started in 1966 (IARC, 1979 in EHC43).
Chlordecone is manufactured by the condensation of 2 molecules of hexachlorocyclopentadiene in the presence of sulfur trioxide, followed by hydrolysis to the ketone. It is also produced during the synthesis of mirex and is a contaminant of technical grade mirex. From the 1950s until 1975, some 1,600,000 kg of chlordecone were produced in the USA, of which between 90% (Sterrett & Boss, 1977 in EHC43) and 99.2% (US EPA, 1976b in EHC43) was exported to Africa, Europe, and Latin America. The bulk of the remainder,12 000 - 70 000 kg (US EPA, 1976b) was used in ant and cockroach traps in the USA or, after 1978, stored until it could be disposed of safely. It has been reported that most of the chlordecone exported was used in the manufacture of kelevan (Cannon et al., 1978 in EHC43).
Chlordecone is used as a fungicide and insecticide (gre96; fra97). Chlordecone is also used for wood preservation (RIWA, 1998). Chlordecone was furthermore used as a base material in the manufacture of the insecticide kelevan (EHC43). Chlordecone has been used extensively in the tropics for the control of banana root borer (Anonymous, 1978a; Langford, 1978 in EHC43). It is regarded as an effective insecticide against leaf-cutting insects, but less effective against sucking insects (Information Canada, 1973 in EHC43). It can be used as a fly larvicide, as a fungicide against apple scab and powdery mildew (Information Canada, 1973 in EHC43), and to control the Colorado potato beetle (Motl, 1977 in EHC43), rust mite on non-bearing citrus, and potato and tobacco wireworm on gladioli and other plants (Suta, 1978 in EHC43).

Airborne chlordecone has been known to spread 60 miles from a point source (Feldmann, 1976 in EHC43), and the potential exists for further dispersion of fine particles (Lewis & Lee, 1976 in EHC43). At present, exposure via drinking water does not present a health hazard with the possible exception of that in the Hopewell area. Values quoted for the lower James River ranged from 0.1 to 10 µg/litre (Suta, 1978 in EHC43).
Two major sources of chlordecone exposure for infants are insect traps and human milk. The USDA (1977a in EHC43) has reported that of 56 cases of non-occupational exposure to chlordecone, 52 were children under the age of 5, and all but 9 of these had come into contact with insect traps. This is understandable as children of this age group are fairly inquisitive and their activity areas are likely to overlap target areas for ant and cockroach traps. The same study also cited exposure of 2 adults and 2 persons of unspecified age (EHC43).
Since tobacco plants were treated with chlordecone, this may have also represented an exposure route, but again no residue data are available (EHC43).

The workers in a chlordecone plant and the area around it were exposed to extremely high concentrations of chlordecone dust. High volume air samplers (Pate & Tabor, 1962 in EHC43), 200 m from the plant, recorded chlordecone levels as high as 54.8 mg/m3, which constituted 50% of the total

particulate load. Lower concentrations of chlordecone were detected in the air 25 km away from the plant. Concentrations of chlordecone dust within the plant were not monitored, but levels reaching 11.8 mg/litre were found in blood samples of workers from the LSP (Heath, 1978 in EHC43).Illness was found in 76 of the 133 current and former workers of the plant examined. It was found that the blood levels of workers who were ill, averaged 2.53 mg/litre, whereas the average level in workers not reporting ill was 0.60 mg/litre (Heath, 1978 in EHC43).
In the past, the release of copious quantities of chlordecone dust from production facilities has represented a major source of environmental and human contamination. It has been suggested that chlordecone emissions from the Hopewell plant "were of a fine particle size having a long residence time in the atmosphere" (Lewis & Lee, 1976 in EHC43).

Vulnerable use and vulnerable groups
Exposure of the general population through its normal use can be regarded as minimal and is mainly related to residues in food. Poisoning amongst workers and severe contamination of the surrounding area and rivers have occurred where manufacture and formulation were carried out in a careless and unhygienic manner. The exposure of people living near these plants must have been considerable. Small children may be exposed through playing with insect traps containing chlordecone and human milk (EHC43).
When used as a herbicide chlordecone presents a considerable risk. Furthermore chlordecone is very persistent. Chlordecone could also present a risk to employees of the producing production plants. It is not clear in how far the substance is still used and produced.

Environmental concentrations
Actual levels of chlordecone in natural waters are extremely low, because most of the chlordecone is transferred rapidly to sediments (EHC43)
With the exception of contamination in the James River system, very little information is available on chlordecone residues in water. Sampling after the closure of the Life Science Plant revealed chlordecone levels of 1 -4 µg/litre in Bailey Creek, 0.1 µg/litre in the Appomattox River, and 0.3 µg/litre in the James River and at the mouth of Bailey Creek (Smith, 1976a in EHC43). Chlordecone was not detected (limit of determination 0.01 mg/kg) in samples taken from the James River several months after the plant was shut down (Huggett et al., 1977 in EHC43). However, it was detected periodically in the water table of Hopewell at levels as high as 3.4 µg/litre but typically 0.1 µg/litre (Dawson, 1978 in EHC43) and was also detected in the New York water supply of the Great Lakes Basin by Suta (1978 in EHC43). Residues as high as 0.21 µg chlordecone/litre have been reported in runoff from a banana plantation in Guadeloupe (Snegaroff, 1977 in EHC43).
Sediment levels were as high as 10 mg/kg in Bailey Bay, and it has been estimated that as much as 47,000 kg of chlordecone lie on the bottom of the James River (Chigges, unpublished data, 1977 in EHC43). Soil residue levels in Hopewell ranged from as high as 10,000 to 20,000 mg/kg near the plant to 2 - 6 mg/kg at a distance of 1 km (US EPA, 1976a) and it was estimated (Anonymous, 1978 in EHC43) that 1000 kg of chlordecone lay within a 1 km radius of the plant. Most of the soils tested in Hopewell contained detectable levels of chlordecone with concentrations generally decreasing with increasing distance from the plant (Dawson, 1978 in EHC43).Chlordecone residues may be expected in sediments of waterways in the vicinity of other production-formulation facilities, but no data are available on this.
The US EPA (Anonymous, 1978a in EHC43) estimated that a field that had been treated with chlordecone (4.2 kg active ingredient/ha) should have a residue level of 100 mg/kg in the top 3 cm of soil, after application. Reports of actual determinations in soil are scarce, but the United Fruit Company (Anonymous, 1978a in EHC43) described a residue level of 15 - 25 mg/kg, 6 months after an application of 6.73 kg active ingredient/ha.Snegaroff (1977 in EHC43) reported soil residue levels of 9.5 mg/kg and a level of 0.135 mg/kg in sediments in streams neighbouring on a banana plantation in Guadeloupe.

The USA action levels for chlordecone residues in foods are 0.3 mg/kg for shellfish, 0.3 mg/kg for finfish, 0.4 mg/kg for crabs, and 0.01 mg/kg for banana peels (Suta, 1978 in EHC43).While the majority of shellfish taken from the polluted James river in 1976 contained less than the 0.3 mg/kg

action level of chlordecone, oyster and clam samples in certain areas contained 0.21 - 0.81 mg/kg, crab samples contained 0.45 - 3.44 mg/kg, and finfish samples 0.02 - 14.4 mg/kg. These data prompted a fishing ban on the James River (Shanholtz, 1976 in EHC43). In 1978, samples of spot, flounder, mullet, trout, and croakers from the James River contained chlordecone but in concentrations below the 0.3 mg/kg action level (Suta, 1978 in EHC43). In bluefish, one sample was above 0.1 mg/kg (0.2 mg/kg) (US FDA, 1977 in EHC43). The shellfish sampled in the same area contained chlordecone, but at levels that could not be reliably determined (Reuber, 1977 in EHC43). All crabs in the area contained chlordecone, but all levels were below the action level. In 1976, samples from the polluted Chesapeake Bay contained levels of 0.037 mg/kg for 75 finfish samples and 0.61 mg/kg for 11 crab samples, and levels in 3 samples of oysters and one sample of clams were non-detectable (US EPA, 1979 in EHC43).
Residues in Atlantic coast bluefish (66 samples) ranged from 0.01 to 0.06 mg/kg, with the higher concentrations found off the Virginia coast (Peeler, 1976 in EHC43). South Atlantic coastal fish were relatively free of chlordecone as only 1 out of 132 samples contained detectable levels (Reuber, 1977 in EHC43). Residues of chlordecone in edible plants have only been reported in New Zealand (Brewerton & Slade, 1964 in EHC43). No data are available in the literature for chlordecone residue levels in bananas (Suta, 1978 in EHC43). Chlordecone has been found in 9 out of 298 samples of human milk, but the detection limit was relatively high (1 µg/kg) (Suta, 1978 in EHC43). Samples were taken in the southern USA, and chlordecone residues were only found in areas that had received bait treatment for fire ants. To date, chlordecone contamination of human milk has only been reported in 9 samples (Suta, 1978 in EHC43) in the southeastern USA. However, relatively few samples have been tested for chlordecone.

Residue levels for phytoplankton in the James River were found to average 1.3 mg/kg (Huggett et al., 1977 in EHC43). Chlordecone residues were also found in several species of birds that inhabit the southeastern USA coast, such as the blue heron, mallard duck, coot, black duck, wood duck, herring gull, Canada goose, hooded mersanger, and the bald eagle (Dawson, 1978 in EHC43). Residue levels were as high as 13.23 mg/kg (Dawson, 1978 in EHC43), but typically between 0.02 and 2 mg/kg. Eggs from the bald eagles and the osprey in Virginia were also examined and were found to contain residue levels ranging from 0.14 to 0.19 mg/kg, and 0.06 to 1.5 mg/kg, respectively (Dawson, 1978 in EHC43).

Legal status
Chlordecone is listed on the OSPAR candidate list, the HELCOM priority list and referred to in Directive 79/119/EEC including restricted substances (shortlist derived from DG ENV);

Conclusion
Chlordecone may give an indication for high concern for breastfeeding infants and employees through occupational exposure. It is measured in the environment especially in the vicinity of production plants and application areas and considered as very persistent under environmental conditions. However it is not clear if this chemical is still used frequently. The final indication is: high concern based on the unknown production and use. When the production and use is proven to be low the final indication may be medium concern.

Chlordecone is also a high concern for wildlife. Because of its persistent and bioaccumulative properties it may cause longterm exposure and biomagnification in the foodchain.

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Di-n-butylphthalate (DBP)

The substance was selected to be evaluated in the expert meeting because it is a HPV chemical which is produced in more than 1000 tonnes/year.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 3 and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1 Physico chemical properties of Di-n-butylphthalate (DBP)

Water solubility	8-13 mg/l (gre96) 8-13/3.5 (ritsema 89 in fra97)
Vapour pressure	2.7E-05 (Staples, 1997)
Henry coefficient	0.27 Pa.m3/mole (gre96), 6.7E-04 (Staples, 1997)
Log Koc	2.7 (gre96) 2.7/3.8 in lake IJssel calc. (ritsema 89 in fra97)
Log Kow	4.6-4.9 (gre96) 4.6-4.9/4 (ritsema 89 in fra97) 4.72 (30 riwa, 1998); 5.38 (1 riwa, 1998)

DBP is poorly soluble in water with a very low volatilisation. BBP and DBP are low persistent to medium persistent chemicals. The log Koc indicates a strong sorption. The log Kow indicates a possible bioaccumulation (fra97). However DBP is bioconcentrated in organisms at low trophic levels (mussels) but are metabolized and excreted by fish, birds and mammals, primarily as phthalic acid and the mono-ester (temanord96 in sepa98).

The limit value in water is 33 µg/l (gre96).

The biodegradation half-lifes in soil are 2 –23 days, in air 7.4 h to 3.1 days, in surface water 1 to 14 days, in ground water 2 to 23 days (fra97). The DT50 is 14 days (30 riwa). Expected removal from sewage treatment plant >90% (riwa, 1998).

If released into water it will absorb moderately to sediment and particulates in the water column. The DBP will disappear in 3-5 days in moderately polluted waters and generally within 3 weeks in cleaner bodies of water. It will not bioconcentrate in fish since it is readily metabolized. If spilled on land it will absorb moderately to soil and slowly biodegrade (66% and 98% degradation in 26 weeks from two soils). DBP is found in ground water under rapid infiltration sites and elsewhere. It has been suggested that its tendency to form complexes with water-soluble fulvic acids, a component of soils, may aid its transport into ground water. Although it degrades under anaerobic conditions, its fate in ground water in unknown. If released to air DBP is generally associated with the particulate fraction and will be subject to gravitational settling. Vapour phase DBP will degrade by reaction with photochemically produced hydroxyl radicals (estimated half life 18 hr). Human exposure is from air, drinking water and food in addition to in the workplace (828 in DHC99).

Use, Exposure and emissions

Reported production volumes of DBP are 10000 to 50000 tonnes/year in the EU (CEFIC164 (p5)). DBP is used as a plasticizer and a softener (CEFIC 164 (u5)). DBP emissions amounts to 500-800 DI tonnes/year (DBP and BBP) (EKOrapport 1995 in DHC99).

DBP is a ubiquitous pollutant due to its widespread use as a plasticizer in plastics; for example it is widely used in PVC and nitrocellulose polyvinyl acetate. Other applications include carpet backing, hair spray, nail polish, and glue. It may contaminate food through its use as a plasticizer in coatings on

cellophane and from its use in links (813 in DHC99). It is also used in cosmetics, safety glass, insecticides, paper coatings, adhesives, elastomers and explosives. It is used as a solvent in polysulfide dental impression materials, solvent for perfume oils, perfume fixative, textile lubricating agent and solid rocket propellent. DBP may be released into the environment as emissions and in wastewater during its production and use, incineration of plastics and migration of plasticizer from materials containing it.

CSTEE, 1998 collected a number of determinations of phthalates leachate from toys. From these leach values they used the highest emission rates as worst-case. The leached amounts of phthalates are calculated per area and time and for DBP the maximum emission rates are estimated at 7 µg/10 cm^2 (max. emission rate of DEHP 610 µg/10 cm^2). Assuming an exposure period of 6 hr; a product surface area, 10 cm^2; and body weight, 8 kg, the following intake dose of 0.81 µg/kg/day (DEHP 75 µg/kg/day) can be calculated (CSTEE, 1998).

Vulnerable use and vulnerable groups
DBP is used as solvent in cosmetics (hairspray), perfumes, in polysulfide dental impression materials and textile lubricating agent. Therefore adults as well as children can be exposed to DBP.

Environmental concentrations
DBP levels between 0.01 and 2 mg/l in river water in industrialized areas of Europe have been reported (temanord96 in sepa98).

Table 2 Occurrence in the environment of Di-n-butylphthalate (DBP)

Compartment	Year	Time	Location	Concentration	Unit	Reference (source)
Water	1993	-	Lakes and rivers	0.2 (max)	µg/l	Gre96
Water	-	-	Lake/river Ijsselmeer	0.2-0.4	µg/l	Ritsema 1989 in Fra97
Water			Ijsselmeer SPM	0.2-0.5	µg/l	(ritsema 89 in fra97)
Water			River Rhine	0.03-1.1	µg/l	Riwa, 1998
Water	1980		River Rhine	0.5	µg/l	32 riwa, 1998
Water			River Rhine	>10	µg/l	22 riwa, 1998
Water			Drentsche Aa	2.03	µg/l	87 riwa, 1998
Water			Groote Beek	1.25	µg/l	87 riwa, 1998
Water			Twente kanaal	1.42	µg/l	87 riwa, 1998
Water			Zuid-Willems kanaal	1.11	µg/l	87 riwa, 1998
Water			Almelo's kanaal	0.71	µg/l	87 riwa, 1998
Water			Ettenlandsch kanaal	1.14	µg/l	87 riwa, 1998
Water			Bergumer meer	1.11	µg/l	87 riwa, 1998
Water			Haringvliet	>10	µg/l	22 riwa, 1998
Water			Info-spec	0.04-0.8	µg/l	Riwa, 1998
Water	1973		Tama river (japan)	0.4-6.6	µg/l	2 riwa, 1998
Water			USA Delaware river	0.1-0.6	µg/l	2 riwa, 1998
Water				0.2-1	µg/l	Gre, bel97 in DHC99
Water				<0.05	µg/l	RIZA/RIKZ LOES in DHC99
Sediment				0.2-0.5	µg/kg	MTC in DHC99
Sediment				<22-114	µg/kg	RIZA/RIKZ LOES in DHC99
Suspended matter				98-1500	µg/kg	RIZA/RIKZ LOES in DHC99
Suspended matter	1986			0.2-0.9	mg/kg	Bel97 in DHC99
Wildlife biota			Biota	190-4380	µg/kg dw	RIZA/RIKZ LOES in DHC99

d.l= detection limit

Legal status

DBP is listed on the first list of priority substances as forseen under council regulation EEC/793/93, Commission Directive 93/10/EEC relating to materials and articles made or regenerated cellulose film intended to come into contact with foodstuffs.

Conclusion

DBP is a substance of high concern for human exposure. The substance is used in cosmetics and dental impression material. Therefore also vulnerable groups like children and medical patients can be exposed to DBP.

DBP is measured at many locations and is considered as slowly biodegradable under environmental conditions in sediments and soil. In mammals and higher DBP is rapidly metabolised and excreted.

In the environment aquatic organisms and especially sediment living organisms are exposed. However there is no evidence on endocrine effects in wild life.

References

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DDT

DDT (technical and p,p'-DDT and tetrachloro DDT) was selected to be evaluated in the expert meeting because it is a HPV chemical which is produced in more than 1000 tonnes/year.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 1 and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1: Physico chemical properties of DDT

Water solubility	0.0031-0.0034 mg/l (Verschueren 96 in fra97) p,p'-DDT
Vapour pressure	25.3 nPa at 20C (EHC83)
Henry coefficient	0.98 Pa.m3/mole (gre96; teunissen 96b in fra97)
Log Koc	5.63 (gre96; teunissen 96b in fra97) 4.9-6.2 p,p'-DDT (Gulden, 1998)
Log Kow	6.9 p,p'-DDT (de boer 95 in fra97) 7.48 p,p'-DDT (EHC83) 5.44-6.91 p,p'-DDT (Gulden, 1998) 4.73 - 6.22 p,p'-DDD (Gulden, 1998) 5.63 - 5.89 p,p'-DDE (Gulden, 1998)

All isomers of the compound DDT are white, crystalline, tasteless, almost odourless solids.
DDT, DDD and DDE are poorly to very poorly soluble in water (Gulden 1998). DDT is soluble in organic solvents and very soluble in animal fats (EHC83).
The compounds are lipofilic (log Kow 6-7) and known to accumulate in organisms. DDT and its metabolites are very persistent. On the basis of the log Koc (5.63) in combination with the poor solubility the compounds are primarily found in sediment (fra97).

The biodegradation half-lives in soil are 2- 15.6 years, in air 17.7 to 177 hours, in surface water 7 to 350 days and in ground water 16 to 31.3 years (howard 91 in fra97).

As is well known, DDT is released into the air by volatilization from soil surface and/or adhered to the dust surface. It is considered that DDT is transported by adsorption to particles in the air (Japan, 1997). DDT is directly transferred into river system by drifting or moved with soil particles or dusts into river by erosion or rainfall. DDT is difficult to be released in water because of strong adsorption with soil particles (Japan). DDT is strongly adsorbed to soil particles. It leaches very poorly in soil (Japan, 1997). DDT is stable under most of the environmental conditions including biological and abiological factors. One of the metabolites, DDE, is similarly or even more stable (Japan, 1997).
Because of high lipophilicity and hydrophobicity together with poor metabolism in living organisms, DDT and its stable metabolites are easily bio-accumulated in fat tissues of living organisms (Japan, 1997).
The physico-chemical properties of DDT and its metabolites enable these compounds to be taken up readily by organisms. Organisms can accumulate these chemicals from the surrounding medium and from food. In aquatic organisms, uptake from the water is generally more important, whereas, in terrestrial fauna, food provides the major source (EHC83).
The rates of accumulation into organisms vary with the species, with the duration and concentration of exposure, and with environmental conditions. In general, organisms at higher trophic levels tend to contain more DDT-type compounds than those at lower trophic levels (EHC83).
Experimental (dynamic flow system) bioconcentration factors of DDT in aquatic life (fish, daphnia and algae) are more than 10,000 for fish, 100,000 for daphnia and 5,000 - 60,000 for algae,

respectively. Bioconcentration of DDT occurs by indirect incorporation from food or via environmental water (Japan, 1997).
Due to the high lipophilicity and persistence, DDT, DDD and DDE are concentrated in aquatic organisms and accumulate in the food chain. Bioconcentration factors of BCF = 1900 - 330,000 (p,p'-DDT), BCF = 64,000 (p,p'-DDE) and BCF = 2700 – 81,000 (p,p'DDE) have been measured in fish. Because of the differing metabolic stability of DDT isomers and metabolites, DDE, contributes in increasing proportion to the total DDT contamination based on time (after exposure) and increasing level within the food chain (Gulden 1998).
Concentration factors can be misleading with compounds such as DDT when exposure is high. The compound is readily taken up and retained at very low concentrations. At high concentrations, no more material can be taken up because a plateau has been reached. The only meaningful way to assess the capacity of organisms to take up and retain DDT is by looking over a wide range of exposure levels. The low concentration factor quoted in Table 2 for earthworms, for example, reflects the high exposure rather than a low capacity for uptake and retention of DDT, because concentration factors are simple ratios between "exposure" and final concentration in the organism (EHC83).

Concentration factors for fish are generally higher than for their invertebrate prey (Table 2). It is now generally agreed that most of the DDT taken into aquatic organisms comes from the water rather than from their food (Moriarty, 1975). Again, the concentration factors can be misleading. Aquatic organisms take in a small proportion of ingested DDT. However, they retain a large proportion of the DDT, which has been absorbed into the body from the food. There has been some controversy in the past over explanations for higher accumulations of DDT at higher trophic levels in aquatic systems. It now seems clear that this is not due primarily to biomagnification up food chains but rather to a tendency for organisms at higher trophic levels to accumulate more DDT directly from the water (EHC83).

Terrestrial organisms do not live in a uniform medium surrounded by a relatively constant concentration of a chemical. Even soil organisms live in a medium with very variable concentrations of DDT or its metabolites at different levels of the soil profile or patchy distribution of the chemical. Some terrestrial organisms could be directly exposed to DDT during application of the insecticide, but most will be exposed to what remains of the DDT after application. Therefore, higher terrestrial organisms will accumulate DDT mostly from their food. The data in Table 2 are taken from controlled laboratory investigations. There is ample evidence from the field that DDT does accumulate in many organisms in different media. There is similarly evidence that the residues of DDT or its metabolites persist in organisms for long periods after exposure has ceased (EHC83).

Table 2. Bioaccumulation of DDT[a] (EHC83)

Organism		Duration	Exposure	Bioconcen-
Reference	(µg/litre)	tration factor		
Bacteria				
Aerobacter aerogenes	24 h	1.2	3736	Johnson &
Bacillus subtilis	24 h	0.676	4303	& Kennedy
Aerobacter aerogenes	4 h	0.64	10,639	(1973)
	4 h	0.64	1784	Johnson &
Bacillus subtilis	4 h	0.64	13 880	Kennedy
	4 h	0.64	1805	(1973)
Marine algae				
Cyclotella nana	2 h	0.7	37 600	Rice & Sikka
	2 h	0.7	58 100	(1973)
Isochrysis galbane	2 h	0.7	11 300	Rice & Sikka
	2 h	0.7	28 800	(1973)
Olisthodiscus luteus	2 h	0.7	4600	Rice & Sikka

	2 h	0.7	7000	(1973)
Amphidinium carteri	2 h	0.7	4300	Rice & Sikka
	2 h	0.7	9600	(1973)
Tetraselmis chuii	2 h	0.7	5200	Rice & Sikka
	2 h	0.7	6300	(1973)
Skeletonema costatum	2 h	0.7	31 900	Rice & Sikka
	2 h	0.7	38 400	(1973)
Diatom Cylindrotheca closterium	21 days	100	300 (1969)	Keil & Priester
Pond snail (Physa 5 sp.)	6 days	3.0	6000	Reinbold et al. (1971)
Freshwater mussel (Anodonta grandis)	3 weeks	0.62	3990[d] (1973)	Bedford & Zabik
Earthworm (Lumbricus terrestris)	4 weeks	17 000	0.47[d]	Davis (1971)
Water flea	3 days	2.0	1330 (1973)	Metcalf et al.
(Daphnia magna)	3 days	0.08	114 100 (1971)	Johnson et al.
Scud (Gammarus fasciatus)	3 days	0.081	20 600 (1971)	Johnson et al.
Glass shrimp (Palaemonetes kadiakensis)	3 days	0.1	5000 (1974)	Johnson et al.
Pink shrimp (Penaeus duorarum)	13 days	0.14	1500 (1970)	Nimmo et al.
Crayfish (Orconectes nais)	3 days	0.08	2900 (1971)	Johnson et al.
Mayfly larva (Hexagenia bilineata)	3 days	0.052	32 600	Johnson et al. (1971)
Mayfly larva (Siphlonurus sp.)	3 days	0.047	22 900 (1971)	Johnson et al.
Dragonfly nymph (Ischnura verticalis)	2 days	0.101	3500 (1971)	Johnson et al.
Dragonfly nymph (Libellula sp.)	2 days	0.079	910 (1971)	Johnson et al.
Midge larva (Chironomus sp.)	3 days	0.046	47 800 (1971)	Johnson et al.
Mosquito larva (Culex pipiens)	2 days	0.105	133 600 (1971)	Johnson et al.
Mosquito larva	3 days	2.0	110[d]	Metcalf et al.
(Culex quinquifasciatus)	3 days	0.9	74 [d]	(1973)
Mosquito fish	3 days	2.0	344 [d]	Metcalf et al.
(Gambusia affinis)	3 days	0.9	217 [d]	(1973)

Rainbow trout	12 weeks	0.176	21 363 [d]	Reinert et al.
(Salmo gairdneri)	12 weeks	0.137	43 158 [d]	(1974)
12 weeks	0.133	51 355 [d]	Reinert et al.	
			(1974)	
Brook trout	120 days	3 mg	0.64 [d]	Macek & Korn
(Salvelinus fontinalis)	/kg diet		(1970)	
120 days	0.003	8533 [d]	Macek & Korn	
		(1970)		
Pinfish	14 days	0.1	40 000 [d]	Hansen & Wilson
(Lagodon rhomboides)	14 days	1.0	11 020 [d]	(1970)
Atlantic croaker	14 days	0.1	12 500 [d]	Hansen & Wilson
(Micropogon undulatus)	14 days	1.0	12 170 [d]	(1970)
Fathead minnow	14 days	45.6 mg/kg	1.17 [d]	Jarvinen et al.
(Pimephales promelas)	14 days	0.5	85 400 [d]	(1977)
14 days	2.0	69 100 [d]	Jarvinen et al.	
112 days	45.6 mg/kg	1.33 [d]	(1977)	
112 days	0.5	93 200 [d]	Jarvinen et al.	
112 days	2.0	154 100 [d]	(1977)	
Tilapia	31 days	1.0	6800	Reinbold et al.
(Tilapia mossambica)	31 days	10	10 600	(1971)
Green sunfish	31 days	1.0	3900	Reinbold et al.
(Lepomis cyanellus)	31 days	10	4020	(1971)
15 days	0.1-0.3	17 500 [d]	Sanborn et al.	
			(1975)	
Chicken eggs	8 weeks	0.1	1.87 [d]	Foster et al.
fat 8 weeks	0.1	5.8 [d]	(1972)	
Broiler hen fat	6 weeks	1.0	10.3 [d]	Kan et al.
			(1978)	
White pelican	10 weeks	72	11.9 [d]	Greichus et al.
(Pelecanus erythrorhynchos)			(1975)	
Double-crested cormorant	9 weeks	0.95	236.3 [d]	Greichus &
(Phalacrocorax a. auritus)			Hannon (1973)	
American kestrel	11-16	2.8	103.9	Porter &
(Falco sparverius)	months		Wiemeyer (1972)	
Mule deere muscle	30 days	5 mg/day	122.8 ug	Watson et al.
(Odocoileus heminonus)	oral	/kg [d]		(1975)

a Unless specified otherwise, bioconcentration factors are based on whole body (WB) measurements.
d Calculated on a wet weight basis.

P,p'-DDT is not noticeably abiotically degraded in water. P,p'-DDT is dehydrochlorinated to p,'p'-DDE microbially and reduced to dechlorinated p,p'-DDD, converted in many steps to p,p'-DDA and further to p,p'-DDM (dichlorophenylmethane), p,p'-DBH (dichlorobenzohydrol) and p,p'-DBP (dichlorobenzophenone). Additional degradation products of p,p'-DDT are also known (Matsumura, 1985; Subba-Rao and Alexander in Gulden 1998). Biological degradation of p,p'-DDT is very slow. A half-life of 3 - 20 years has been estimated for soil and sediment. DDD and DDE are known to be even more persistent (Gulden, 1998). Humic material represents a major source of adsorptive capacity for DDT; the degree of sorption, however, is strongly connected with the degree of humification. Soil containing large amounts of humic material may not adsorb DDT as greatly as other soils where humification is more advanced. Wheatley (1965) estimated half-times for the loss of DDT applied to

soils. After surface application, 50% of DDT was lost within 16-20 days. The estimated time for the loss of 90% of surface-applied DDT was 1.5 to 2 years. With DDT mixed into the soil, 50% loss occurred in 5 to 8 years, and it was estimated that 90% of applied insecticide would be lost in 25-40 years (EHC83).

Albone et al. (1972) Investigated the capacity of river sediments, from the Severn Estuary, United Kingdom, to degrade DDT. p,p' -DDT (14C-labelled) was applied to sediments either in situ on the mud flats or in the laboratory. Sediment movement in the area of the in situ study was sufficiently small to neither bury nor expose the incubation tubes set into the mud. Incubation in situ over 46 days led to very little metabolism of DDT in the sediments. Some p,p' -TDE was produced, but the ratio of DDT to TDE was 13 : 1 and 48 : 1 in two replicate experiments. There was no production of extractable polar products; metabolism beyond TDE did not occur (EHC83). Incubation of the same sediments in the laboratory, over 21 days, led to much greater metabolism (ratios of 1 : 1.1 and 1 : 3.3, DDT to TDE, in replicate incubations) and the production of some unidentified, further breakdown products. Investigation of the microbial population of the sediment showed that some of the organisms were capable of degrading DDT; little metabolism appeared to take place in situ (EHC83).

Microorganisms, plants, insects and birds produce many of the DDT metabolites found in mammals and humans (Smith, 1991 in Gulden, 1998). Species differences have been found. In mammals DDT is either first reduced to dechlorinated DDD, and finally converted to DDA or metabolized to DDE through the removal of HCI, this holds true primarily for p,p'-DDT. DDE is significantly more stable than DDT and its other metabolites. DDT is primarily excreted as DDA. o,p'-DDT and its metabolites are more rapidly eliminated from mammalians than p,p'-DDT and its metabolites. DDT is not intensively metabolized by fish. The metabolites p,p'-DDE and p,p'-DDD have been found in fish. lt is possible, however, that a part of the metabolic activity seen in fish results from microbial activity.
Different organisms metabolise DDT via different pathways. Of the two initial metabolites, DDE is the more persistent, though not all organisms produce DDE from DDT. The alternative route of metabolism, via TDE leads to more rapid elimination (WHO, 1979). Much of the retained DDT and its metabolites are stored in lipid-rich tissues (EHC83). Because there is an annual cycle in lipid storage and utilization in many organisms, there is also a related annual cyclic pattern in the handling of DDT (EHC83).

The uptake and accumulation of DDT from the culture medium by microorganisms has been reviewed by Lal & Saxena (1982). All of the microorganisms studied showed some capacity to take up DDT from their growth medium, but the relative amount taken up varied greatly from species to species. Many species took up more than 90% of the DDT when exposed to concentrations ranging from 1 to 1000 µg/litre, whereas a few species took in only 0.5% of the available DDT. The concentration factors for DDT were variable but always high (EHC83).

Concentration factors are also variable in aquatic invertebrates. In all cases there is considerable uptake and retention of the DDT, though often as DDE or other metabolites rather than as the parent compound. The main point of interest is the ability of aquatic organisms to take up large amounts of the compound, over time, from water where DDT is present at very low concentrations, and to retain it (EHC83).

Eberhardt et al. (1971) applied radioactively labelled DDT, at a rate of 220 g/ha, to a freshwater marsh and followed the distribution of the compound and its metabolites. Concentration factors in ten species of plants varied between 5500 and 84 000. Various invertebrates showed high concentration factors: ramshorn snail (Planorbidae), 4700; backswimmer (Notonectidae), 10 000; crayfish (Orconectes immunis), 22 000; bloodworm (Tendipes), 25 000; and red leech (Erpobdella punctata), 47 000. Reporting earlier on the same study, over 15 months, Meeks (1968) showed that plants and invertebrates accumulated DDT to a maximum mainly within the first week after treatment, whereas vertebrates required longer to attain maximum residues. Residues of DDT in the surface water and suspended particles had fallen below detectable levels within 1 month. Residues in sediments stabilized at about 0.3 mg/kg after 9 months (EHC83).

A rise in temperature results in increased uptake of DDT by fish (Reinert et al., 1974 in EHC83). Increasing salinity decreases DDT uptake significantly, but has no effect on the uptake of DDE or TDE by fish (Murphy, 1970 in EHC83).
Birds with the highest residues of DDT or its metabolites were either terrestrial predators feeding on other birds or aquatic predators feeding on fish (EHC83).

There are marked geographical differences throughout the United Kingdom, related to usage patterns of DDT (Cooke et al., 1982 in EHC83), and also marked seasonal changes in residues. These seasonal changes appear to relate more to physiological changes in body composition, which occur with climatic and breeding seasons, than to the environmental availability of pollutants.

Some, though very little, DDT was detected in black bears by Benson et al. (1974). There was no evidence that the area had been directly sprayed with DDT (EHC83).

Use, Exposure and emissions
A typical example of technical DDT had the following constituents: p,p' -DDT, 77.1%; o,p' -DDT, 14.9%; p,p' -TDE, 0.3%; o,p' -TDE, 0.1%; p,p' -DDE, 4%; o,p' -DDE, 0.1%; and unidentified products, 3.5% (EHC83).
In the production process o,p'-DDT is also formed as a byproduct (15-20%) and in addition trace amounts of tris(4-chlorophenyl)methane (TCPM) is formed. TCPM-OH has been suggested to be a metabolite of TCPM (buser 1995 in SEPA 1998). TCPM and TCPM-OH are both persistent and bioaccumulating as indicated by high concentrations of these compounds in biota at higher trophic levels (SEPA 1998).
DDT has been commercially produced since the early 1940s and was used intensively worldwide as an agricultural insecticide and in the effective fight against insects carrying e.g. malaria, typhus, yellow fever and sleeping sickness. The production and use of DDT was forbidden in most industrial countries in the late '60s (FRG: 1972). DDT is, however, still produced and used in tropical countries, in particular to combat malaria. DDT was used in the German Democratic Republic (GDR) until the late 1980s. It is estimated that by the end of the 1960s approximately two million tons of DDT were distributed over large areas. With the distribution of technical DDT, primarily p,p'-DDT (up to > 70%) and o,p'-DDT (up to > 20%), and in small quantities the corresponding DDD and DDE isomers were released into the environment (Gulden 1998). DDT was produced in 60000 tonnes/year in 1965 (EHC83).
DDT is forbidden in the Netherlands (gre96) and in the EU. In Japan, after the registration as a pesticide in 1948, DDT had been used and sold as insecticide for agricultural and household use and as termite control agent. However, in 1971 it was prohibited to be used as insecticide for agricultural and household use in Japan because of long-term persistency in the environment. It was specified as Class I Chemical by Law concerning Examination and Regulation of Manufacture, etc. of Chemical Substance in 1981 and stopped manufacture, sale and use as termite control and all other uses (Japan, 1997). DDT has also been banned in Sweden for more than 20 years (SEPA 1998).
DDT is on Annex 1 and 2 of the EU council regulation 2455/92 which prohibits all plant protection products containing DDT as an active ingredient, to be used or placed on the market. DDT is also on the PIC list and in EC directives 76/769/EC and 79/117/EC. It is still permitted in Bhutan, Bolivia, Brazil, Ethiopia, Guinea, India, Kenya, Madagaskar, Mexico, Nepal, the Phillipines, Sudan and Slovenia (ISPRA, 2000).

Vulnerable use and vulnerable groups
Because of their lack of degradation, their resulting widespread persistence in the environment, their high acute toxicity to organisms at the base of food chains, and their high potential for bioaccumulation, DDT and its metabolites should be regarded as a major hazard to the environment. DDT should not be used when an alternative insecticide is available (EHC83).
DDT still presents a risk to vulnerable groups.

Environmental concentrations

Concentrations of DDT, DDD, and DDE in surface water are below the detection limit of 0.001 µg/l in 1991 and 1996 (fra97).

In the Fraunhofer report DDT (50-29-3) is measured in water with a median concentration of 0.0050 µg/l (mean 0.0057 µg/l) based on 1427 data from 47 stations (1221 data were above the determination limit). In sediment DDT (50-29-3) is measured with a median concentration of 5.5 µg/l (mean 150.64 µg/l) based on 1065 data from 57 stations (759 data were above the determination limit) (Fraunhofer, 1999).

DDT and its metabolites DDD and DDE were not found above detection limits of 10-50 ng/l in rivers in Germany (incl. Rhein, Neckar, Main, Donau, lnn, Satzach, Schmilka) in Baden-Württemberg (1992), Bayern (1994), Mecklenburg-Vorpommern (1993) and Sachsen (1994) (Gulden 1998).
In the Elbe River in Germany above (Zollenspieker) and below (Seemannshöft) the Hamburg harbor, 1993 measurements revealed no o,p'-DDE (8-10 water samples) (DL = 1 ng/1), o,p'-DDT only in 50% of the samples from Zollenspieker and o,p'-DDD in the majority of the samples. p,p'-DDT was found in almost every water sample, p,p'-DDD and p,p'-DDE in almost every sample above, but rarely bélow, the Hamburg harbor (Gulden, 1998).

In the German Elbe River from Schnackenburg (1991- 1993) and in the Weser River (1994) DDT and its metabolites rarely were found in detectable concentrations from max. 0.4 - 6 ng/l. Higher concentrations were measured in single cases in Thüringen (1995) and Sachsen-Anhalt (1995).
With a detection limit of 10 ng/l, the following measurements were made in Nordrhein-Westfahlen at 7 measuring stations (incl. Rhein , Sieg, Wupper, Ruhr, Lippe) some samples contained p,p'- and o,p'-DDT, -DDD and -DDE in typical concentrations of < 100 ng/l, in rare cases at higher concentrations (Wupper/Leverkusen-Rheindorf: p,p'-DDT up to 280 ng/l and o,p'-DDE up to 300 ng/1; Lippe/Wesel: o,p'-DDT up to 220 ng/l) (Gulden, 1998).

In 1994 in Brandenburg in Germany, at 5 stations samples were taken monthly (Schwarze Elster, Spree, Havel). P,p'-DDD and p,p'-DDE were found only in rare cases or just over the detection limit of 10 ng/l. In the majority of the cases, p,p'-DDT was not found in water measurable concentrations. Between October and November, however, in water from the Schwarze Elster at Liebenwerda and from the Spree at Cottbus, unusually high p,p'-DDT concentrations were found, 700-900 ng/l, and in November in water from the Havel at Potsdam even 1400 ng/l p,p'-DDT was measured (Gulden, 1998).

In 1994 no measurable concentrations (detection limit = 5 µg/kgDW) of DDT or its breakdown products were recorded from two measuring stations on the Rhine in Nordrhein-Westfahlen in Germany (Gulden, 1998).
In 1995 suspended particles from various rivers in Hessen were examined (20 measuring stations, detection limit = 1 - 5 µg/kg). o,p'-DDT was not detected at any station, in about 30% of cases o,p'-DDT at a max. of 4 µg/kg and o,p'-DDD with a max. of 85 µg/kg. p,p'-DDT (max. 88 µg/kg) was measured in 45% of the samples, p,p'-DDE (max. 68 µg/kg) and p,p'-DDD (max. 195 µg/kg) in about 95 to 75 % of the samples. The highest contamination was measured in the Schwarzbach River flowing into the Rhine (Gulden, 1998).

Samples taken from the ARGE Elbe from 8 measuring stations distributed along the Elbe River from Schmilka to Cuxhafen revealed the highest contamination near Magdeburg. The suspense particle concentration of DDT and its metabolites decreased strongly along the Elbe to Cuxhafen. p,p'-DDT (max. 980 µg/kg) was found at higher concentrations than o,p'-DDT (max. 503 µg/kg) at all measuring stations. In the lower Elbe, o,p'-DDD (max. 242 mg/kg) was found at higher concentrations than o,p'-DDT. In general contamination with p,p'-DDT was greatest, and among its breakdown products the

concentration of p,p'-DDD (max. 443 µg/kg) was higher than that of p,p'-DDE (max. 90 µg/kg) (Gulden, 1998).

In measurements made by the IKSR and DKRR at 10 measuring stations distributed along the Rhein from Village-Neuf, to Kampen, the highest contamination was at Koblenz. The maximum values were significantly lower than measured in the Elbe near Magdeburg. In general p,p'-DDT was found at higher concentrations than o,p-DDT and p,p'-DDD and p,p'-DDE in approximately equal concentrations but significantly lower concentrations than p,p'-DDT (Gulden, 1998).

Results from studies on sediments in the Hamburg harbor (32 measuring stations) were published by Gotz et al. in 1990. p,p'-DDD was found in all samples, p,p'-DDT, p,p'-DDE and o,p'-DDD in almost all samples, and in 50-70% of the cases o,p'-DDT and o,p'-DDE were additionally found. In general p,p'-DDT was found in the highest concentrations, its metabolites (p,p'-DDD, p,p'-DDE) and o,p'-DDT in low concentrations. In the majority, DDT, DDD and DDE were measured in concentrations < 100 µg/kg, in individual cases p,p'-DDT, p,p'-DDD and o,p'-DDT in concentrations > 100 µg/kg. Very high concentrations (> 1000 µg/kg) were measured in sediment frorn the Müggenburger Canal in the vicinity of a former DDT production facility (Gulden, 1998).

In 1992 sediments from numerous rivers (103 rneasuring stations) in Niedersachsen were tested. In the majority of cases, p,p'-DDT or its metabolites were found at concentrations >0.1 µg/kg, o,p'-DDT rarely in detectable concentrations, in 50% of the cases, however, its metabolite o,p'-DDD was found. The highest contamination with DDT and its metabolites (p,p'-DDD > 10 µg/kg) was found in the Weser River (Boffzen, Hajen, Petershagen) and in the Elbe River (Bleckede, Gorleben, Schnackenburg) (Gulden, 1998).
Sediments from the Elbe and its tributaries shortly before their junction with the Elbe, were studied in 1992 and sediments from the most important tributaries in 1994, by the GKSS Geesthacht. The metabolites p,p'-DDE, p,p'-DDD and o,p'-DDD were present in most samples at concentrations > 0.5 mg/kg, p,p'-DDT in about 50% of the cases and o,p'-DDE only in isolated cases, o,p'-DDT was not studied. In the Elbe River, high contamination (> 10 µg/kg) was found with DDT, DDD and DDE in the upper segment (Schmilka, Torgau) near Tangermünde and Lauenburg, the highest contamination (DDD > 100 µg/kg) near Dessau and Breithagen above Magdeburg. Of the tributaries, the Zwickauer Mulde, which flows into the Elbe near Dessau, and the white Elster near Leipzig were most contaminated. In general p,p'-DDE was found at lower concentrations than p,p'-DDD. In two cases (Elbe/Schmitka, Mulde/Trebsen) the concentration of p,p'-DDT was higher than that of its breakdown products DDD and DDE. This suggests an emission of DDT recently (Gulden 1998).

Based on the environmental monitoring data by the Japanese Environmental Agency, DDT and its analogue levels in water in Japan are below detection limit (<0.0003 ppb - <0.0001 ppb). In contrast, they are detected in bottom sediment (0.029 ppm in maximum) (Japan, 1997).

For example, according to the environmental monitoring in 1995 by the Environmental Agency, the level of such compounds in water was: p,p'-DDT 0.00001 ppm; p,p'-DDE 0.00001 ppm; and dieldrin 0.00001 ppm (less than detectable limit in all samples). The residues of DDT were detected in 7 out of 930 samples (30 kinds) of imported agricultural products, namely in pumpkin (detected/tested: 1/42), celery (2/8), wheat (1/27), soy beans (2/33), and immature peas (1/9). The maximum residue was 0.004 ppm as compared with the tolerance of 2 ppm (Japan, 1997).

DDT is very highly accumulative and is accumulated in human fat tissues and mother's milk, and is detected in fat tissues and blood of fetuses and newborns whose levels are, however, lower than those in mothers. No recent data on human monitoring for DDT residues are found in Japan (Japan, 1997).

Table 3. Occurrence in the environment of DDT

Compartment	Year	Substance	Location	Concentration	Unit	Reference (source)
Water	1992		Rivers and lakes Netherlands	0	µg/l	Gre96 (phernambucq 96 in fra97)

Compartment	Year	Substance	Location	Concentration	Unit	Reference (source)
Water	1992		North-sea coast Netherlands	0	µg/l	Gre96 (phernambucq 96 in fra97)
Water	1992		Wadden-sea Netherlands	0	µg/l	Gre96 (phernambucq 96 in fra97)
Water	1991 and 1996		Eijs, Harvss, Ijmdn, Lobptn, Schaar in the Netherlands	<0.001d	µg/l	(riza in fra97)
Water		p,p'-DDT		<0.001	µg/l	Mtc in DHC99
Water	1974	p,p'-DDT	Japan	Not detected in 55 samples (dl 2 –100)	Ppt	Japan, 1997
Water	1994	p,p'-DDT	Japan	Not detected in 17 samples (dl 10)	Ppt	Japan, 1997
Suspended matter	1994	p,p'-DDT	N-Rhein-West in Germany	< 5 (dl)	µg/kg	Gulden, 1998
Suspended matter	1995	p,p'-DDT	Hessen Germany	<1 - 88	µg/kg	Gulden, 1998
Suspended matter	1993	p,p'-DDT	Elbe Germany	<0.1 - 980	µg/kg	Gulden, 1998
Suspended matter	1994	p,p'-DDT	Rhine-IKSR Germany	<1 – 96	µg/kg	Gulden, 1998
Suspended matter	1994	p,p'-DDT	Rhine-DKRR, Germany	<2 -96	µg/kg	Gulden, 1998
Sediment	1974	p,p'-DDT	Japan	0.8-7.3	Ppb	Japan, 1997
Sediment	1994	p,p'-DDT	Japan	0.082-20	Ppb	Japan, 1997
Wildlife biota	1995		Mussels ES-D	0.7 av	µg/kg ww	RIKZ in fra97
Wildlife biota	-		Cod liver	8 (min) 47 (max)	µg/kg	De boer, 95 in fra97
Wildlife biota	1995		Mussels ES-D	53 av	µg/kg fat	RIKZ in fra97
Wildlife biota	1996		Mussels ES-D	0.1 av	µg/kg ww	RIKZ in fra97
Wildlife biota	1996		Mussels ES-D	8 av	µg/kg fat	RIKZ in fra97
Wildlife biota	1995		Mussels WRS	0.3 av	µg/kg ww	RIKZ in fra97
Wildlife biota	1995		Mussels WRS	17 av	µg/kg fat	RIKZ in fra97
Wildlife biota	1996		Mussels WRS	0.4 av	µg/kg ww	RIKZ in fra97
Wildlife biota	1996		Mussels WRS	25 av	µg/kg fat	RIKZ in fra97
Wildlife biota		p,p'-DDT	Molluscs, fish	<10	µg/kg fat	IVM in DHC99
Wildlife biota		p,p'-DDT	Birds, cormorant eggs	<10-9180	µg/kg fat	IVM in DHC99
Wildlife biota		p,p'-DDT	Birds, cormorant eggs	<0.2-171	µg/kg ww	IVM in DHC99
Wildlife biota		o,p'-DDT	Molluscs, fish	<10	µg/kg fat	IVM in DHC99
Wildlife biota		p,p'-DDT	Birds, cormorant eggs	<10-4840	µg/kg fat	IVM in DHC99
Wildlife biota		p,p'-DDT	Birds, cormorant eggs	<0.2-282	µg/kg ww	IVM93 in DHC99
Wildlife biota		o,p'-DDE	Molluscs	47-132	µg/kg	IVM in DHC99

Compartment	Year	Substance	Location	Concentration	Unit	Reference (source)
					fat	
Wildlife biota		o,p'-DDE	Molluscs	0-300	µg/kg fat	MTCin DHC99
Wildlife biota		o,p'-DDE	Fish	<10-600	µg/kg fat	IVM in DHC99
Wildlife biota		p,p'-DDE	Birds, cormorant eggs	6650-130000	µg/kg fat	IVM in DHC99
Wildlife biota		p,p'-DDE	Birds, cormorant eggs	388-4460	µg/kg ww	IVM93 in DHC99
Wildlife biota		pp'-DDD	Molluscs	40-160	µg/kg fat	MTC in DHC99
Wildlife biota		pp'-DDD	Molluscs	<10	µg/kg fat	IVM in DHC99
Wildlife biota		o,p'-DDD	fish	<10	µg/kg fat	IVM in DHC99
Wildlife biota		p,p'-DDD	Birds, cormorant eggs	<10-500	µg/kg fat	IVM in DHC99
Wildlife biota		p,p'-DDD	Birds, cormorant eggs	<0.2-29	µg/kg ww	IVM93 in DHC99
Wildlife biota		SDDT*	Baltic herring Sweden	4250	Ppb lw (lipid weight)	Jansson et al 1993 in sepa98
Wildlife biota		p,p'-DDE	Fin whales (Balaenoptera physalus)	260	Ppt lw	Aquilar d borrell 1994 insepa98
Wildlife biota		o,p'-DDT	Fin whales (Balaenoptera physalus)	390	Ppt lw	Aquilar d borrell 1994 insepa98
Wildlife biota		o,p'-DDT	Brain of Bald eagles in lake superior	0.05 and 0.18	Ppm ww	Kozie and anderson 1991 insepa98
Wildlife biota		p,p'-DDE	Brain of Bald eagles in lake superior	1.5 and 16	Ppm ww	Kozie and anderson 1991 insepa98
Wildlife biota		sDDT*	Ringed seal blubber	230	Ppm lw	Jansson 1993 in sepa98
Wildlife biota	1974	p,p'-DDT	Fish, Japan	0.9-1.3	Ppb	Japan, 1997
Wildlife biota	1994	p,p'-DDT	Fish, Japan	1-50	Ppb	Japan, 1997
Wildlife biota	1994	p,p'-DDT	Shell-fish, Japan	Not detected in 30 samples (dl 1 ppb)	Ppb	Japan, 1997
Wildlife biota	1974	p,p'-DDT	Birds, Japan	1 (dl) detected in 5/5 samples	Ppb	Japan, 1997
Humans		sDDE*	Mother milk in sweden	251**	Ppb lw	Lundgren and noren 1998 in sepa98
Humans			Mother milk in mexico	14000 (mean value) (500-162000)	Ppb lw	Waliszewski 1995 in sepa98
Sediment	1974	p,p'-DDT	Japan	0.8-7.3	Ppb	Japan, 1997

d.l= detection limit
* sDDT= sum of DDT, DDE and DDD.
Lw= lipid weight
** declined from 3200 ppb lw sDDT in 1972 to 250 ppb lw sDDT in 1992.

Legal status
DDT is listed on List I of Council Directive 76/46/EEC, furthermore the substance is listed on Annex 1A of the Third North Sea Conference, the OSPAR candidate list and referred to in Directive 76/769/EEC including banned substances (shortlist derived from DG ENV);

Conclusion

DDT is a insecticide used against sickness. Although it is forbidden in the EU, the USA and Japan, it is still used in some countries. Because it is very persistent, bioaccumulative and still widely found in the environment there is a high concern for exposure.

References

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Fraunhofer-Institute, 1999. Revised Proposal for a List of Priority Substances in the Context of the Water Framework Directive (COMMPS Procedure). Declaration ref.: 98/788/3040/DEB/E1

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Gülden, M., et al, (1998), Endocrinically active chemicals and their occurrence in surface waters, UBA-FB 97-068, Research report 102 04 279

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Di(2-ethylhexyl)phthalate (DEHP)

The substance was selected for evaluation in the expert meeting because it is a HPV chemical, which is produced in more than 1000 tonnes/year.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 3 and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1 Physico chemical properties of Bis(2-ethylhexyl)phthalate (DEHP)

Physical state	Liquid at normal temperature
Water solubility	0.003 – 1.3 mg/l at 20-25ºC (NCI98) 7.7*10E-8 Molair (sta97 in cefic165 (s1)) 0.3 mg/l (wam87 in DHC99)
Vapour pressure	$3.4 * 10^{-5}$ Pa at 20-25ºC (NCI98)
Log Kow	4.8 - 7.9 (NCI98) (wam87 and bel97 in DHC99) 5 (30 riwa, 1998)

Large ranges of values for water solubility and log Kow are available for DEHP. A probable explanation of this is that DEHP easily forms more or less colloid dispersions in water, which increase the amount DEHP in the water phase. The high log Kow of 7.9 is considered as most reliable. The low vapour pressure indicates a low evaporation rate from its pure state. Nevertheless, DEHP does evaporate from products. High concentrations are observed in indoor environment. The temperature is probably a key factor in this process. In the environment high peak temperature occur during sun light radiation. Up to 70ºC has been reported from cars exposed to sun. The vapour pressure will increase considerably at such temperatures (20 to 70ºC → 320 times higher vapour pressure). DEHP evaporated during high temperature peaks. Adsorption of DEHP on particles is enhanced by the presence of salt. This is consistent with reported decreased water solubility with increasing salinity. In a comparative laboratory study with salinity between freshwater and seawater the adsorbed amount increased 4-5 times when salinity increased from 0 to 29.5 promille (NCI98).

Photodegradation is the main degradation pathway in the atmosphere ranging between 0.32 and 1.07 days. Abiotic degradation in water is very slow. Aerobic biodegradation is considered as very variable. However, in one OECD 301 B Modified sturm test DEHP is shown to be “readily biodegradable” and also fulfilling the “10-days window criteria” (NCI98). Several more or less standardised biodegradation tests with aerobic sediment indicate slow degradation rate of DEHP. Laboratory studies on anaerobic degradation show that DEHP can be expected to be persistent in aquatic anaerobic environments. Also anaerobic conditions in municipal landfills seems to prevent degradation.
Studies on degradation in soil indicate half-lives of the parent compound under environmentally relevant conditions vary between 14 and 200 days. Volatilisation is not an important fate process and leaching to groundwater is probably negligible (0.51% of the total applied radioactivity over 111 days) (NCI98).

In fish, the bioconcentration factors based on radioactivity range between 114 and 1380. Equilibrium time vary between one and >56 days. The studies are performed with ^{14}C-labelled DEHP. The measured radioactivity refers to total ^{14}C-residues, and the concentration of DEHP may be overestimated. However since the main metabolisation product of DEHP is the reprotoxic MEHP these data are assumed to be valid. In invertebrates higher BCF values were measured of 2500 in mussels to 5400 in zooplankton.

After oral administration of DEHP (in the form of MEHP) it is rapidly and extensively absorbed from the gastrointestinal tract of rodents. Maximal plasma concentration of MEHP generally appears within 1-3 hours after dosing. In primates, including man, a smaller percentage of an oral dose is absorbed in comparison to rodents. Following oral administration of DEHP, DEHP and its metabolites are excreted rapidly (within 24 or 48 hours in urine and faeces, respectively). When inhaled a similar rapid excretion through urine takes place.
The metabolites of DEHP can be conjugated with glucuronic acid before excretion. Species differences have been observed as urinary glucuronides are absent in rats, low (15%) in hamsters, moderate (60-65%) in mice and high (65-80%) in primates including humans.
BCF is 100-10000 in aquatic organisms (wam87 in DHC99).

The DT50 in water is 10 (30 riwa, 1998).

Use, Exposure and emissions
DEHP is widely used as a plasticizer in polymer products, mainly PVC. Plasticizers have the function of improving the polymer material's flexibility and workability. The production volume in Western Europe was estimated to be 674,000 tpa in 1994 (NCI, 1999). Others report that the amounts of world DEHP production are increasing for recent 10 years, and reaches about one million t now (1/3 in U.S.A and 1/3 in Europe).

Phthalates are used in industry, especially for flexible PVC. The worldwide production of phthalates is estimated to be 2.7 million/year in 1980 (fra97). A small amount of the esters are used as industrial paints and insulation liquid of condenser.

DEHP is produced in the Netherlands (DSM Hoek van Holland, Exxon Rotterdam-Botlek), Germany (BASF, Hoechst, Bayer) (Riwa, 1998).

The expected removal from the sewage treatment plant is > 90%.
The wastes of plastics are burnt up or thrown away. When DEHP is burnt up at high temperature, it is completely burnt, whereas at low temperature a significant amounts of DEPH are released to the air (Japan, 1997).

CSTEE, 1998 collected a number of determinations of phthalates leachate from toys. From these leach values they used the highest emission rates as worst-case. The leached amounts of phthalates are calculated per area and time and for DEHP the maximum emission rates are estimated at 610 µg/10 cm^2. Assuming an exposure period of 6 hr; a product surface area, 10 cm 2; and body weight, 8 kg, the following intake dose of 75 µg/kg/day can be calculated (CSTEE, 1998).

The anticipated amounts of exposure of DEHP to humans are shown below. Patients given blood transfusion or medical treatment by use of plastic apparatus have the possibilities of exposure to DEHP (Japan, 1997).

Table 2 Exposure levels Bis(2-ethylhexyl)phthalate (DEHP)

Average exposure levels of Americans (1974)	300 g/man/day
Average exposure levels of English (1986)	20 g/man/day
Lung Tissues of the patients	13.4 - 91.5 mg/kg dry wt.

Vulnerable use and vulnerable groups

DEHP has been applied in many PVC products used as toys for children and in tubes and bags used for blood transfusion and other medical equipment.

In the environment DEHP is found as dispersion in the water phase and adsorbed to the sediment.

Environmental concentrations

It is monitored in Sweden, Norway, Denmark, UK, and Germany at 133 sites, 300 out of 320 measurements in the aquatic environment were beyond detection limit. In sediments it was monitored in UK, the Netherlands, Germany, Sweden, Norway, Golf of Mexico, Japan at 122 sites with 138 out of 146 measurements above the detection limit.

Table 3: Occurrence in the environment (ranges) Bis(2-ethylhexyl)phthalate (DEHP)

	Surface water conc (mg/l)	Sediment conc. (mg/kg dw)
90 Perc.	0.2	30.3
5 Perc.	0.000031	0.0585
Median	0.00061	2.5
Avg	0.0378	50.6
Number measurements	320	146

Table 4: Occurrence in the environment Bis(2-ethylhexyl)phthalate (DEHP)

Compartment	Year	Location	Concentration	unit	Reference (source)
Water		Rhine	0.1-0.9	µg/l	Riwa, 1998
Water		Rhine	1.1	µg/l	32 riwa, 1998
Water		Rhine	0.3	µg/l	33 riwa, 1998
Water		Rhine	1-<10	µg/l	22 riwa, 1998
Water		Meuse	1-<10	µg/l	22 riwa, 1998
Water		Haringvliet	1-<10	µg/l	22 riwa, 1998
Water		Info-spec	0.03-8.4	µg/l	riwa, 1998
Water	1983		<0.1-3.5	Ug/l	Wam87 in DHC99
Water			0.04-0.61	Ug/l	RIZA/RIKZ Loes in DHC99
Water	1986		0.3-50	Ug/l	Bel97 in DHC99
Ground Water	1984	Polluted ground water	20-45	Ug/l	Wam87 in DHC99
Sediment	-	River	<70	Mg/kg	Fra97
Sediment	1978	sediment	1-36	Mg/kg	Wam87 in DHC99
Sediment			106-2866	Ug/kg ww	RIZA/RIKZ Loes in DHC99
Suspended matter	1986		10-100	Mg/kg	Bel97 in DHC99
Suspended matter			590-27000	Ug/kg ww	RIZA/RIKZ Loes in DHC99
Air	-	Production plant	0-100	Mg/m3	(EHC158)
Air (city and polluted area)			<300	ng/m^3	Japan, 1997
Air (on the ocean)			0.5 - 5	ng/m^3	Japan, 1997
Rain water			<200	ng/m^3	Japan, 1997
Dropping materials		near plastic manufacturing plant	0.7 - 4.7	g/m^2/day dry wt	Japan, 1997
Wildlife biota	-	Canada eel	10	Mg/kg fresh weight	Fra97
Wildlife biota			<200-8040	µg/kg dw	RIZA/RIKZ Loes in DHC99

Legal status

DEHP is listed on the OSPAR candidate list and the Council Regulation 793/93/EEC 1.3. priority list.

Conclusion

DEHP is a substance of high concern for human exposure. Vulnerable groups that are exposed are children and medical patients.

DEHP is measured at many locations and is considered as persistent under environmental conditions in sediments.

In the environment aquatic organisms and especially sediment living organisms are exposed. However there is no evidence on endocrine effects in wild life.

References

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3,4-Dichloroaniline

The substance was selected to be evaluated in the expert meeting because it is a HPV chemical which is produced in more than 1000 tonnes/year.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 1 and the Human health relevant endocrine disruption data as category 2.

Chemical characteristics

Table 1 Physico chemical properties of 3,4-Dichloroaniline

Water solubility	600 mg/l (gulden98)
Vapour pressure	-
Log Kow	2.7 (gulden98)

The behaviour of 3,4-DCA in the aquatic environment has been summarized by crossland 1990. 3,4-DCA is soluble in water to 0.6 g/l (20C) and moderately lipophilic (log Kow 2.7). Accordingly 3,4-DCA has a relatively low tendency to bioaccumulate. Bioconcentration factors of 30 (kalsch 91 in gulden98) and 45 resp. have been measured for zebrafish and rainbow trout. 3,4-DCA adsorbs rapidly to sediment and soil particles and builds stable, probably covalent bonds with organic substances (Beyerle-Pfnur and lay 90 in gulden 98). 3,4-DCA is biologically degraded slowly in water and sediment; it is primarily abiotically degraded through photodegradation (gulden98).
Photolysis is the major degradation pathway in the hydrosphere (half life 4.1 to 6.3 days) (wol85 in CEFIC 53 (CW5)). 3,4-DCA is not readily biodegradable (CITI, 1992, Bayer report, 1987 in CEFIC53).

Use, Exposure and emissions
>10000 tonnes of 3,4- DCA is produced in the EU (CEFIC 53 (p7)). 3,4-DCA is an intermediate for chemical synthesis (closed system)(Cefic53 (u7)) .
3,4-Dichloroaniline is a metabolite of the herbicide linuron (gulden 98).
3,4-DCA is a byproduct from the production of herbicides (linuron, diuron, neburon and propanil), dyes and pharmaceuticals. About 42000-47000 tonnes of 3,4-DCA are produced worldwide, about 60% of that in the EU (livingston and willacy 91 in gulden98). The majority is used in herbicide production (gulden98). 3,4-DCA reaches the environment through industrial wastewater that has not been sufficiently purified and arises first through microbial conversion from 3,4-dichloro-1-nitrobenzol in industrial water treatment plants (grote 83 in DHC99). In addition 3,4-DCA arises through the biological degradation of phenylurea herbicides, like diuron and linuron.

Vulnerable use and vulnerable groups

In principal there is no use of 3,4-Dichloroaniline that means a risk for a vulnerable group. However since 3,4-DCA is the degradation product of herbicides like linuron and diuron, which are used as herbicides on food crops. This could mean that residues of 3,4-DCA are to be found on food (vegetables and fruit). However 3,4-DCA is not very persistent and degraded biologically.
Conclusion: no indication for high risk group or situation.

Environmental concentrations

3,4-DCA is hardly measured in the environment. On two places in Germany concentrations above the detection limit (min) have been measured. The observed concentrations vary from 0.09 to 0.14 µg/l.

Table 2 Occurrence in the environment of 3,4-Dichloroaniline

Compartment	Year	Time	Location	Concentration	Unit	Reference (source)
Water	1994		Baden-Wurttemberg	<0.05 max	µg/l	Gulden98
Water	1992-1994		Hessen	<0.05 (min) 0.14 (max)	µg/l	Gulden 98
Water	1994		Nordrhein-Westfahlen	< 1 (max)	µg/l	Gulden 98
Water	1994		Rheinland-Pfalz	<0.05 (min) 0.09 (max)	µg/l	Gulden 98
Water	1995		Thuringen	<1 (max)	µg/l	Gulden 98

d.l = detection limit

Legal status

3,4-Dichloroaniline is listed on the OSPAR candidate list and the Council Regulation 793/93/EEC 1.3 priority list;

Conclusion

3,4-Dichloroaniline is used as an intermediate in closed systems but is also formed as a metabolite of linuron and diuron, which are used on food crops. Exposure is expected indirectly through food. 3,4-Dichloroaniline is persistent but does not bioaccumulate and is found in the environment in water systems. The substance is classified as having high concern for exposure.

References

CEFIC53: Cefic, 1999, Information provided for the expert meeting on endocrine disruptors.

DHC99: Dutch Health Council (1999). Endocrine-disrupters in the Netherlands.

Gülden, M., et al, (1998), Endocrinically active chemicals and their occurrence in surface waters, UBA-FB 97-068, Research report 102 04 279

Hexachlorobenzene

The substance was selected to be evaluated in the expert meeting because it is a HPV chemical which is produced in more than 1000 tonnes/year.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 3 and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1 Physico chemical properties of hexachlorobenzene

Water solubility	0.006/0.11/0.004 mg/l 24ºC (Verschueren 96 in fra97) 0.005 mg/l (cefic134 (s5))
Vapour pressure	55 nPa at 20ºC (wor91) 2.3 mPa at 25ºC (cefic134 (v2))
Henry coefficient	10.3 Pa.m3/mole at 20ºC (ARS 95 in fra97) 131 Pa.m3/mole at 25ºC (CEFIC 1999)
Log Koc	6.62/6.55 (evers, 93 in fra97)
Log Kow	-0.969 at 21ºC (haz--) 5.5 (shiu 86 in fra97) 5.86 (1 riwa, 1998); 6.1 (2 riwa, 1998)

Hexachlorobenzene is practically insoluble in water, but is highly lipid-soluble and bioaccumulative. Technical grade HCB contains up to 2% impurities, most of which is pentachlorobenzene. Hexachlorobenzene has a low vapour pressure and therefore does not enter the atmosphere (EHC195).

The biodegradation half-lives in soil are 2.7 to 5.7 years, in air 156.4 days to 4.2 years, in surface water 2.7 to 5.7 years and in ground water 5.3 to 11.4 years (howard, 1991 in fra97).
Cefic information indicates dat HCB is persistent with half lives in soil from aerobic and anaerobic degradation ranging from 2.7 to 22 years (CEFIC, 1999). Half life in air is 1.76 year due to OH radicals (CEFIC, 1999).

The removal from aeration is 60-90%, slow sand-filtration up to 70% at 2m/day. Ozon up to 60% at 2-3 mg/l. Bankfiltration up to 50%. Expected good adsorption to coal (riwa, 1998).

BCF ranges from 3000 to >35000 (IPCS97 in cefic 134 (b3)) and indicate bioaccumulation. Field studies also indicate biomagnification although birds and mammals may excrete HCB.

Use, Exposure and emissions
In the seventies HCB was produced in 15000 tonnes/year in 1975 (gre96).
HCB is used as seed dressing and as fungicide for a variety of crops and is an additional product of the production of chlorinated aromates. It has also been used in a number of manufacturing processes such as aluminium and graphite rods. The use of HCB in such applications has now virtually ceased although it is possible that HCB may still be in use in some parts of the world (CEFIC, 1999).

At the end of the 70s the use of HCB is forbidden in agriculture in Germany and the Netherlands (bruhn 1998, fra97). In EC council regulation 2455/92 Annex 1 is referred that HCB is banned or severely restricted to certain uses by community legislation owing to their effect on health and the environment.

HCB is produced in Germany (Bayer). It is unknown whether this is still the case.

While HCB commercial production and use has been virtually eliminated in Europe. lt is still generated as a trace byproduct in a number of chlorination and combustion reactions. Today the major sources of HCB emissions are reported to be:

- Trace contaminant in certain pesticides.
- Emissions from chemical process such as production of perchloroethylene, chlorobenzenes and other chlorinated organics. (Today in Europe HCB formed in such manufacturing processes is separated and incinerated with a high degree of efficiency).
- Emissions from metals industries.
- Emissions from combustion processes.
- Volatilisation and leaching from landfills (CEFIC, 1999).

Vulnerable use and vulnerable groups

HCB is used on seed and soil before culturing although HCB also may be used on food crops. Because HCB is a trace contaminant in certain pesticides on food crops this could mean a certain concern. HCB could present a concern to agricultural workers applying the herbicide. Assumed is that these workers take the necessary precautions using the substance.
However HCB is persistent and bioaccumulates.

Environmental concentrations

The concentrations of HCB in fresh waters are below the detection limit of 0.001 µg/l. Concentrations in organisms have been found (fra97).
In the Fraunhofer report HCB is measured in water with a median concentration of 0.0050 µg/l (mean 0.0099 µg/l) based on 2354 data from 79 stations (1757 data were above the determination limit). In sediment HCB is measured with a median concentration of 2.88 µg/l (mean 42.89 µg/l) based on 1617 data from 124 stations (1465 data were above the determination limit).

Table 2 Occurrence in the environment of hexachlorobenzene

Compartment	Year	Time	Location	Concentration	Unit	Reference (source)
Water	1993		Lakes and rivers	0	µg/l	Gre96
Water	1996		Eijsd, Harvss, Ijmdn, Lobptn, Schaar	<0.001	µg/l	Fra97
Water	1991		Harvss, Lobptn	<0.001	µg/l	Fra97
Water	1991		Andijk	<0.02	µg/l	Fra97
Water			Rhine	<0.01	µg/l	6 riwa, 1998
Water			Lobith	0.002-0.01	µg/l	24 riwa, 1998
Water			Meuse	<0.01	µg/l	6 riwa, 1998
Water			Boezemwater	<0.01	µg/l	6 riwa, 1998
Water			Other waters	Not found < 0.1	µg/l	Riwa, 1998
Water	1988		Germany Rhine Bad Honnef	<0.01	µg/l	24 riwa, 1998
Water	1969-1973		Italy Tiber	1-10	Ppt	24 riwa, 1998
Suspended matter	1991		MALZN	$5^{E+}11$ av	µg/kg	Riza in fra97
Suspended matter	1992		MALZN	3.075 av	µg/kg	Riza in fra97
Suspended matter	1993		MALZN	1.8 av	µg/kg	Riza in fra97
Suspended matter	1994		MALZN	1.45 av	µg/kg	Riza in fra97
Suspended matter	1995		MALZN	1.425 av	µg/kg	Riza in fra97
Suspended matter	1991		VLSB	$5^{E+}11$ av	µg/kg	Riza in fra97

Compartment	Year	Time	Location	Concentration	Unit	Reference (source)
Suspended matter	1992		VLSB	2.275 av	µg/kg	Riza in fra97
Suspended matter	1993		VLSB	0.925 av	µg/kg	Riza in fra97
Suspended matter	1994		VLSB	0.625 av	µg/kg	Riza in fra97
Suspended matter	1995		VLSB	0.575 av	µg/kg	Riza in fra97
Suspended matter	1991		NWK	$7.5^{E+}11$ av	µg/kg	Riza in fra97
Suspended matter	1992		NWK	$2.5^{E+}11$ av	µg/kg	Riza in fra97
Suspended matter	1993		NWK	3.8 av	µg/kg	Riza in fra97
Suspended matter	1994		NWK	1.625 av	µg/kg	Riza in fra97
Suspended matter	1995		NWK	2.9 av	µg/kg	Riza in fra97
Suspended matter	1993		VLSB	1 av	µg/kg	Riza in fra97
Suspended matter	1993		DVB	1.5 av	µg/kg	Riza in fra97
Soil	-			60-200	Pg/m3	Cefic134 (ca1)
Wildlife biota	1995		Mussels ES-D	0.5 av	µg/kg ww	RIKz in fra97
Wildlife biota	1995		Mussels ES-D	355 av	µg/kg fat	RIKz in fra97
Wildlife biota	1996		Mussels ES-D	0.1 av	µg/kg ww	RIKz in fra97
Wildlife biota	1996		Mussels ES-D	8 av	µg/kg fat	RIKz in fra97
Wildlife biota	1995		Mussels WRS	0.14 av	µg/kg ww	RIKz in fra97
Wildlife biota	1995		Mussels WRS	9 av	µg/kg fat	RIKz in fra97
Wildlife biota	1996		Mussels WRS	0.1 av	µg/kg ww	RIKz in fra97
Wildlife biota	1996		Mussels WRS	6.9 av	µg/kg fat	RIKz in fra97
Wildlife biota	1996		Bot liver ES-D	8 av	µg/kg product	RIKz in fra97
Wildlife biota	1996		Bot liver ES-D	41 av	µg/kg fat	RIKz in fra97
Wildlife biota	1996		Bot liver WNZ	1 av	µg/kg product	RIKz in fra97
Wildlife biota	1996		Bot liver WNZ	- av.	µg/kg fat	RIKz in fra97
Wildlife biota	1996		Bot liver WRS	3 av	µg/kg product	RIKz in fra97
Wildlife biota	1996		Bot liver WRS	21 av	µg/kg fat	RIKz in fra97
Wildlife biota	1996		Bot ORS	1.5 av	µg/kg product	RIKz in fra97
Wildlife biota	1996		Bot ORS	11.4 av	µg/kg fat	RIKz in fra97
Wildlife biota	1996		Bot HK	4 av	µg/kg product	RIKz in fra97
Wildlife biota	1996		Bot HK	26 av	µg/kg fat	RIKz in fra97
Wildlife biota	1996		Schar TG1	1.9 av	µg/kg product	RIKz in fra97
Wildlife biota	1996		Schar CG	1.8 av	µg/kg product	RIKz in fra97
Wildlife biota	1996		Schar BM	1.5 av	µg/kg product	RIKz in fra97
Wildlife biota	1996		Schar TG2	1.3 av	µg/kg product	RIKz in fra97
Wildlife biota	1996		Schar DK	1.6 av	µg/kg product	RIKz in fra97
Wildlife biota	1996		Schar NL	1.6 av	µg/kg product	RIKz in fra97
Wildlife biota	1996		mussels	6.9-8	µg/kg fat	Mtc in DHC99
Wildlife biota	1996		mussels	0.1-0.14	µg/kg ww	Mtc in DHC99
Wildlife biota	1996		Fish	10-40	µg/kg fat	Mtc in DHC99
Wildlife biota	1996		Fish (bot, schar)	1.5-8	µg/kg ww	Mtc in DHC99
Wildlife biota	1993		Birds cormorant egg	<4-2300	µg/kg fat	Ivm in DHC99
Wildlife biota	1993		Birds cormorant egg	<0.04-120	µg/kg ww	ivm in DHC99
Wildlife biota	1996		Fish (rode aal)	1-110	µg/kg ww (%	rivo in DHC99

Compartment	Year	Time	Location	Concentration	Unit	Reference (source)
					fat 6-29)	
Humans	1986			2.5	Ppm	PTI96 in cefic134 (ch6)
Humans	1993			0.5	Ppm	PTI96 in cefic134 (ch6)
Food	1973		Cow's milk	6	Ppb	PTI96 in cefic134 (ch6)
Food	1973		Cow's milk	<1	Ppb	PTI96 in cefic134 (ch6)

d.l= detection limit

Legal status

HCB is listed on List I of Council Directive 76/46/EEC, Annex 1A of the Third North Sea Conference, the OSPAR candidate list, the HELCOM priority list and referred to in Directive 76/769/EEC including banned substances (shortlist derived from DG ENV);

Conclusion

HCB is used on seed and soil before culturing although HCB also may be used on food crops. However in the EU HCB is severely restricted. HCB is persistent, bioaccumulates and is found widely in the environment and in fish (food) and mother milk. Because this indicates exposure through food and mother milk, the substance is prioritised as having high concern.

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Lindane

The substance was selected to be evaluated in the expert meeting because it a HPV chemical which is produced in more than 1000 tonnes/year.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 2 and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1 Physico chemical properties of lindane

Physical state	Colourless, crystalline solid, faint to odourless
Water solubility	7.6 mg/l (gre96) 7.6 and 6.94 mg/l (shiu 90 in fra97) 8.52 mg/l at 25 C(cefic 57 (s3))
Vapour pressure	
Henry coefficient	0.2 Pa.m3/mole (gre96) (teunissen 96b in fra97)
Log Koc	2.97 (gre96) (teunissen 96b in fra97)
Log Kow	3.7 (gre96) (teunissen 96b in fra97) 3.72 (1 riwa, 1998) 3.5 (Cefic, 1999)

Lindane is poorly soluble in water but has a relatively low log Kow value. Therefore lindane accumulates poor to medium in organisms. Lindane will be primarily found in the water phase and not bound to the sediment. Lindane has a low volatility from water (fra97).

Beta HCH and alpha HCH are more persistent than gamma-HCH and are bioaccumulated in the food webs (Jansson 1993 in sepa98). The BCF of lindane in algae is 240 (average), in mussels from 150 to 350, in rainbow trout from 1200 to 2000 and the log BCF in vegetation is –0.41 (fra97).

The half-life of lindane in water is 28 to 60 days (gre96) (teunissen 96b in fra97). It is also reported that the DT50 in water is 1 year (riwa, 1998).

According to CEFIC information lindane is rapidly transformed into polar metabolites and an elimination half life of 0.3 to 4 days. Relevant accumulation and bioaccumulation is not expected. The dissipation half life of lindane in soil, based on reliable field studies, is about 1.4 to 40 days. The DT50 values in sediment are 40-48 days. Atmospheric residence time in air is 11.8 days using the Atkinson method (CEFIC, 1999).

The removal from sewage treatment by powder coal dosing is >90%, 1-12 mg/l, contact time 1 hour. Slow sand filtration 30% at 2 m/day, 3% at 5 m/day. Ozone 10-55%, 10-40 mg/l possibly up to 70%. Hyperfiltration >85%. Active Coal filtration >90%, 15 min contact time, 3000 l/g (estimated). Bank filtration up to 70%. Removal percentage by active coal > 90%, >85% by hyperfiltration, up to 70% by bank filtration, up to 55% by ozone and up to 30% by slow sand-filtration (riwa, 1998).

Use, Exposure and emissions

Lindane is an insecticide and the intended uses are only seed treatment and treatment of soil with subsequent incorporation into the top soil layer. All other uses, especially foliar spraying, are not supported by CIEL. The use restrictions to seed and soil treatment were self-imposed and voluntarily proposed by CIEL in order to minimise the evaporation of Lindane (CEFIC, 1999). Nevertheless, if not applied properly Lindane may be transported by long-range transport (SEPA, 1998). Furthermore

the crude product with higher levels of alpha and beta-HCH is still often in use in the developing countries.

The quantities of Lindane used world-wide have been reduced to a third during the last years. The production rate of gamma-HCH (Lindane) of CIEL- quality (purity >99.5 %) is about 900 metric tonnes per year (CEFIC, 1999).

In the Netherlands 29 tonnes lindane was used in 1985, 24.3 tonnes in 1988, 21 tonnes in 1991 and 19 tonnes in 1994 (Ordelman et al., 1993). The emission is 14 tonnes/year in 1994 (gre96). Lindane is used as an insecticide in agriculture and forestries, to protect wood and also as a veterinary and human medicine. The use of lindane is now limited in Germany (bruhn, 1998).
Erodation of lindane gives the most important contribution to the emission of lindane (>65%). After 1992 the permission for the use of lindane are strongly reduced. However in 1993 the use was hardly lower than in 1992 (Teunissen-Ordelman 1995 in fra97).

In Europe Lindane is produced in 5 –50 tonnes/year. It is produced in Germany (Shell agrar, France and Spain. The total production in Europe was 3000 tonnes/year in 1981 (RIWA, 1998). Other information reports that Lindane is produced only in one known plant in Europe (Rumania) (Fraunhofer, 1999).

The emission of HCHs is regulated under 84/491/EEC.

Vulnerable use and vulnerable groups
Because Lindane is on seed and soil before culturing and thus is not directly used on food crops. HCH could also present a risk to agricultural workers applying the herbicide. Assumed is that these workers take the necessary precautions using the substance.

The substance has been proposed for adoption in the priority list because of specific concern for drinking water suppliers (EUREAU) (fraunhofer report, 1999).

Environmental concentrations

In the Fraunhofer report (1999) lindane is measured in water with a median concentration of 0.0083 µg/l (mean 0.0168 µg/l) based on 11666 data from 546 stations (8260 data were above the determination limit). In sediment lindane is measured with a median concentration of 3.19 µg/l (mean 9.15 µg/l) based on 953 data from 53 stations (689 data were above the determination limit).
Lindane is found in freshwater in 1992 and 1993, in rain water in 1988, 1989, 1990/91 and 1992 and in shallow/deep ground water. No measurements have been done in marine water (Ordelman, 1996).

Table 2 Occurrence in the environment of lindane

Compartment	Year	Location	Concentration average (max.)	Unit	Reference (source)
Water	1993	Lakes and rivers	0.01 (0.01)	µg/l	Gre96
Water		North-sea coast	0.03 (0.05)	µg/l	Gre96
Water		Wadden-sea	0.01 (0.01)	µg/l	Gre96
Water	1996	Eijs	0.01	µg/l	Riza in fra97
Water	1991	Harvss	0.006	µg/l	Riza in fra97
Water	1996	Harvss	0.005	µg/l	Riza in fra97
Water	1996	Ijmdn	0.005	µg/l	Riza in fra97
Water	1991	Lobptn	0.003	µg/l	Riza in fra97
Water	1996	Lobptn	0.003	µg/l	Riza in fra97
Water	1992	VLSB	0.00815	µg/l	Riza in fra97
Water	1993	VLSB	0.00415	µg/l	Riza in fra97

Compartment	Year	Location	Concentration average (max.)	Unit	Reference (source)
Water	1994	VLSB	0.0048	µg/l	Riza in fra97
Water	1995	VLSB	0.0045	µg/l	Riza in fra97
Water	1992	NWK	0.00405	µg/l	Riza in fra97
Water	1993	NWK	0.00255	µg/l	Riza in fra97
Water	1994	NWK	0.002	µg/l	Riza in fra97
Water	1995	NWK	0.00185	µg/l	Riza in fra97
Water		Rhine	Max. 0.02	µg/l	6 riwa, 1998
Water		Rhine	<0.1	µg/l	22 riwa, 1998
Water		Meuse	<0.1	µg/l	22 riwa, 1998
Water		Dutch waterway (boezemwater)	0.04	µg/l	6 riwa, 1998
Water		Twente Kanaal	0.01	µg/l	87 riwa, 1998
Water		Ijsselmeer	<0.1	µg/l	22 riwa, 1998
Water		Haringvliet	<0.1	µg/l	22 riwa, 1998
Water	Oct. 1988	Yonne	19	ng/l	53 riwa, 1998
Water	Jan. 1988	Yonne	11	ng/l	53 riwa, 1998
Water	March 1991	Yonne	36	ng/l	53 riwa, 1998
Water	1984-1985	France Seine	0.01-0.05	µg/l	24 riwa, 1998
Water	1988	Germany Rhine Koblenz	0.001-0.012	µg/l	24 riwa, 1998
Sediment	Sept.1986	Yonne	22	µg/kg	53 riwa, 1998
Sediment	Oct. 1988	Yonne	<0.01	µg/kg	53 riwa, 1998
Suspended matter	1992	Salt waters	4.85 av	µg/kg	Riza in fra97
Suspended matter	1991	Salt waters	0.0041 av	µg/l	Riza in fra97
Suspended matter	1991	Salt waters	5E+11 Av	µg/l	Riza in fra97
Wildlife biota	1994	Red eel	199 av.	µg/kg fat	De boer 95 in fra97
Wildlife biota	1995	Driehoeksmossel	92 av.	µg/kg fat	Pieters 95 in fra97
Wildlife biota	1992	Cod liver North-sea	37 av.	µg/kg fat	Teunissen 95 in fra97
Wildlife biota	Sept.1986	Yonne mollusc	67-110	µg/kg	53 riwa, 1998
Wildlife biota	June 1987	Yonne mollusc	76	µg/kg	53 riwa, 1998
Wildlife biota	Oct.1988	Yonne mollusc	36	µg/kg	53 riwa, 1998
Wildlife biota	Apr. 1991	Yonne fish	45	µg/kg	53 riwa, 1998
Wildlife biota		Red eel	7-84	µg/kg ww	Rivo in DHC99
Wildlife biota		mollusc	0.009-0.1	mg/kg fat	Ivm in DHC99
Wildlife biota		fish	<0.02-8.4	µg/kg ww	Ivm in DHC99
Wildlife biota		Fish eel	0.02-0.2	mg/kg fat	Ivm in DHC99
Wildlife biota		Cormorant egg	5-58	µg/kg ww	Ivm in DHC99
Wildlife biota		Cormorant egg	0.1-1.4	mg/kg fat	Ivm in DHC99
Humans		Italian human milk	180	ppb lw beta HCH	Larsen 1994, johanssen 1994 in sepa98)
Humans		Norwegian human milk	33	ppb lw beta HCH	Larsen 1994, johanssen 1994 in sepa98)
Humans		Adipose tissue from Iran	730	ppb lw beta HCH	Burgaz 1995 in sepa98
Humans		Adipose tissue from Iran	18	ppb lw alpha HCH	Burgaz 1995 in sepa98

d.l= detection limit

Legal status

HCH is listed in List I of Council Directive 76/46/EEC, the Annex 1A of the Third North Sea Conference, the OSPAR candidate list, the HELCOM priority list and Priority pesticides list under Directive 91/414/EEC (and specified under Council Regulation 3600/92).

Conclusion

CEFIC, 1999 reports that the Lindane isomer B-HCH, which is a contaminant in the production of gamma HCH, may be of relevance regarding interference with the endocrine system, especially, that it might mimic an oestrogen. Furthermore they report that there are indications that not Lindane itself is, is an in vitro endocrine modulator (CEFIC, 1999). Therefore it should be checked in greater detail whether, the observed endocrine effects might have been caused by the B-HCH isomer.

Although no information available, there might be a concern for the use of drinking water.

Due to the persistent character of lindane and its potential to distribute through the environment, there might be a concern for aquatic organisms.

Lindane is used on seed and soil before culturing. Lindane is inherently biodegradable, bioaccumulates and is found widely in the environment and in fish (food) and mother milk. Lindane is also found in human tissues. Because this indicates exposure through food and mother milk. The substance is prioritised as having high concern.

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Linuron

The substance was selected for evaluation in the expert meeting because it is a HPV chemical, which is produced in more than 1000 tonnes/year.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 3 and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1 Physico chemical properties of linuron

Water solubility	81 mg/l at 25 deg C (Worthing 1987)
Vapour pressure	2.0 mPa at 24 deg C (Worthing 1987)
Log Kow	3.2 (Greve, 1996)
Henry constant coefficient	5400 Pa.m3.mol-1 (DHC, 1999)

Linuron is moderately soluble.

If released in water, linuron will adsorb to sediment and particulate matter in the water column. Linuron biodegrades in soil and therefore would be expected to degrade in aerobic sediment. Linuron degrades in anaerobic sediment, although no degradation rates are available (HSDB, 2000; 5). Photolysis will occur in surface waters. In one experiment, 43% of linuron exposed to sunlight outdoors was photolyzed in 24 days (HSDB, 2000; 3). While linuron biodegrades in soil, no linuron biodegraded when incubated with river water and sewage for 4 months (HSDB, 2000; 4). Linuron would not be expected to volatilize or hydrolyze. Bioconcentration in aquatic organisms should be minimal. After linuron (1 ppm) was added to each of three 1 m3 outdoor ecosystems, linuron concentration in the water columns declined exponentially over the 42-day experiment; the calculated half-lives ranged from 16 to 42 days (HSDB, 2000; 1). It was not determined what happened to the linuron. The half-life of linuron in Lake Balatan (Hungary) water was 10 weeks (HSDB, 2000; 2).

When applied to soil, linuron will adsorb moderately to soil particles and will remain primarily in the upper 2.5 cm of the soil. It should be photolyzed on the soil surface. Loss, due to biodegradation will also occur and will increase with increasing organic matter and moisture content of the soil and with increasing temperature (HSDB, 2000; 1,3,5). In most soils, at normal application rates, linuron degrades within 3-4 months (HSDB, 2000; 5). The dissipation of linuron was followed in field plots planted with potatoes and treated with linuron at 1 and 2 kg/ha (HSDB, 2000; 2). The disappearance of linuron was very fast and followed first order kinetics with a half life of 25 and 22 days, respectively, at the two application rates (HSDB, 2000; 2). After about 75 days, the residual concentration of linuron in the soil was constant, suggesting that a small amount of linuron is strongly adsorbed to soil colloids and not available to microbial attack (HSDB, 2000; 2). In test field plots in an onion growing area of Ontario, 64% of linuron had dissipated in 5 months from the organic soil (92% organic matter) (HSDB, 2000; 4). It was concluded that linuron residues did not accumulate in soil in onion farms of area growers (HSDB, 2000; 4).

Most evidence indicates that linuron's loss in soil is due to biodegradation (HSDB, 2000; 1-2). Biodegradation is mainly cometabolic(1-2). Evidence for linuron's biodegradation in soil is found in the fact that degradation proceeds far slower in soil sterilized by heat or with chemicals (HSDB, 2000; 1). Loss of linuron also correlates with soil respiration (HSDB, 2000; 3). The degradation rate also is correlated with soil organic carbon content, adsorptivity and clay content (HSDB, 2000; 1,3). The rate of biodegradation increases as the concentration of linuron in the soil decreases (HSDB, 2000; 4). The rate constant for the decomposition in soil increases as the square root of the concentration at high concentrations and linear with the concentration at low concentrations (HSDB, 2000; 2). At higher soil moistures and temperatures, linuron degrades faster (HSDB, 2000; 1,5). In most soils, at normal

application rates, linuron degrades within 3-4 months (HSDB, 2000; 1). The half-lives of 4 ppm linuron incubated at 25 deg C in 18 mineral soils ranged from 22 to 86 days (HSDB, 2000; 5). The degradation rate of linuron in a sandy loam soil at 20 deg C was 0.0080 per day (half-life 87 days)(HSDB, 2000; 3).

The half-life of linuron in a forest brown soil (6.4% OC, pH 5.5) with high biological activity at 24 deg C and an initial concentration of 21 ppm (dry wt) was approximately 85 days (HSDB, 2000; 1). In contrast to other studies, a study performed on four Hungarian soils, both sterilized and untreated, in which the soil was incubated for 10 weeks at different temperatures, concluded that degradation proceeds primarily by abiotic means (HSDB, 2000; 2). Chemical decomposition predominated at 10 deg C with the proportion of chemical to microbial degradation decreasing with increasing temperature and then increasing at 37 deg C. Mixed populations of microorganisms in river water or sewage did not biodegrade linuron during a period of 4 months (HSDB, 2000; 3). A possible explanation for linuron's biodegradation in soil and lack of biodegradation in solution may be a surface catalyzation as has been offered for a similar pesticide, diuron (HSDB, 2000; 4). Anaerobic sediment adapted to diuron dehalogenated linuron at the para position (HSDB, 2000; 5).

The persistence of linuron in soil is less than that of diuron and diuron is very persistent in soil. Linuron is microbially, or through plants, broken down into 1-(3,4-dichlorophenyl)-3-methoxyurea, 1-(3,4-dichlorophenyl)-3-methylurea, 1-(3,4-dichlorophenyl)-urea and 3,4-dichloroaniline (Gülden, 1998).

Use, Exposure and emissions
Linuron, is a herbicide with systemic action by inhibition of photosynthesis. The product is primarily used in cereals, sunflower, maize, potato, soybean and carrots. Additional indications are peas, Vicia faba, vegetables, celery, leek, vine, orchards, nursery, flax and ornamentals (CEFIC, 1999).

The usage of Linuron active ingredient in tonnes is 500 - 1000 in the EU per year. Main markets are France, ltaly, Netherlands, Spain, UK and Greece (CEFIC, 1999).

Vulnerable use and vulnerable groups
Because linuron is used as a herbicide on food crops this could mean a certain risk. However linuron is not persistent. Linuron could also present a risk to agricultural workers applying the herbicide. Assumed is that these workers take the neccessary precautions using the substance.

Environmental concentrations

Linuron is measured in Dutch lakes and rivers with a mean concentration of 0.01 µg/l (max 0.03 µg/l). In coastal waters of the North Sea a mean concentration of 0.01 µg/l (max 0.01 µg/l) was measured and in the Wadden sea a mean concentration of 0.05 µg/l (max 0.1 µg/l) (Greve, 1996).

Linuron was found only in isolated cases at the detection limit (0.03-0.06 µg/l) or slightly above in Germany. In the vast majority of the over 700 samples from 31 different measuring stations, linuron was not found at measurable concentrations (Gülden, 1998).

In an adult total diet study in the USA of the 1044 composites analyzed, 19 contained linuron (Yess, 1991 in HSDB, 2000). Whereas in another diet study the mean concentration of linuron in root vegetable composites was 0.0089 ppm (Winter, 1992). Linuron intakes estimated from the 1990 FDA Total Diet Study were 0.0027, 0.0010, and 0.0012 ug/kg/day for a 6-11 month old, a 14-16 yr male, and a 60-65 yr female, respectively (Winter, 1992 in HSDB, 2000).

In a pesticide screening survey, linuron was found in 4 out of 6970 produce samples, including 226 carrot samples (DL = 0.2 ppm) (Schattenberg, 1992). All the samples containing linuron were carrots

(Schattenberg, 1992). Of the 6,568 samples of domestic food and feed the FDA analyzed between 1982 and 1986, 30 (29 of them carrots) contained linuron with levels up to 0.5 ppm (Hundley, 1988). None of the 13,283 imported agricultural commodities contained linuron (Hundley, 1988). No linuron residues were found in 25 potatoes from farms in the USA at a detection limit of 0.1 ppm (Mattern, 1989).

Legal status
Linuron is listed on List 2 of Council Directive 76/46/EEC, Annex 1D of the Third North Sea Conference, the OSPAR candidate list, the Priority pesticides list under Directive 91/414/EEC (and specified under Council Regulation 3600/92).

Conclusion
Linuron is used on food crops. Human exposure may be expected by food but because these substances are not persistent, exposure is less likely. The substance is not bioaccumulative. The substance is prioritised as having high concern for exposure.

It should be checked whether there is indeed no or hardly any exposure.

References

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Maneb

The substance was selected to be evaluated in the expert meeting because it is a HPV chemical which is produced in more than 1000 tonnes/year.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 3 and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1 Physico chemical properties of maneb

Water solubility	<1 mg/l (gre96) <0.1 mg/l (4 riwa, 1998) 5 mg/l (keith in DHC99)
Vapour pressure	-
Log Kow	4.5 (30 riwa, 1998) 1.18 (ETU –0.85) (rivm mpc in DHC99)

Maneb is not very soluble in water. Is absorbs strongly to soil particles. Thus, despite its lengthy soil half life (60 days), maneb is not expected to contaminate ground water. It may enter surface waters if erosion of soil with absorbed maneb occurs. Maneb breaks down under aerobic and anaerobic soil conditions. Maneb's half-life in soil is four to eight weeks (829 in DHC99).
Maneb is hydrolysed in water with a half life of 0.2 days at pH 5.7, 5 days at pH 7 and 11 days at pH 8 (Ordelman, et al, 1993a). The removal from the sewage treatment plant is expected to be >90% by active coal (riwa). The limit value for maneb is 1 μg/l in the Netherlands (gre96).

Dithiocarbamates are generally instabile compounds. Important metabolites formed at all dithiocarbamates are carbon disulfide (CS2) and sulfur hydrogen (H2S) (Van Leeuwen, 1986 in Ordelman et al, 1993a). Furthermore a distinction can be made between the metabolites of the ethylenebisthiocarbamates (maneb and zineb) and the mono- and dialkyldithiocarbamates (metam natrium, thiram). From the ethylenebisthiocarbamates a joint 1,2,4-dithiazole (DIDT) is formed. DIDT is presumed to be the active compound. Other metabolites of the ethylenebisthiocarbamates are ethylenediisothiocyanate (EDI) and ethylenethiourea (ETU). ETU can be metabolised further. DIDT and ETU cannot be formed in the absence of oxygen (Vonk, 1975 in Ordelman, et al, 1993a). The metabolisation of DIDT to EDI does not occur in the presence of zinc- or ironsulfate (Vonk, 1975 in Ordelman, et al, 1993a) in water but does occur in the presence of copperoxide (Hunter and evans 1991 Ordelman, et al, 1993a).
The ethylene-bisdithiocarbamates (EBDCs) are noted for their instability in the environment. The application of heat can break these chemicals down into a number of metabolites. In addition to natural environmental processes that break down EBDCs, cooking of vegetables that are contaminated with these fungicides can also change them into different metabolites. Ethylene thiourea, an EBDC metabolite that is considered to be cancer-producing, is formed when maneb-treated vegetables are cooked (829 in DHC99).
Dithiocarbamates are partially chemically broken down, or metabolized, to carbon disulfide, a neurotoxin capable of damaging nerve tissue. EBDC residues in or on foods convert readily to ethylenethiourea (ETU), a known teratogen, during commercial processing or home cooking (829 in DHC99). Maneb degrades to ETU and other transients in water and soil. ETU is stable in water at pH 5-9 and under sunlight and the degradation of ETU on soil is not enhanced by sunlight radiation. Maneb degrades very rapidly under anaerobic aquatic soil conditions but ETU is relatively stable under these conditions. ETU is the degradate of major environmental concern. There are indications that ETU may leach and enter groundwater. However additional data are required to complete the groundwater assessment (gr99).

Expected is that the most dithiocarbamates will be metabolised quickly in the environment and in aquatic organisms and therefore will not spread to greater water systems and will not bioaccumulate or biomagnify (Ordelman et al 1993a).

Use, Exposure and emissions
Maneb is produced as follows: ethylenediamine and sulfurcarbon are combined together with a sodium hydroxide solution and decalcinated water (1 Ordelman et al 1993a). To the reaction product mangane sulfate is added after which maneb is formed (2 Ordelman, et al, 1993a).

Maneb is produced in more than 50 tonnes/year. It is produced in the Netherlands (Akzo chemicalsbv, Elf Atochem Agri bv), Germany (BASF) and France and Spain (riwa, 1998).
In the Netherlands maneb is produced in in Rotterdam in 10830 tonnes/year in 1986 with an emission of 1.4 tonnes/year to surface water (Nieuwe waterweg). After 1992 the emission is reduced by starting to use a sewage treatment plant (Riza 1992 in Ordelman et al, 1993a). Before the waste water enters the sewage treatment plant it is first lead through an installation to regain the mangane. It was expected in 1993 that the industrial emissions in 1995 would be reduced with 90% compared to 1985 (Riza 1992 in Ordelman et al, 1993a). At formulation about 8000 tonnes and about 10000 maneb are used in 1984 and 1986 resp. with an emission of 0.005 tonnes/year at one location and <0.001 tonnes/year from the sewage treatment plant at another location (Ordelman et al, 1993a). The turnover of maneb has decreased with 37% from 1985 to 1991 (Ordelman, et al, 1993a).
In 1985 1991 tonnes maneb was used in the Netherlands, 1748 tonnes in 1988, 1241 tonnes in 1991 and 852 tonnes in 1994 (Ordelman et al , 1993a).

Maneb is a fungicide used in vineyards, potatoes, vegetable and ornamental plants and sugarcanes (perkow and ploss 1996 in bruhn 1998; bui99 in CEFIC96(u11)).
Maneb is registered as a general use pesticide by the US EPA. In july 1987 the EPA announced the initiation of a special review of the ethylene bisdithiocarbamates (EBDCs), a class of chemicals to which maneb belongs. This special review was initiated because of concerns raised by laboratory tests on rats and mice. As part of the Special Review, EPA reviewed data from market basket surveys and concluded that actual levels of EBDC residues on products purchased by consumers are too low to affect human health. Maneb is a fungicide used on crops such as fruits, vegetables, seed crops, nuts, flax, and grains and non-food crop including ornamentals, lawns, turf. It's predominate use is with apples, potatoes, tomatoes and sweet corn where it is effective for controlling foliar fungal diseases. It is manufactured in numerous formulations including dusts, granular, wettable powders, flowable concentrates and ready-to-use products. Maneb is typically applied in foliar applications to vegetable crops and apples by aerial equipment or ground equipment (829 in DHC99).

Vulnerable use and vulnerable groups
Because maneb is used as a herbicide on food crops this could mean a certain risk. However maneb is metabolised quickly in the environment. The EPA (see above) concludes that actual levels of maneb are too low to affect human health. Maneb could also present a risk to agricultural workers applying the herbicide. Assumed is that these workers take the neccessary precautions using the substance. There is no indication that maneb presents a specific risk to vulnerable groups or creates high risk situations. However the metabolite ETU could present a risk but this substance is not evaluated as an endocrine disruptor. This substance should be researched, to find out if it could have endocrine effects.

Environmental concentrations
Dithiocarbamates have not been measured in the different application areas in the Netherlands. From the available measurements follows that the different metabolites of dithiocarbamates have been found in surface water. ETU is found in Flevoland in the Netherlands in 46% of the measurements up till 0.9 ug/l. ETU is descended from maneb in this area. MITC is only found incidentally up to 0.13 ug/l. Dithiocarbamates (as CS2) have incidentally been found up to 7.5 ug/l. Dithiocarbamates are not measured in ground water in the Netherlands. Only ETU and MITC have been found. Especially in areas with bulb cultivation high levels of ETU occur in ground water of up to 42 ug/l. MTIC has been

found up to 2.5. In Flevoland CS2 (226 ug/l max) and ETU (max 75 ug/l) have been found in rain water. CS2 is not found in marine water in the Netherlands. In Zeeland in the Netherlands in 55% of the measurements low concentrations CS2 have been found in sediment. Dithiocarbamates have not been measured in organisms (Ordelman, et al 1993a).
No measurements of maneb in freshwater, marine water, rain water and groundwater have been done (Ordelman, 1996).

Legal status

Maneb is listed on the Priority pesticides list under Directive 91/414/EEC (and specified under Council Regulation 3600/92).

Conclusion
Maneb is used as a herbicide on food crops this could mean a certain concern for exposure. As maneb is metabolised quickly in the environment, actual levels of maneb are too low to affect human health. However the metabolite ETU could present a risk but this substance is not evaluated as an endocrine disruptor. This substance should be researched, to find out if it could have endocrine effects.

On the basis of its metabolite ETU, maneb is considered as a substance of high concern.

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Metam natrium

The substance was selected for evaluation in the expert meeting because it is a HPV chemical, which is produced in more than 1000 tonnes/year.

Expert evaluation
The Wildlife relevant endocrine disruption data are evaluated as category 3 and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1 Physico chemical properties of metam natrium

Water solubility	722 g/l at 20 C (Ordelman et al, 1993a)
Vapour pressure	3.4 Pa at 20 C (Ordelman et al, 1993a) < 1.7 E-4 Pa at 20 C (IUCLID96 (v10))
Log Koc	0.4 (estimated based on water solubility; Kenaga 1980 in Ordelman, et al, 1993a) 2.6 (Ordelman, et al, 1993a)
Log Kow	<1.89 (OECD 107) (IUCLID96 (k9))

Metam natrium is very good soluble in water.
Metam natrium is hydrolysed with a half life of about 8 days and fotolysed with a half life of <1 day (Ordelman, et al, 1993a).
Dithiocarbamates are generally instabile compounds. Important metabolites formed at all dithiocarbamates are carbondisulfide (CS2) and sulfurhydrogen (H2S) (Van Leeuwen, 1986 in Ordelman et al, 1993a). Furthermore a distinction can be made between the metabolites of the ethylenebisthiocarbamates (maneb and zineb) and the mono- and dialkyldithiocarbamates (metam natrium, thiram). From the ethylenebisthiocarbamates a joint 1,2,4-dithiazole (DIDT) is formed. DIDT is presumed to be the active compound. Other metabolites of the ethylenebisthiocarbamates are ethylenediisothiocyanate (EDI) and ethylenethiourea (ETU). ETU can be metabolised further. DIDT and ETU cannot be formed in the absence of oxygen (Vonk, 1975 in Ordelman, et al, 1993a). The metabolisation of DIDT to EDI does not occur in the presence of zinc- or ironsulfate (Vonk, 1975 in Ordelman, et al, 1993a) in water but does occur in the presence of copperoxide (hunter and evans 1991 Ordelman, et al, 1993a). Metam natrium is metabolised to methylisothiocyanate (MITC) which is sometimes regarded as the active compound (Panman and Linders, 1990 a and a in Ordelman, et al, 1993a). MITC hydrolysed to dimethylthioureum (DMTU, DT50 = 10-19 days, pH=7). From metam natrium a 1,2,4-dithiazole and a 1,2,4-thiadizole are formed by (air) oxidation (Thorn, 1960 in Ordelman, et al, 1993a).
Expected is that the most dithiocarbamates will be metabolised quickly in the environment and in aquatic organisms and therefore will not spread to greater water systems and will not bioaccumulate or biomagnify (Ordelman et al 1993a).

Under aerobic conditions, the half life of metam natrium was found to be 23 minutes. At pH 7 the hydrolysis DT50 is 180 hours and the photolysis DT50 1.6 hours. Therefore metam natrium is not considered persistent (CEFIC, 1999).

Use, Exposure and emissions
In 1985 5534 tonnes metam natrium was used in the Netherlands, 5372 tonnes in 1988, 5178 tonnes in 1991 and 1480 tonnes in 1994. The turnover has been fairly constant over the years 1986-1991. Since 1993 there is a regulation in the Netherlands to reduce the use of soil desinfectants (Ordelman et al , 1993a).
Metham natrium is a soil fungicide, nematicide and herbicide with a fumigant action. Its activity is due to decomposition to methyl isothiocyanate. It is phytotoxic and planting in treated soil must be delayed until decomposition and aeration are complete as shown by the normal germination of cress seed sown on a sample of treated soil. Under moist conditions this occurs within 14 days (Worthing 1987).

Metam-natrium is used as a fungicide and nematocide. It also has herbicide characteristics. Metam-natrium is used to disinfect soil in several cultures (potatoes, strawberries, orchards) and in limited way as herbicide in vegetable and ornamental cultures (Ordelman et al, 1993a). Metam natrium is a soil desinfectant that does not leave any residues in the soil and thus no residues in the crops grown on desinfected soil. Therefore no dietary risk exists for metam natrium (CEFIC, 1999).

Vulnerable use and vulnerable groups
Because metam natrium is used as a herbicide on food crops this could mean a certain risk. However metam natrium is expected to degrade quickly in the environment. Metam natrium could also present a risk to agricultural workers applying the herbicide. Assumed is that these workers take the neccessary precautions using the substance. The metabolite MITC also is metabolized fairly quickly.

Environmental concentrations
Dithiocarbamates have not been measured in the different application areas in the Netherlands. From the available measurements follows that the different metabolites of dithiocarbamates have been found in surface water. ETU is found in Flevoland in the Netherlands in 46% of the measurements up till 0.9 µg/l. ETU is descended from metam natrium in this area. MITC is only found incidentally up to 0.13 µg/l. Dithiocarbamates (as CS2) have incidentally been found up to 7.5 µg/l. Dithiocarbamates are not measured in ground water in the Netherlands. Only ETU and MITC have been found. Especially in areas with bulb cultivation high levels of ETU occur in ground water of up to 42 µg/l. In the Netherlands MITC has been found up to 2.5 µg/l In Flevoland. CS2 (226 µg/l max) and ETU (max 75 µg/l) have been found in rain water. CS2 is not found in marine water in the Netherlands. In Zeeland in the Netherlands in 55% of the measurements low concentrations CS2 have been found in sediment. Dithiocarbamates have not been measured in organisms (Ordelman, et al 1993a).
No measements of metam natrium in marine water, rain water and groundwater have been done. In freshwater metam natrium has been researched in 1992 but has not been found (Ordelman, 1996).

Legal status
Metam natrium is listed DBP is referred to in council regulation EEC/793/93 on the evaluation and control of risks of existing substances and Council Directive 67/548/EEC relating to the classification and labelling of dangerous substances.

Conclusion
Metam natrium is used as a herbicide on food crops this could mean a certain concern for exposure. As Metam natrium is metabolised quickly in the environment, actual levels of Metam natrium are too low to affect human health. However the metabolite MITC substance should be researched, to find out if it could have endocrine effects.

On the basis of its metabolite MITC, metam natrium is considered as a substance of high concern.

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Mirex

The substance was selected to be evaluated in the expert meeting because it is a very persistent chemical.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 2 and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1 Physico chemical properties of mirex

Water solubility	0.2 (verschueren 96 in fra97) 0.2 (riwa,1998)
Vapour pressure	3*10E-7 mm Hg (IARC, 1979 in WHO, 1984))
Henry coefficient	779 Pa.m3/mole (ARS95 in fra97)
Log Koc	6 (ARS95 in fra97)
Log Kow	6.9/7.5 (verschueren 96 in fra97) 6.9 (28 riwa, 1998)

Mirex is very poorly soluble in water. Because it is practically insoluble in water, sediments act as a sink for mirex that enters waterways.

The log Kow values are between 6 and 7. The poor water solubility of mirex in combination with the high log Kow points towards accumulation in organisms. From the BCF value of mirex it follows that the substance is highly accumulated in organisms (fra97).
The BCF in algae is 12200, in molluscs 4900, in fish 2580 and in daphnids 14650. In vegetation the log BCF is –1.14 (ARS95 in fra97).
BCF for algae 3200-7300 after 7 days (Hollister 1975 in WHO, 1984), blue crab 1100-3000 after 3 weeks (Bookhout & Costlow, 1975), pink shrimp 2600-24000 after 3 weeks (Lowe 1974 in WHO, 1984), fathead minnow 51400 after 56 days (Huckins 1982 in WHO 1984) and chickens 69-138 after 20-26 weeks (Medley 1974 in WHO, 1984).
Mirex bioaccumulates at all trophic levels and is biomagnified through food chains (WHO, 1984).
In organism higher in the food chain, concentrations increased significantly. Water contained < 10µg/l and sediment 0-0.07 mg/kg. Birds contained 0-0.17 mg/kg and mammals 0-4.4 mg/kg, 1 year after treatment of coastal areas with mirex (Borthwick 1973 in WHO 1984).

Mirex is a stable chemical (persistent). Biodegradation by microorganisms does not take place except occasionally, under anaerobic conditions, and, even then, at a slow rate (WHO, 1984).

Photodegradation under the influence of UV radiation is slow, photomirex being the major degradation product. The environmental half life of mirex is of the order of many years and its breakdown products are equally stable (WHO, 1984).
The half life of mirex dispersed in water under intense UV radiation at 90-95 C was 48.4 h (similar to DDT: 42.1 h).
The half-lives in field dissipation is 3000 days (ARS95 in fra97).

Carlson (1976 in WHO, 1984) showed that 16-19.5% of the total mirex-related residues from soil samples, recovered 12 years after treatment at 1.12 kg/ha was photomirex and lesser amounts of chlordecone (3.1-6.3%), 10-monohydro-mirex and 1 isomers of dihydromirex (WHO, 1984). Levels in 12 year old experimental plots in Mississippe suggested that mirex had an environmental half life of many years (Carlson 1976 in WHO, 1984). An environmental half life of 5-10 years has been cited in

other studies (WHO, 1984). Slow photodegradation is likely to be the ultimate fate of mirex in the environment (WHO, 1984).
After oral ingestion, mirex is only partly absorbed into the body and the remainder (depending on the dose administered) is excreted unchanged in the faeces. Mirex can also be absorbed following inhalation and through the skin. It is a lipophilic compound and, as such, is stored in adipose tissue to a greater extent than in any other tissue. Mirex is transferred across the placenta to the fetus and is excreted with the milk (WHO, 1984). Mirex does not appear to be metabolized to any extent in any animal species investigated. Its elimination from the body is slow. Depending on the species tested, its half life in the body is several months (WHO, 1984).
Mirex is quite resistant to pyrolysis. Hexachlorobenzene is a major pyrolysis product with lesser amounts of carbon monoxide, carbon dioxide, hydrogen chloride, chlorine, carbon tetrachloride and phosgene (vapour) (WHO, 1984).
Mirex does not appear to be metabolized to any significant extent in any animal species (mice, rats, rabbits, monkeyes) (Waters, 1976 in WHO, 1984). Data from studies on rat, monkey, quail and goat, exposed to mirex, showed fast tissue uptake and slow elimination (WHO, 1984). The half life of mirex following oral administration was estimated to be more than 100 days (WHO, 1984). In a feeding study on rats, quail and mosquito fish a 40% decline in mirex levels in adipose tissue was found over a ten month period, while the half life of mirex was 20-30 days in the adipose tissue of quails and 4 months in fish. In rats 12-25% of the dose was eliminated in the faeces after 1 week (Ivie 1974 b in WHO, 1984).
The removal from a sewage treatment plant is expected to be >90% (riwa, 1998).

Use, Exposure and emissions
Technical preparations of mirex contain 95.19% mirex and 2.58% chlordecone (WHO, 1984). Insect bait formulations for aerial application contain 0.3-0.5% mirex and fire ant formulations 0.075-0.3% mirex, which have been used in the USA (WHO, 1984).
Mirex is used as an insecticide (gre96, RIWA, 1998). Mirex is also used as a polymer (RIWA, 1998). In Germany mirex has never been used in allowed insecticides. In the USA mirex containing products have been redrawn from the market since 1997 (UN 1994 in bruhn 1998).
The same chemical substance is also used, under the name dechlorane, as a flame retardant in plastics, rubbers, paints, paper and electrical goods and as a smoke-generating compound, when combined with zinc oxide and powdered aluminium (WHO, 1984).
Mirex is mainly used as as a flame-retardant and as a stomach insecticide for the control of ants, especially fire ants and harvester ants. The USA appears to be the main country in which mirex was used for pest control, but this use was discontinued in 1978. To combat the problem during 1962-75 250,000 kg mirex was applied to the fields (WHO, 1984). Mirex is not allowed in the Netherlands (fra97). The canadian guideline for consumption is 100 ng/g wet weight (41 riwa, 1998)
The application as a flame retardant is not restricted to the USA (WHO, 1984). Statistics show that between 1959 and 1975 400 tonnes of mirex and 1500 tonnes of dechlorane were sold of which 74% was used the USA for non-agricultural purposes (US NRC, 1978 in WHO, 1984). Recently non-agricultural mirex has been replaced by dechlorane plus, dechlorane 4070, 510, 602, 603 and 604, which have similar flame retardant properties (WHO, 1984).
Little information is available on the world-wide production and use, but patents for the use of mirex exist in several countries including Belgium, France, Germany, Japan, the Netherlands and the UK (Task Force on Mirex, 1977 WHO, 1984).
The use as insecticide leads to distribution to air and atmospheric transportation.
Very little information is available concerning the leaching of dechlorane from landfill sites or disposal of flame retardant material, but this may also present an important source of contamination (WHO, 1984).

Vulnerable use and vulnerable groups
A known source of exposure for the general population is food. However intake from this source is below the promulgated tolerance levels. No data are available on occupational exposure to mirex. Within the food groups the largest intake of mirex would results from fish consumption, followed by

wild game and then commercial meats. (WHO, 1984). Mirex may also be excreted in human milk and is actually measured in human milk. It is bioaccumulative and biomagnifies through food chains. There is therefore an indication that mirex present a specific risk to a breast feeding infants.

Environmental concentrations

Atmospheric exposure to mirex could result from the air-borne dust from production and processing of mirex, combustion of dechlorane plastics or dechlorane smoke compounds or volatilization of mirex from insecticide (WHO, 1984).

Mirex has been found in one sample of ground water in the USA and in waters (0.0001 µg/l) shortly after insecticide application. Pond water in drainage areas are also known to contain higher levels of mirex after treatment (0.2 and 0.53 µg/l) (Spence & Marklin in WHO, 1984). It has also been determined in rural drinking water at levels of 0-437 ng/l (Sandhu 1978 in WHO, 1984). However mirex has not been found in tap water with a detection limit of 5 ng/l (Smillie 1977 in WHO, 1984).

Mirex residues have been observed in beef fat in the southeastern USA (0.001 mg/kg-0.125 mg/kg) but it was not found in areas where no insecticide was used (Fird 1973 in WHO, 1984).

Mirex residues of 0.01-1.71 mg/kg were found in soya beans, garden beans, sorghum and wheat seedlings (grown on substrates with 0.3-3.5 mg/kg mirex) (WHO, 1984). Plant issues grown on contaminated soil could contain between 0,2 ng/kg and 2 µg/kg mirex (US EPA, 1978 inWHO, 1984).

Bird residues levels typically ranged from less than 1 mg/kg to 10 mg/kg. Residue concentrations of 210 mg/kg have been reported in lipids extracted from homing gulls from Lake Ontario (WHO, 1984). Vertebartes as frogs, lizards and shrews have been observed to contain 9 mg/kg, 5.46 mg/kg and 41.3 mg/kg mirex, resp. Residues levels for frog and lizard range from 1-10 mg/kg and for shrew from 20-40 mg/kg (WHO, 1984). These residue levels are maximal shortly after application. These residue levels decrease over time but small amounts have been observed 3 years after application in tissues (WHO, 1984). Fish in Lake Ontario and the St Lawrence river contained levels as high as 0.27 mg/kg (Suta, 1978 inWHO, 1984). Mirex has been reported in seals in the Netherlands (Ten Noever de Brauw 1973 in WHO, 1984).

Within the food groups the largest intake of mirex would results from fish consumption, followed by wild game and then commercial meats. The average consumption of mirex through finfish would be 0.39 µg/dag if the fish where from the St Lawrence river and 0.34 µg/l from the Lake Ontario and in the Southern states of the USA 0.13 µg/dag (WHO, 1984).

Mirex may be excreted in milk. In 1436 samples of human milk, collected in the USA, no mirex was detected (Suta, 1978 in WHO, 1984). However, in Canada in 3 out of 14 human milk samples 2-21.5 µg/kg fat were found (Mes 1978 in WHO, 1984).

In human adipose tissue levels of 0.16 to 5.94 mg/kg were found (Kutz 1974 in WHO, 1984). Mirex was also found in the blood of pregnant women in 106 of the 142 samples at a mean blood concentration of 0.5 µg/l (Lloyd 1974 in WHO, 1984).

Table 2 Occurrence in the environment of mirex

Compartment	Year	Location	Concentration	Unit	Reference (source)
Wildlife biota		Canada salamander	0-19.9 av. Wet weight	Ng/g	41 riwa, 1998
Wildlife biota		Canada salamander Sexual glands	0-26.5 wet weight	Ng/g	41 riwa, 1998
Wildlife biota		Canada salamander Liver	0.8-13.1 av. wet weight	Ng/g	41 riwa, 1998
Wildlife biota		Canada salamander	0-19.9 av. Wet weigth	Ng/g	41 riwa, 1998
Wildlife biota		Canada turtle eggs	1.4-223.4 av. wet weight	Ng/g	41 riwa, 1998
Food	<1978	Canada milk	0.06 av. 0.05 med.	Ng/g	42 riwa, 1998
Food	<1978	Canada milk fat	1.89 av. 1.55 med.	Ng/g	42 riwa, 1998

d.l= detection limit

Legal status

Mirex is listed on the OSPAR candidate list.

Conclusion

Exposure is expected to occur mainly through food. Although intake is low, mirex is very persistent, bioaccumulative and biomagnifying and may therefore present a risk. There is an indication that mirex presents a specific risk to breast feeding infants through the occurrence in human milk. It is also measured in the environment. The final indication is: high concern.

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Polybrominated biphenyls

The substances were selected for evaluation in the expert meeting because they are very persistent chemicals.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category ? and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1 Chemical and physical data of commercial PBB products (WHO 1994 a; Pijnenburg, 1995; WHO, 1995; Watanabe, 1990; Stenzel,1997 in BKH, 2000)

Compound	HexaBB	0ctaBB	NonaBB	DecaBB
CAS no	36355-01-8	2858-07-7	69278-62-2	13654-09-6
Commercial product	FM BP-6	XN-1902	Bromkal 80-9D	Adine 0102
Vapour Pressure (Pa)	25°C 0.000007	0.0000016[A]	0.000001[A]	< 0.0000006
Henry Coefficient (atm.m3/mole 10^{-7})	8.7	3.5	2.6	1.9
Solubility	10-50	30-40	32.5[A]	20-30
Solubility H20 (25°C ; µg /1)	11 – 610	30-40	Insoluble	<30
- distilled	0.32			
- deionized	0.06			
- as pure BB 153	30			
Log Koc	5.93	6.6	6.83	7.05
Log Kow	7.20	8.03[A]	8.31[A]	8.58

A interpolated value

The solubility of all PBBs in water is very low. The solubility of higher brominated PBBs is lower than for the lower brominated PBBs. Determination of the water solubility of these hydrophobic compounds is difficult to perform due to preferent adsorption on particles. Solubility in water can further vary due to co-solubilisation by other substances/pollutants (WHO, 1994a). For instance, PBBs were found to be 200 times more soluble in landfill leachate than in distilled water (Prins, 1996). Brominated compounds have a lower solubility in water than corresponding chlorinated compounds (Pijnenburg, 1995 in BKH, 2000).

PBBs are a lipophilic substances. The bioconcentration factors of OBBs in fish are around 10000 to 1000000. 3,4-PBB is an exception to this and does not accumulate in food and hardly in water. For all PBBs the bioconcentrationfactors are a factor 3-4 higher than the biomagnificationfactor (fra97). A broad range of values for octanol-water partition coefficient was measured for the various PBB congeners, with log Kow = 5.72 for dibromobiphenyl to log Kow = 7.20 for hexabromodiphenyl (gre96; Pijnenburg95 in fra97).
The log Kow for 4,4'-di-B is 5.72 and the log BCF (Poecilia reticulata) is 5.43, for 2,4,6-tri-BB the log Kow is 6.03 and the log BCF (Poecilia reticulata) is 5.06, for 2,2',5,5'-tetra-BB the log Kow is 6.5 and the log BCF (Poecilia reticulata) is 6.16 and for 2,2',4,4', 6,6'-hexa-BB the log Kow is 7.2 and the log BCF (poecilia reticulata) is 5.85 (Gobas, 1989 in BKH, 2000).
The log BCF is 5 to 6.1 on fat basis in salmon (tetraBB, triBB, DBB) and 5.1-6.2 in guppy (triBB, DBB, HBB and tetraBB (pijnenburg 1995 in fra97). Most PBBs have a log Kow values >7, and are therefore regarded as superlipophilic compounds (Prins, 1996). Specific partition coefficients for soil, sediment and suspended sediment indicate that the PBB congeners will be adsorbed moderately to strong to solid media (BKH, 2000).

Biomagnification factors were measured in an experiment where fish were fed with a PBB-spiked diet with the same PBB compounds. The bromine content of the food was 7.75 $\mu g/g^{-1}$.
The 96h bioconcentration and biomagnification factors on a wet weight basis in Salmon (Sakmo salar) are for di-BB (10 congeners) 1.2 x 10^6 and 0.179 resp., for di-BB (8 congeners) 1.3 x 10^6 and 0.318 resp., for di-BB (12 congeners) 63 x 10^3 and 0 resp., for tri-BB 425 x 10^3 and 0.449 resp., for tetra-BB (49 congeners) 314 x 10^3 and 0.589 resp. and for tetra-BB (70 congeners) 110 x 10^3 and 0.571 resp. (Zitko and Hutzinger, 1976; Zitko, 1977 in BKH, 2000).
For all PBBs, bioconcentration factors are several orders of magnitude higher than biomagnification factors. Di-BB (congener 12) was not accumulated via food and only moderately from water. Corresponding dichlorinated biphenyls showed the same effect (Zitko, 1977 in BKH, 2000). Compounds with a low bromine content bioconcentrated more strongly from water than compounds with a high bromine content. PBBs with more than six bromine atoms were hardly bioconcentrated at all (Zitko, 1977 in BKH, 2000). However, compounds with one to four bromine atoms were taken up more from food when the bromine content was higher. PBBs with six to eight bromine atoms were only accumulated to a slight degree from food. Zitko (1977 in BKH, 2000) found only hexa-BBs in fish tissue after exposure to a diet spiked with octa-BBs. This dehalogenation e.g. enrichment of lower halogenated congeners in tissue is not known for corresponding chlorinated biphenyls.

PBBs show further an unusual chemical stability and resistance to acids, bases, reduction, oxidation or heat. In that sense PBBs are chemically comparable to PCBs. However, chlorine atoms are stronger bound to polybiphenyl than bromine atoms (WHO, 1994a in BKH, 2000). As for PCBs, chemical stability of PBBs varies strongly with halogenation and substitution patterns (Safe, 1984 in BKH, 2000). Unlike PCBs, the reactivity of PBBs has not been well studied and documented in the literature (de Boer, 1999 in BKH, 2000).

All PBBs have a very low vapour pressure. Their volatility can vary over a wide range, but it is still significantly lower than that of corresponding PCBs.

Under laboratory conditions, PBBs are easily degraded by UV. The predominant photochemical reaction of PBBs in organic solvents is reductive debromination. Irradiation of 4-monobromodiphenyl at 33 nm in various polar and nonpolar solvents led to quantitative formation of biphenyl. Studies using lower brominated PBB congeners (i.e. tetra and lower) reported a preferential loss of ortho bromines. Irradiation of higher brominated congeners yielded a series of photoproducts, but a stepwise cleavage of orthobromines did not appear to be preferred above meta or para debromination (WHO, 1994a in BKH, 2000).
The photoreactivity of 2,2',4,4',5,5'-hexabromobiphenyl, the main component of commercial hexaBB, was found to be relatively high. Degradation occurred more rapid than with the hexachloro analogue. Consistent with the dehalogenation pathway, photodegradation of the commercial FireMaster mixture led to reduced concentrations of the more highly substituted PBB congeners. Technical octabromobiphenyl has been reported to photodegrade in xylene by reductive debromination with a half-life of 40 h (WHO, 1994a in BKH, 2000).

In laboratory investigations, mixtures of PBBs appear to be fairly resistant to microbial degradation. Soil incubation studies using FireMaster BP-6 and ^{14}C-PBB showed only little degradation of the major hexa- and heptabromobiphenyl congeners after 6-12 months. Only pentabromobiphenyl was shown to degrade slightly, based on recovery rates of PBBs from soil, $^{14}CO_2$ production, and the lack of ^{14}C-PBB intermediates (WHO, 1994a in BKH, 2000).
Soils incubated with photodecomposition products of ^{14}C-hexa and heptabromobiphenyl caused enhanced, but still minor, degradation (ca. 3%) as measured by $^{14}CO_2$ production. These findings are consistent with observations that degradation of PCBs by bacteria decreases with increasing chlorination (WHO, 1994a in BKH, 2000).
In other incubation experiments with FireMaster BP-6 with sterilized and nonsterilized soil, from the penta-, hexa-, and heptabromobiphenyl recovery was evaluated that all PBBs persisted for 6 months

with no significant microbial degradation. They observed the same kind of persistence over a period of 4 weeks in PBB incubations with mixed cultures of microorganisms (predominantly Alkaligenes odorans, A. denitrificans, and an unidentified bacterium). This culture was known to degrade water-soluble PCBs. However, no PBB metabolites were found in the PBB- saturated mineral solution after 4 weeks of incubation (WHO, 1994a in BKH, 2000).

The half-life for excretion from fish was determined for two PBB congeners, Firemaster and octa-BB-product. The excretion half-lives after uptake from fish are for 2,2', 4,5' tetra-BB 21 days and for 2,4', 5 tri-BB 13 days. The excretion half-lives after uptake from food are for 2,2', 4,5' tetra-BB 28 days, for 2,4', 5 tri-BB 26 days, for Firemaster 93 days and for OctaBB 93 days (Zitko, 1977 in BKH, 2000).

Table 2 Results of EPIWIN and EUSES estimation programmes for environmental distribution of selected brominated compounds in BKH, 2000.

Property	Decabromo Biphenyl
Log Kow (measured)	8.58
Log Koc (TGD)	7.05
H (measured) (atm m^3/mole)	$1.9*10^{-7}$
Volatilisation half-life from river	604 days
Volatilisation half-life from lake	4400 days
Half-life for reaction with hydroxyl radicals [c]	852 days
Removal in WWTP To effluent To sludge To air	 8.1 % 91.9 % < 0.01%

H: Henry's law constant. WWTP: Wastewater Treatment Plant TGD: Technical Guidance Document
[c] : Calculated from OH reaction rate constant estimated by the method of Atkinson and assuming a OH radical concentration of 1.5×10^6 molecules/cm^3 and 12 hours sunlight/day

Use, Exposure and emissions
In commercial production of decaBB (Adine 0102), biphenyl is brominated in a large excess of bromine. Due to incomplete bromination, also several lower brominated compounds can be formed (WHO, 1994a; Brinkman, 1980 in BKH, 2000). PBBs can have variety of possible congeners, depending on the number and position of bromine atoms on the phenyl rings. The systematic numbering system, developed by Ballschmiter et al. (1992 in BKH, 2000), for characterisation of polychlorinated biphenyl (PCB) congeners, has also been adopted for the corresponding PBBs congeners (Pijnenburg, 1995 in BKH, 2000). From the 209 possible PBB congeners, 101 compounds are listed in the Chemical Abstracts Service (CAS) register. PBBs are not known as natural product (WHO, 1994a in BKH, 2000).

The brominated flame-retardant industry currently consists of around 10 major companies that produce in total 50 chemicals, of which 30 compounds are used commercially. The major brominated flame-retardant of the PBBs is decabromodiphenyl (BKH, 2000).

The major producers of brominated flame-retardants are: Broomchemie in the Netherlands, Great Lakes Chemical Ltd and Warwick Int. Specialities Ltd in Great Britain, Albemarle and Great Lakes Chemical Corp. In the USA, Dead Sea Bromines in Israel and Tosoh/Matsunga/Nippo in Japan (EU, 1996). PBBs are exclusively produced by CECA Atochem (RIVM, 1993; Eurobrome, 1999 in BKH, 2000).

Most important industry sectors where brominated flame-retardants are used, are the electrical engineering and electronics sector, wires and cables, transportation and the building & construction

sector. Generally, applications can be divided into the use in plastics (polymers) and textiles (upholstery). Major application areas are:

- consumer electronics housings and backplates, office electronics housings and backplates, printed circuit boards and appliances in Electrical Engineering and Electronics
- wire & cable and upholstery in motor vehicles
- compartment linings and coverings, insulation and upholstery in rail vehicles
- panels, carpets and flooring in aircraft industry
- thermal insulation for roofs, facades, walls, sheeting for roofs, floor coverings, ducting & conduits, panels, linings and coverings in building and construction industry.

The electrical engineering and electronics sector is the largest application area for brominated flame-retardants. In 1998, approximately 60% of the global demand of 300,000 tonnes was used in this sector (CW, 1998a). When this use is related to the regional consumption of electrical engineering and electronics (E&E) plastics, it shows that specific use of brominated flame-retardants in European E&E plastics is considerably lower than in the USA and Japan (see table 3).

Table 3 Use of all brominated flame-retardants (BFR) in (E&E applications) (in BKH, 2000)

1998	W-Europe	USA	Japan	
Total plastics[1]	28000	27500	11000	Kton/y
E&E plastics[1]	2500	2500	1000	Kton/y
BFR's[2]	39	57	39	Kton/y
BFR / E&E	0,016	0,023	0,039	Ton/ton

1: APME 1999a and OECD 1994 2: computed from CW 1998a

The lower use of brominated flame-retardants in European E&E plastics is largely caused by less stringent fire safety requirements for consumer electronics in Europe. As a consequence of international discussions on fire safety standardisation, it is expected that use of (brominated) flame-retardants in E&E applications in Europe will increase in the future (APME, 1999b in BKH, 2000).

Table 4 Demand estimates for all brominated flame-retardants (1992-2004) (in BKH, 2000)

	Brominated Flame Retardants (BFR)					Total FR	
	W-Europe	USA	Japan	Other*	World	World	
1992	38,000	60,000	38,000	14,000	150,000	600,000	Tonnes/y
1998	60,000	95,000	60,000	85,000	300,000	850,000	Tonnes/y
2004 **	95,000	155,000	95,000	145,000	490,000	1,170,000	tonnes/y
Growth							
1992-1998	8.0	8.0	8.0	35	12.2	6.0	% / y
1998-2004**	8.0	8.5	8.0	9.3	8.5	5.5	% / y

*: non-OECD (China / other Asian countries) **: projected figures FR: flame-retardant

From table 3.6 can be concluded that between 1992 and 1998:

- BFR demands in Japan and western Europe increased to 60,000 tonnes;
- BFR demands in non-OECD countries increased from 14,000 to 85,000 tonnes.

Furthermore, it is demonstrated that at future growth rates:

- the global BFR market will be increased by 60% in 2004;.
- in 2004 the major BFR market will be in the USA and non-OECD countries;

The use of PBBs was 265 tonnes/year in 1988 (gre96; janus, 1994 in fra97). In the Netherlands it is recently forbidden. The estimated total worldwide production is 5-6000 tonnes/year (brinkman 1980 in

fra97). PBBs are on Annex 1 and 2 of the EU council regulation 2455/92. They are permitted in the EU but prohibited in Switzerland. PBBs are also on the PIC list and in EC directive 76/769/EC (ISPRA, 2000).
Production in the EU further includes decabromobiphenyl in France. 1000 Tonnes decabromobiphenyl are produced in the EU and are used in industrial processing. In the Netherlands 40 tonnes are used for industrial processing (EU 1999a,b,c; EU 1996 in BKH, 2000)

Flame-retarded wastes are either generated in the form of used consumer goods or as industrial waste materials. Flame-retarded waste materials consist mainly of plastic waste materials from cars, computers and consumer electronics such as TV sets, audio equipment, etc.
Being a constituent of regular industrial, company and municipal waste, these flame-retarded waste materials are currently disposed of with these waste streams. In 1998 however, in the Netherlands a waste disposal directive came into force with respect to separate collection and treatment of white and brown goods (VROM, 1998). According to this directive, flame-retarded plastics used in brown and white goods, must be separated and disposed of by controlled landfilling or incineration. It is estimated that, by diverting these brown and white goods from municipal waste incineration, the brominated pollutants content in bottom and fly ash can be lowered by approx. 90%. However, due to the short implementation period of the new directive, flame-retarded plastics are still largely disposed of through landfilling and incineration of (unseparated) municipal waste (VROM, 1998).
According to draft EU directives on banning integral disposal of hazardous compounds in solid wastes e.g. municipal solid waste (EU, 1998), industry initiatives are being developed to source separate brominated wastes and recycle these waste materials. Most initiatives are focused on separate collection of electrical/electronical goods and mechanical removal of flame-retarded plastics. Separated plastics are crushed to pellets (recyclate) for reuse or separate disposal BKH, 2000).

A significant disadvantage of brominated flame-retardants is that PBDDs and PBDFs may be formed during thermal treatment or waste incineration. These brominated compounds are estimated to be just as toxic as their chlorinated congeners PCDD and PCDFs, but little is still known about the actual toxicity of PBDDs and PBDFs (WHO, 1994a,b). Most research reports indicate that maximal PBDF or PBDD production is observed at temperatures between 400 and 600^{o}C, depending on the type of brominated flame-retardant. However, highly toxic 2,3,7,8-substituted compounds were measured only in low concentrations (WHO, 1994b in BKH, 2000).

Laboratory pyrolysis experiments with PBDEs and PBBs show that PBDFs and PBDDs are formed in various concentrations, depending on the nature of PBDEs and PBBs, polymer matrix, processing temperature, presence of oxygen and type of moulding equipment used (Lahaniatis, 1991). Antimony oxide, frequently used as a synergist in flame-retardant systems, can further play a catalytic role in formation of PBDFs and PBDDs (WHO, 1994b in BKH, 2000).
Quarts flask pyrolysis in N_2/H_2 atmosphere at 1100^{o}C demonstrated that flame-retardants in printed circuit boards and electronic components still produce small amounts of toxic 2,3,7,8-TeBDFs in incineration ashes (29 µg/kg; Dumler-Gradl, 1995). Therefore, as with PCBs, thermal destruction of PBB and PBDE containing wastes should be carefully controlled. For PCBs, a residence time of 2 seconds at temperatures >1000^{o}C is recommended. Since PBDDs and PBDFs are not detected at temperatures >800^{o}C, a similar approach might be effective for adequate destruction of PBDEs and PBBs (WHO, 1994a in BKH, 2000).

Brominated dioxines and furanes can also be formed in plastic processing, as shown in a study with 78 TV's and 34 personal computers. Composed samples of plastics, flame-retarded with PBDEs and PBBs, already contained traces of PBDFs and PBDDs. When only PBBs were used, these levels further increased during plastic recycling. In that sense, possible formation of PBDFs and PBDDs should also be included in the overall toxicity and risk assessment of PBBs and PBDEs (Riess, 1998 in BKH, 2000).

The emissions at the decabromobiphenyl production are estimated at ca. 120 kg to effluent and 1,400 kg through disposal of sludge (see table 5).

Table 5 Emissions at production site in the EU (year 2000; tonnes/year) (in BKH, 2000)

	Source emissions		Wastewater treatment			Direct to atmosphere		Solid waste
	Waste water	Process air	Effluent	Sludge	Vapour	Vapour	Dust	
Decabromobiphenyl			8.1%	91.9%	0.0%			
Reactor wash out	0.50	-	0.041	0.460	0.000	-	-	-
Product wash (centrifuge)	26	-	0.041	0.460	0.000	-	-	25
Product grinding / packaging	0.50	9.5	0.041	0.460	0.000	-	0.07	-
Total	27	10	0.122	1.379	0.000	-	0.07	25

Vulnerable use and vulnerable groups

PBBs give no indication for exposure of vulnerable groups or high risk situations.

Environmental concentrations
Surface water monitoring in the vicinity of PBB producing and processing facilities, revealed that the major PBB compounds detected in surface water were hexaBB and decaBB. Levels were generally in the range 1-20 µg/l, which is in good agreement with experimentally obtained values (WHO, 1994a in BKH, 2000).

Near the former production site of the Michigan Chemical Corporation, PBB levels in sediment were measured from 1974 to 1977. During production, levels in sediment near the production site were as high as 77 mg/kg dry weight (as hexaBB) and declined to 6.2 mg/kg dry weight at half a mile downstream respectively to 0.1 mg/kg dry weight at approx. 30 miles downstream. Sediment levels upstream of the plant were typically < 30 µg/kg dry weight. Measurements after plant shut-down in 1976 demonstrated that after termination of the production, PBB levels in sediment sharply decreased. In 1977 hexaBB levels were only 100-500 µg/kg dry weight (WHO, 1994 in BKH, 2000). Similar measurements at another PBB production location revealed that activated sludge treatment of PBB containing wastewater adequately prevented sediment pollution. Only 40 µg/kg dw hexaBB was found downstream the plant, whereas the sewage sludge contained several PBB congeners, varying from 0.5 mg/kg dw monobromobiphenyl to 390 mg/kg dw of decaBB (WHO, 1994 in BKH, 2000).

Measurement in river sediments around the North Sea demonstrated that decaBB is the major compound in sediment, ranging from 0.33 µg/kg dw in the Mersey river to 0.40-0.80 µg/kg dw for the Rhine and the Elbe river. Parallel to decaBB, traces of hexaBB were found in levels of < 0.01-0.024 µg/kg dw (de Boer, 1999 in BKH, 2000). Measurement data on the presence of PBB in marine sediments are scarce, although various investigations have been performed on the environmental occurrence of PBB. From measurement results from joint surveys on PBB and PBDE levels in Sweden and UK can be assessed that PBB are seldom found in levels above the detection limit (de Boer, 1999; Sellström, 1990 in BKH, 2000).

Measurements in various fresh water fish (carp, pike, bulhead) near the former PBB production location in Michigan revealed that hexaBB was the predominant congener in fish tissue. During PBB production, levels of hexaBB near the plant ranged from 540 µg/kg wet weight (pike) to 1,330 µg/kg wet weight (carp). After shut-down of the production (1976) levels in pike roughly halved (WHO, 1994a in BKH, 2000).

For seal and guillemot in remote areas (Northern Ice Sea and Spitsbergen) PBB levels have increased 3-10 times between 1987 and 1993, which might be an indication that PBB are spreading faster and

more widely in the environment than previously assumed on basis of their hydrofobicity and low aquatic mobility (see table 6).

Table 6. Total PBB concentrations calculated as technical mixture equivalents in herring, seals, and sea birds (µg/kg lipid) (Source: Jansson et al. 1987, 1993 in BKH, 2000)

Organism	Area	PBB total	
		1987	1993
Herring	Baltic Sea	-	0.16
	Bothnian Gulf	-	0.09
	Skagerrak	-	0.27
Seal	Baltic Sea	20/26	90
	Kattegat	3	10
	Spitsbergen	4	40
	Northern Ice Sea	-	0.42
Guillemot	Baltic Sea	16	370
	North Sea	-	80
	Northern Ice Sea	50	130
Sea eagle	Baltic Sea	280	

A recent study has identified brominated biphenyls in various whales, dolphin and seals from around the coast of the Netherlands around 1995 (de Boer et al, 1998). The authors concluded that the presence of the substances in sperm whales indicated that they had reached deep ocean waters, since sperm whales do not usually occur in shelf seas and usually feed in deep waters. Levels of penta- and hexaBB were found to be higher than tetraBB levels. DecaBB levels were not found above the detection limit (in BKH, 2000).

Table 7 Levels of brominated biphenyls in marine wildlife samples (de Boer, 1998 in BKH, 2000)

Species	Tissue	Fat (g/kg)	PBB concentration (µg/kg wet weight)				
			tetraBB	pentaBB	hexaBBD		decaBB
Sperm whale 1	Blubber	722	0.24-0.40	0.91	<0.1	1.9	<0.5
Sperm whale 2	Blubber	234	0.13-0.21	0.40	0.05	0.73	<0.3
Sperm whale 3	Blubber	317	020-0.36	0.70	<0.1	1.1	<0.4
Whiteb. dolphin	Blubber	990	4.1-7.5	8.3	<0.2	13	<0.9
Minke whale	Blubber	140	0.24-0.27	0.54	<0.02	0.82	<0.1
Harbour seal 1	Blubber	244	5.7-34	9.3	12	61	<1
Harbour seal 2	Blubber	963	2.3-3.1	1.4	<0.2	18	<1
Harbour seal 3	Blubber	722	0.52-3.0	1.1	<0.1	13	<1
Sperm whale 2	Liver	23	<0.01	0.63	<0.04	18	<0.3
Whiteb. dolphin	Liver	27	0.03-0.06	0.74	<0.02	19	<0.1
Harbour seal 2	Liver	35	0.05-0.10	0.62	<0.02	1.5	<0.1
Harbour seal 3	Liver	51	0.03-0.10	0.04	<0.01	0.82	<0.1
Harbour seal 4	Liver	30	0.14-0.90	0.44	<0.02	13	<0.1

Table 8. Occurrence in the environment of polybrominated biphenyls

Compartment	Year	Location	Concentration average	Unit	Reference (source)
Wildlife biota		North sea Zeekoet	80	µg/kg fat	Jansson, 1993, deboer 1989 inFra97
Wildlife biota	BB15, 52, 49, 101, 153, 169, 209 apart	Cetaceans	0.6-19.2	µg/kg ww fat	BEON in DHC99

Compartment	Year	Location	Concentration average	Unit	Reference (source)
Wildlife biota	BB15, 52, 49, 101, 153, 169, 209 apart	Mink whale	0.03-61.1	µg/kg ww fat	BEON in DHC99
Wildlife biota	BB15, 52, 49, 101, 153, 169, 209 apart	Fish muscles	0.2-0.04	µg/kg ww	BEON in DHC99
Wildlife biota	BB15, 52, 49, 101, 153, 169, 209 apart	Eidereend Liver	0.003-3.2	µg/kg wwt	BEON in DHC99

d.l= detection limit

Table 9 Overall emissions to the environment (year 2000; tonnes/year)

European Union	Industrial Amounts	Source emissions		Waste water treatment			Direct to atmosphere			Total emissions to the environment			
		Waste water	Process Air	Effluent	Sludge	Vapour	Vapour	Dust	Solid waste	Water	Air (v)	Air (d)	Solid waste
Production													
Decabromobiphenyl	1000	27	9.5	0.122	1.38	< 0.001	-	0.07	25	0.12	< 0.001	0.07	26.4
Compounding, processing and service life													
Decabromobiphenyl	1000	0.50	9.7	0.041	0.46	< 0.001	0.495	1.00	8.70	0.041	0.50	1.00	9.2

Air (v): as vapour Air (d): as dust particles

Legal status

Polybrominated flame-retardants are referred to in Directive 79/119/EEC including restricted substances (shortlist derived from DG ENV);

Conclusion

PBB are used as flame-retardants. Exposure is only expected through the production site, the waste stage and food (fish). PBBs are persistent and most are bioaccumulated and biomagnified. PBBs are found in biota (fish). The substances are classified as having high concern.

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Polychlorinated biphenyls (PCBs)

PCB47 (2,2',4,4'-Tetrachlorobiphenyl), PCB77 (3,3',4,4'-Tetrachlorobiphenyl), PCB153 (2,2',4,4',5,5'-Hexachlorobiphenyl), PCB169 (3,3',4,4',5,5'-Hexachlorobiphenyl), Aroclor 1242, Aroclor 1254, Aroclor1248 and Aroclor1260 were selected for evaluation in the expert meeting because they are very persistent chemicals.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category ? and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

PCBs are produced as mixtures. Their nomenclature is presented in two ways: IUPAC nomenclature which gives a number to each of the congeners (eg. PCB58). Whereas mixtures of specific congeners often are referred to as Aroclors (eg. Aroclor 1242).

Table 1 Physico chemical properties of PCBs

	PCB47 B	PCB77 B	PCB153 A	PCB169 B	Aroclor 1248	Aroclor 1260
Physical state					Mobile oil	sticky resin
Water solubility (mg/l 25°C)	0.058	0.030	0.00086	0.0025	0.054	0.0027
Vapour pressure (Pa 25°C)	1.1E-03	1.1E-03	1.1E-03	7.7E-05	6.5E-2	5.3E-3
Density (g/cm3 25°C)					1.41	1.58
Henry's coeff. (Pa-m3/mol 25°C)	12.5	12.5	13.4-87.3 6.85	6.85	280	460
Log Kow	6.29	6.63	7.75	7.41	6.34 B	8.7 B

A derived from Fra97

B from Syracuse calculations, 1996

PCBs are poorly to very poorly soluble in water (0.001-0.1 mg/l). The log Kow values of PCBs vary from 5.2 to 8.3 which indicates lipophilic compounds. PCBs accumulate strongly in organisms. Furthermore they are persistent. The high log Kow values indicate that PCBs have a medium to high sorption to sediment (fra97). The high Henry's law coefficients indicate a relative high volatility.

After evaporation into the air PCBs persist in vapour phase at first, and thereafter immediately adsorbed with particles. The tendency of adsorption is stronger depending on the degrees of chlorination. The distribution of PCBs all over the world is essentially considered to be due to the transportation through troposphere. At present it is demonstrated that source of exposure of PCBs is due to such re-distribution of previously released portions.

PCB in water is adsorbed with bottom soil and other organic compounds. The experimental data or monitoring data show that PCB levels in bottom soil or in floating materials are higher than that in water. The degree of adsorption is remarkably high with highly chlorinated analogs due to tight adsorption to bottom soil. From the solubility and octanol/water partition coefficient, PCB analogues that have lower chlorine contents are more weakly adsorbed.
As the adsorbed PCBs are slowly released to water, PCBs in bottom soil have a role as sink of PCBs. The loaded amounts of PCB to the environment are assumed to exist in the bottom of water ways.

PCBs have low water solubility and leach very limitedly in soil due to strong adsorption with soil particles.

Degradation of PCBs in the environment depends on degree of chlorination of biphenyl moiety. In general the more highly chlorinated PCB analogues undergo shower degradation. In the vapor phase the reactions with hydroxyl radical, which is photochemically produced by sunlight, are main conversion processes, with varying half - lives from 10 days (monochlorobiphenyl) to 1.5 years (heptachlorobiphenyl).

Neither hydrolysis nor oxidation reaction in the environmental water is important to degrade PCBs. Photolysis is presumed to be majorly responsible for degradation, but the actual significance in the environmental degradation is to be studied.

Microorganisms moderately degrade Mono-, Di- and Trichloro biphenyls. The rate of degradation of Tetrachlorobiphenyls is slow, and additionally more highly chlorinated biphenyls are not degraded by biodegradation.
The analogs, which have two chlorine atoms in *ortho* position of one or both rings, are remarkably slowly degraded, while the analogs in which chlorine atoms are localized concentrate to one ring are easily degraded.
PCB analogues, which have high chlorine contents, are metabolized into analogues which have lower chlorin contents by reductive dechlorination reactions under anaerobic conditions. Two types of dechlorination reactions are well known. One of the reactions is related to dechlorination degree occurring at *ortho*, *meta* and *para* positions and reductive potential, and another is the reaction that is related to deecholrination degree occurring at *meta* and *para* position as well as the molecular shape.

Since PCBs are highly lipid-soluble and the rate of metabolism and excretion is slow, they tend to bioaccumulate particularly in adipose tissues of most living animals and plants. The degrees of bioconcentration in the adipose tissues are decided by various factors such as period and levels of exposure, chemical structure of compound, including position and pattern of substitution. Such analogues that have higher chlorine contents, have the larger Kow and are more easily bioaccumulated (Japan, 1997).

Use, Exposure and emissions
PCBs are produced in 1-2 million tonnes/year (gre96). In the past PCBs have been used in electrical equipment, heath-transfer systems, hydraulic systems and as components in plastics, coats, paints, glues, drill- and cutting oil and carbon-free paper (Devoogt en Brinkman 1989 in fra97). PCBs may also be formed as an unwanted by-product of industrial production of other chemicals. Important sources of emission of PCBs are the waste incineration processes.

PCBs, which had been previously released to the environment, are widely distributed in global environment at present. PCB is volatilised into the air from soil and water, transferred into the air and re-distributed in both soil and water by rainfall again. A large quantity of PCBs persists in soil sediment of water, which is considered to have a role as sink. In Japan, its manufacture was prohibited in 1972. PCB, which had been manufactured for 17 years, was 50,000 t - 60,000 t and their cumulative amounts used were 44,800 t (Japan, 1997).

In EC council regulation 2455/92 Annex 1 chemicals are listed that are banned or severely restricted to certain uses. In this regulation is referred that PCBs except mono- and dichlorinated biphenyl's, or preparations, including waste oils, with a PCB content higher than 0.005% by weight may not be used. The production and use of PCB-containing products are forbidden in the Netherlands (RIVM, 1994 in fra97).

Vulnerable use and vulnerable groups
PCBs are released in the environment during production, use of PCB containing products, in case of fires/explosions and during incineration of PCB containing waste.

Humans become exposed to PCB's by 3 main routes:
- Uptake from the environment by fish, birds, livestock (via food chains) and crops.
- Migration from packaging materials into food (mainly below 1 mg/kg, but in some cases up to 10 mg/kg.
- Direct contamination of food or animal feed by an industrial accident.

Vulnerable groups like babies may become exposed to PCBs via breast milk.

Surface water may be contaminated by PCBs from atmospheric fallout, from direct emissions from point sources, or from waste disposal.

Vulnerable wildlife groups are predators of e.g. fish or mussels contaminated with PCBs.

Environmental concentrations

The concentrations of PCBs have been measured in several organisms, like flounder, dab and mussels. The PCB concentrations in the organisms are between 0.021 and 2.1 mg/kg. They have also been measured in sediment and suspended matter (fra97).
In the Fraunhofer report PCB 153 is measured in sediment with a median concentration of 14.90 µg/l (mean 23.58 µg/l) based on 202 data from 85 stations (191 data were above the determination limit).

Table 2 Occurrence in the environment of PCBs

Compound	Sample	Year	Location	Concentration	No. detected/ tested	Reference
PCB153	Water	1990	Hamburg (Ger)	1.8 ng/l (max)		UBA98
PCB153	Water	1992	Baden-Würtenberg (Ger)	<10 ng/l (max)		UBA98
PCB153	Water	1993	Germany	2-9 ng/l (max)		UBA98
PCB153	Water	1994	Bayern (Ger)	2-10 ng/l (max)		UBA98
PCB153	Water	1995	Germany	19-29.9 ng/l (max)		UBA98
PCB153	Sediment	1992	Germany	Max. 27-57 µg/kg dw		UBA98
PCB153	Sediment	1994	Elbe tributaries	Max. 12 µg/kg dw		UBA98
PCB153	Suspended matter	1990	Hamburg	Max. 25 µg/kg dw		UBA98
PCB153	Suspended matter	1993	Elbe + Rhine (Ger)	Max. 30-61 µg/kg dw		UBA98
PCB153	Suspended matter	1994	Germany	Max. 17-47 µg/kg dw		UBA98
PCB153	Suspended matter	1995	Germany	Max. 47-165 µg/kg dw		UBA98
PCB169	Suspended matter	1994	Hamburg + Elbe (Ger)	Max. 26-37 µg/kg dw		UBA98
PCB169	Sediment	1993	Hamburg (Ger)	Max. 119 µg/kg dw		UBA98
PCB77	Suspended matter	1994	Hamburg + Elbe (Ger)	Max. 1025-1343 µg/kg dw		UBA98
PCB77	Suspended matter	1995	Hesseb (Ger)	Max. 5000 µg/kg dw		UBA98
PCB77	Sediment	1993	Hamburg (Ger)	Max. 5015 µg/kg dw		UBA98
PCBs	Fish	1978	Netherlands	0.04-0.27 mg/kg fat	7 pos	EHC140
PCB153	Fish	1996	Z-Northsea ES-D	Avg: 45 µg/kg fw, 250 µg/kg fat	19	Fra97
PCB153	Fish	1996	Z-Northsea WNZ	Avg: 58 µg/kg fw, 500 µg/kg fat	25	Fra97
PCB153	Fish	1996	Z-Northsea WRS	Avg: 155 µg/kg fw, 1260 µg/kg fat	25	Fra97

Compound	Sample	Year	Location	Concentration	No. detected/ tested	Reference
PCB153	Mussels	1995	Z-Northsea ES-D	Avg: 7 µg/kg fw, 530 µg/kg fat	5	Fra97
PCB153	Mussels	1996	Z-Northsea ES-D	Avg: 5 µg/kg fw, 387 µg/kg fat	5	Fra97
PCB153	Mussels	1995	Z-Northsea WRS	Avg: 29 µg/kg fw, 1809 µg/kg fat	5	Fra97
PCB153	Mussels	1996	Z-Northsea WRS	Avg: 24 µg/kg fw, 1638 µg/kg fat	5	Fra97
PCBs	Food	1978	Netherlands	0.13-0.17 mg/kg fat	2 pos	EHC140
PCBs	Drinking water	1978	Netherlands	0.035	1 pos	EHC140
PCBs (analogs analyzed)	Fish Shellfish, Birds	1994	Japan	0.01 - 0.33 A 0.01 - 0.02 A -	39/70 16/30 0/5	Japan, 1997
3,4,3′,4′-tetra CB *	Sediment Fish	1994	Japan	0.0067 - 0.013 A 0.000015 - 0.0013 A	2/3 3/3	Japan, 1997
3,4,5,3′,4′-penta CB *	Sediment Fish	1994	Japan	0.000099 - 0.00017 A 0.000005 - 0.00018 A	2/3 3/3	Japan, 1997
3,4,5,3′,4′,5′-hexa CB *	Sediment Fish	1994	Japan	0.000010 - 0.000011 A 0.000008 - 0.000019 A	2/3 2/3	Japan, 1997
Total PCBs	Sediment Fish	1994	Japan	0.380 - 1.4 A 0.750 - 1.5 A	2/3 2/3	Japan, 1997

A : mg/kg
*: coplanar PCBs

PCBs are accumulated in both human adipose tissues and mother′s milk. PCB levels in various organs and tissues except in brain depend on the contents of fats. Mean levels of total PCBs in human milk fat vary from houses and lifestyle of donors who offer their samples, and the analytical methods used. Milk of women living in heavy industrialized area or city, or who eat fishes caught in heavily polluted area, has the possibility of containing high levels of PCBs.

Main foods that have problems are fishery products, the crustacea, meats, milk and other dairy products. If compared with the previous data, PCB levels in fishes are moderately decreasing (Japan, 1997).

Most of compositions of PCBs extracted in the samples analyzed in the environment including human adipose tissues and milk are not similar to those of PCB mixtures on the market.
The patters of gas chromatogram in human adipose tissues and mother′s milk show that highly chlorinated PCBs predominantly are detected in higher levels. For example the analogs shown below are typically found:
PCB105: 2,4,5,3′,4′-Pentachlorobiphenyl, PCB153 2,4,5,2′,4′,5′-Hexachlorobiphenyl,
PCB128 2,3,4,2′,4′,5′-Hexachlorobiphenyl, PCB180: 2,3,4,5,2′,4′,5′-Heptachlorobiphenyl,
PCB170: 2,3,4,5,2′,3′,4′-Heptachlorobiphenyl
Levels of other PCB homologs such as highly toxic coplanar PCB77: 3,4,3′,4′-Tetrachloro-biphenyl, PCB126: 3,4,5,3′,4′-Pentachlorobiphenyl and 3,4,5,3′,4′,5′-Hexachlorobiphenyl, are very low.

Legal status

PCBs are listed in List 2 of Council Directive 76/46/EEC, the OSPAR candidate list and HELCOM priority list.

Conclusion

PCBs are of high concern for human exposure. Vulnerable groups that are exposed are breast-feeding babies.

PCBs are measured at many locations in sediments, suspended solids and biota and are considered as persistent.

In the environment especially predators of fish and mussel are of high concern.

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Polychlorinated dibenzodioxins and dibenzofurans (PCDDs/Fs)

The substances 2,3,7,8- tetrachlorodibenzodioxin (2,3,7,8-TCDD), 1,2,3,7,8 Pentachlorodibenzodioxin (1,2,3,7,8-PCDD) and 2,3,4,7,8-Pentachlorodibenzofuran (2,3,4,7,8-PCDF) were selected to be evaluated in the expert meeting because they are very persistent chemicals.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category ? and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1 Physico chemical properties of Polychlorinated dibenzodioxins and dibenzofurans (PCDDs/Fs)

	2,3,7,8-TCDD	1,2,3,7,8-PCDD	2,3,4,7,8-PCDF B
Water solubility (ng/l 25°C)	7.9 – 317	67 fra97	236 fra97
Vapour pressure (Pa 25°C)	4.5E-6 – 9.8E-8	8.8E-8 A	2.17E-5 fra97 4.6E-5 B
Henry's coeff. (Pa-m3/mol 25°C)	1.63 – 10.34 0.0021-1.93 fra97	0.31 fra97	1.14 B
Log Kow	7.02	6.64 fra97	6.98 fra97

A Data of 1,2,3,4,7-PeCDD

B Syracuse calculations

PCDDs and PCDFs are very poorly soluble in water (1 to 500 ng/l) and have a high lipophilic character (log Kow 6.7- 8.0). PCDDs are strongly bioaccumulating in organisms. The BCF for several organisms are known to be higher than 1000. Because the degradation/metabolisation in organisms is low, the level of PCDD/Fs in organisms higher in the food chain, will be higher than of organisms lower in the food chain. PCDD/Fs primarily accumulate in the liver and the fat of the organisms. The Henry's law coefficients for the different dioxines vary a lot from very low to medium volatisation from water. Despite of the fact that dioxines are not volatile distribution primarily takes place through air (CCRX, 1991 in fra97).

PCDD/Fs are strongly adsorbed onto bottom sediment and benthos, etc. and present over 90% in water under adsorbed conditions. Soil and bottom soil sediment are assumed to be a large sink in the environment.

PCDD/Fs are generally very stable in the environment and persist for a long time. PCDD/Fs have a possibility of degradation in every environmental compartment, however, the rate of degradation is very slow and they are considered to be stable compounds.
TCCD is hardly degraded by hydrolysis and photolysis and its half-life time is 10 to 12 years.
2,3,7,8-TCDD is hardly degraded in living organisms. In an experiment using 2,3,7,8-[14C] TCDD its half-life time in lakes or ponds in the field was approximately one year (Japan, 1997). Metabolism of PCDDs and PCDFs occurs via the aren oxide formation and hydroxylated metabolites have been determined for at least tetrachlorinated congeners (Larsen, 1996 in sepa98) In addition cleavage of the ether bridges between the two phenyl rings were shown to occur resulting in catechols (sepa98).
DT50 of 2,3,7,8 TCDD in water and soil is estimated at >1 year (34 riwa, 1998). Expected removal from a sewage treatment plant is >40% by active coal (riwa, 1998).

Use, Exposure and emissions
PCDDs and PCDFs are unintentionally formed during combustion (e.g. municipal waste incineration, traffic, cable roating houses), metal production and reclamation, production of pulp and paper, chlorophenols, and chlorinated phenoxy herbicides, and at chlorine-alkali plants using graphites electrodes (rappe 1994 in sepa98). Incinerators of wastes are the greatest source of PCDD/PCDF (80% of the total emission). Another emission route is the volatisation from with pentachlorophenol preserved wood (RIKZ nota in fra97). In addition PCDD has been shown to be formed enzymatically in composts and sewage sludge (Oberg 1990, 1992 and 1993 in sepa98).

PCDD consists of 75 possible congeners and PCDF of 135 congeners. The relative amounts of PCDD and PCDF congeners vary with production and congener pattern can therefore be used to identify a source (rappe 1994 in sepa98). Both groups of compounds are highly hydrophobic and log Kow in the range of 6 to 9 for tetra- to octaCDD have been reported (gotz 1994 in sepa98). The most toxic PCDD congener is 2,3,7,8-TCDD and it is the standard of toxicity. The Toxic equivalence factor (TEQ) for other compounds is set as the potency of the compound/the potency of 2,3,7,8-TCDD. The concentrations of PCDD/Fs are often presented as the sum of the concentration multiplied with the TEF, yielding toxic equivalents (TEQs) in the sample (sepa98).

Vulnerable use and vulnerable groups
PCDDs and PCDFs are produced as unintential by products. Vulnerable groups are breast feeding babies and small children.

Vulnerable wildlife groups are predators of e.g. fish or mussels contaminated with PCDDs or PCDFs.

Environmental concentrations
The analysis of dioxines is complex and expensive. Therefore few measurements have been made. In the river Rhine concentrations have been measured of 0.01 ng TEQ/kg dry sediment upstream to 310 ng TEQ/kg dry sediment downstream. The highest measured concentration in the Rhine sediment is 219 ng TEQ/kg dw. The most important source of dioxines for water organisms will be through suspended matter and water bottom. Dioxin concentrations in eel, fish liver and molluscs have been measured. Through deposition from air, the overflowing of the river forelands, the use of contaminated fertilizer and dumping cq incineration of waste, dioxines may contaminate the soil. In total yearly > 2 kg TEQ falls onto the soil (fra97).
Geopraphically PCDD/Fs are of approx. the same level in herring at different locations along the Swedish east coast (150 ppt TEQ lw) but are lower in herring from the west coast (24 ppt TEQ lw in 1994). The levels of PCDDs/Fs are very low in terrestrial species (de wit in sepa98). Chemically levels of total PCDD/Fs in herring from the Baltic have been reported to be around 700 ppt lw. The levels in seals are considerably lower, 11 and 50 ppt in ringed seal and grey seal resp. (asplund 1990 in sepa98). The levels in fish-eating birds is much higher, 1,00 and 2,700 ppt in guillemot and sea eagle resp., but the levels have been decreased about five times from 1972 to 1992 (de wit 1994 in sepa98).

Dioxins levels in the air have been monitored as one of the measurement parameters of “The monitoring on non-regulated pollutants in the air” by the Environmental Agency, Japan, every other year from 1986. It is reported that Dioxins levels in the air is highest in the residential areas neighbouring industrial factories and in large areas, followed by medium sized cities and background area (mountain region). Namely, from the average levels from 1991 to 1994 the representative levels are assumed to be 0.6 pgTEQ/m3 in a large city area, 0.5 pgTEQ/m3 in a small-to-medium city area and 0.06 pgTEQ/m3 in a background area.

Table 2 Occurrence in the environment of Polychlorinated dibenzodioxins and dibenzofurans (PCDDs/Fs)

Compound	Compart-ment	Year	Area	Pg TEQ/m^3	Reference
PCDDs	Air	1987	Germany indoors (wood impregnated with PCP)	Max (min) 2.1 (0.004) pg TEQ/m^3 Median 0.1 pg TEQ/m^3	RIVM, 1993
PCDDs	Air	1987	Germany outdoor (not polluted)	0.013 pg TEQ/m^3	RIVM, 1993
PCDDs	Air	1990	Netherlands (rural areas)	Mean 0.01-0.04 pg TEQ/ m^3	RIVM, 1993
PCDDs	Air	1990	Netherlands (urban-industrial areas)	Mean 0.05-0.1 pg TEQ/ m^3	RIVM, 1993
PCDDs	Air	1990	Netherlands (industrial areas) point sources	Mean 1 pg TEQ/ m^3	RIVM, 1993
PCDDs	Air	1990	Residential area neighbouring industrial zone Japan	Mean (Median) 0.51(0.37) Pg TEQ/m^3 Max. (Min.) 0.90(0.10) Pg TEQ/m^3	Japan, 1997
PCDDs	Air	1992	Residential area neighbouring industrial zone Japan	Mean (Median) 0.59(0.54) Pg TEQ/m^3 Max. (Min.) 1.03(0.12) Pg TEQ/m^3	Japan, 1997
PCDDs	Air	1994	Residential area neighbouring industrial zone Japan	Mean (Median) 0.68(0.69) Pg TEQ/m^3 Max. (Min.) 1.30(0.69) Pg TEQ/m^3	Japan, 1997
PCDDs	Air	1990	Metropolis Japan	Mean (Median) 0.54(0.19) Pg TEQ/m^3 Max. (Min.) 1.76(0.02) Pg TEQ/m^3	Japan, 1997
PCDDs	Air	1992	Metropolis Japan	Mean (Median) 0.58(0.57) Pg TEQ/m^3 Max. (Min.) 1.15(0.04) Pg TEQ/m^3	Japan, 1997
PCDDs	Air	1994	Metropolis Japan	Mean (Median) 0.48(0.40) Pg TEQ/m^3 Max. (Min.) 1.10(0.02) Pg TEQ/m^3	Japan, 1997
PCDDs	Air	1990	Small and medium cities Japan	Mean (Median) 0.71(0.83) Pg TEQ/m^3 Max. (Min.) 1.16(0.01) Pg TEQ/m^3	Japan, 1997
PCDDs	Air	1992	Small and medium cities Japan	Mean (Median) 0.45(0.22) Pg TEQ/m^3 Max. (Min.) 1.36(0.01) Pg TEQ/m^3	Japan, 1997
PCDDs	Air	1994	Small and medium cities Japan	Mean (Median) 0.25(0.19) Pg TEQ/m^3 Max. (Min.) 0.59(0.01) Pg TEQ/m^3	Japan, 1997
PCDDs	Air	1990	Background area Japan	Mean (Median) 0.14(0.10) Pg TEQ/m^3 Max. (Min.) 0.32(0.01) Pg TEQ/m^3	Japan, 1997
PCDDs	Air	1992	Background area Japan	Mean (Median) 0.01(0.01) Pg TEQ/m^3 Max. (Min.) 0.02(0.00) Pg TEQ/m^3	Japan, 1997
PCDDs	Air	1994	Background area Japan	Mean (Median) 0.02(0.02) Pg TEQ/m^3 Max. (Min.) 0.04(0.00) Pg TEQ/m^3	Japan, 1997
PCDDs	Soil	<1990	Germany without sludge application	Max (min) 2.2 (0.1) ng TEQ/kg dwt (9)	RIVM, 1993
PCDDs	Soil	<1990	Germany with sludge applica.	Max (min) 261(4.4) ngTEQ/kg dwt (15)	RIVM, 1993
PCDDs	Soil	<1990	Germany wire reclamation incinerators	Max (min) 79 (254) ng TEQ/kg dwt (4)	RIVM, 1993
PCDDs	Soil	<1991	Italy pesticide plant	3400 ng TEQ/kg dwt	RIVM, 1993
2,3,7,8-TCDD	Soil	<1980	Italy Seveso	86000 ng TEQ/kg dwt	RIVM, 1993
PCDD	Eel		Netherlands	Max (min) 50 (10) ng TEQ/kg fat	CCRX/Fra97
PCDD	Fish liver		Netherlands	Max (min) 400 (10) ng TEQ/kg fat	CCRX/Fra97
PCDD	Shell fish		Netherlands	Max (min) 120 (50) ng TEQ/kg fat	CCRX/Fra97
PCDD	Fish	1993-95	inland sea Japan	Mean 9 ng TEQ/kg	Japan, 1997

Compound	Compart-ment	Year	Area	Pg TEQ/m^3	Reference
PCDD	Fish	1993-95	Open sea Japan	Mean 0.08 ng TEQ/kg	Japan, 1997
PCDD	Sediment	1990	Netherlands estuaries West-Scheldt	Max (Min) 16 (15) ng TEQ/kg dw (2)	RIVM, 1993
PCDD	Sediment	1987 &90	Netherlands sea Waddenzee	Max (Min) 21 (8) ng TEQ/kg dw (3)	RIVM, 1993
PCDD	Sediment	1990	Netherlands estuaries Eems Dollard	10 ng TEQ/kg dw (1)	RIVM, 1993
PCDD	Sediment		Rhine upstream	0.01 ng TEQ/kg dw	CCRX/Fra97
PCDD	Sediment		Rhine down stream	320 ng TEQ/kg dw	CCRX/Fra97
2,3,7,8-TCDD	Sediment Fish Shellfish	1994	Japan	0.001 - 0.002 µg/kg (3/36) 0.001 µg/kg (1/34) - (0/1)	Japan, 1997
1,2,3,7,8-penta-CDD	Sediment Fish Shellfish	1994	Japan	0.001 - 0.006 µg/kg (21/36) 0.002 µg/kg (2/34) - (0/1)	Japan, 1997
2,3,4,7,8-penta-CDF	Sediment Fish Shellfish	1994	Japan	0.001 - 0.024 µg/kg (25/36) 0.001 - 0.007 µg/kg (12/34) - (0/1)	Japan, 1997

*: TEQ (toxic equivalent)
Note: Figure in the parentheses is the number of chlorine atom.

Recently the US EPA has set a limit value for PCDDs/Fs at 0.3 µg TEQ/kg dry sludge, based a comprehensive human risk assessment for PCDDs/Fs in sludge assuming a standard consumption pattern (Hayward, 2000). In 1990 a tolerable daily intake of 10 pg TEQ/kg bw per day was derived for 2,3,7,8-TCDD. In Europe the estimated intake of PCDDs/Fs is estimated at 1 pg TEQ/kg bw per day (70 pg TEQ/man/day) and for breast-fed infants appr. 150 pg TEQ/kg bw per day (RIVM, 1993).

It is reported that total intake amounts of PCDDs from foods in Japan was 163 pg TEQ/man/day. Fish are the largest intake source and accounts for 64% of the whole intake. Meats and dairy products, which are the largest intake source in foreign countries, are presumed to be only 10% in Japan (Japan, 1997).

Legal status

Dioxin is referred to in Council directive 94/67/EC on the incineration of hazardous waste, Council directive 96/82/EC on the control of major-accident hazards involving dangerous subtances, Council decision 93/98/EEC on the control of transboundary movements of hazardous wastes and their disposal (Basel convention) and Council directive 76/768/EEC relating to cosmetic products.

Conclusion
PCDD/Fs are of high concern for human exposure. Vulnerable groups that are exposed are breast-feeding infants.

PCDD/Fs are measured in air, sediments, suspended solids and biota and are considered as persistent. Decreased breeding success, and developmental aberrations in terns and have been attributed to food contaminated with PCDD/Fs, DDE, PCB's, in the period 60s to 90s. In the last years effects have become more subtle (CSTEE, 1999).

In the environment especially predators of fish and mussel are of high concern.

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Resorcinol

The substance was selected for evaluation in the expert meeting because it is a HPV chemical, which is produced in more than 1000 tonnes/year.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 3 and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1 Physico chemical properties of resorcinol

Water solubility	840 g/l at 0 C (Verschueren, 1983) 2290 g/l at 30 C (Verschueren, 1983)
Vapour pressure	5 mm at 138 C (Verschueren, 1983) 1 mm at 42 C (JT Baker)
Log Kow	0.77/0.80 (Verschueren, 1983) 0.8-0.93 measured (iuclid96 in cefic686 (k1))

Resorcinol is completely soluble in water. When released into water, this material is expected to readily biodegrade. This material has an estimated bioconcentration factor (BCF) of less than 100. This material is not expected to significantly bioaccumulate. Biodegradation in an adapted culture gives 89% removal after 48 hr incubation (feed 446 mg/l) (292 Verschueren). The impact on biodegradation processes is at 200 mg/1, inhibition of degradation of glucose by *Pseudomonas fluorescens* and at >1,000 mg/l inhibition of degradation of glucose by *E. coli* (293 Verschueren). The major degradation processes for resorcinol in natural water may be biodegradation and photooxidation. A number of biological screening studies has found resorcinol to be readily biodegradable (Spectrum laboratories 2000).

By analogy to other phenol compounds, resorcinol may react relatively rapidly in sunlit natural water with photochemically produced oxidants such as hydroxyl and peroxy radicals; typical half-lives for hydroxyl radical and peroxy radical reaction with phenol are on the order of 100 and 19.2 hours of sunlight, respectively. Aquatic hydrolysis, volatilization, adsorption to sediments, and bioconcentration are not expected to be important.

The major degradation process for resorcinol in soil is expected to be biodegradation. A number of biological screening studies has found resorcinol to be readily biodegradable. In a laboratory soil degradation study using a chernozem soil, which had been exposed to the coking industry, 500 ppm resorcinol was degraded in 2 days. Resorcinol is expected to leach readily in soil; however, leaching may not be important if concurrent biodegradation occurs at a rapid rate (Spectrum laboratories 2000). In soil decomposition by a soil microflora occurs in 8 days (176 Verschueren).

When released into air, this material is expected to be readily degraded by reaction with photochemically produced hydroxyl radicals. When released into the air, this material is expected to have a half-life of less than 1 day (JT Baker, 2000). Based upon an extrapolated vapor pressure of 0.051 Pa at 25 deg C(1,SRC), resorcinol can be expected to exist almost entirely in the gas-phase in the ambient atmosphere(2,SRC). Gas-phase resorcinol is expected to degrade rapidly in air by reaction with photochemically produced hydroxyl radicals; the half-life for this reaction in an average atmosphere has been estimated to be 1.9 hr (3,SRC). Night-time reaction with nitrate radicals may also contribute to the atmospheric transformation of resorcinol (Spectrum laboratories 2000).

When absorbed through the skin or gastro-intestinal tract, Resorcinol is virtually totally excreted within 24 hours. Repeated administration does not lead to storage or accumulation (cefic686 (ch5)).

Use, Exposure and emissions
Resorcinol is used in the manufacture of adhesives and dyes and as ingredient in pharmaceutical preparation for topical treatment of skin condition (cefic 686 (u9)).
Resorcinol is also used in tanning, explosives, dyes, cosmetics, as a reagent for zinc dyeing and printing of textiles, as keratolytic, as topical antipruritic and antiseptic, as intestinal antiseptic cross-linking agent for neoprene; as rubber tackifier; as MFR styphnic acid in photography chemical and as intermediate in the synthesis of Resorcinol-formaldehyde resins, resin progenitors and as wood adhesive resins. As medical Resorcinol is employed in the treatment of acne, ringworm, psoriasis, eczema, seborrheic dermatitis and other cutaneous lesions. It is also used as a tire and rubber adhesive, as an ultraviolet absorber and as a starting material for the preparation of dyestuffs. Resorcinol is an indirect food additive polymer for use as a basic component of single and repeated use food contact surfaces. For use only as a reactive adjuvant substance employed in the production of gelatin-bonded cord composition for use in lining crown closures (Spectrum laboratories 2000).
In 1977, the United States use of Resorcinol was 65% in the manufacture of rubber products, 20% in wood adhesives and 15% for miscellaneous uses (Spectrum laboratories 2000).

There are 2 major producers of Resorcinol in the world: Sumitomo in japan and INDSPEC in the USA. In addition, there are three small plants in the People's Republic of China, two in India and an additional plant in Japan. There was a small plant located in Russia, but production is believed to have stopped in that country. Western Europe's only remaining Resorcinol plant was closed in late 1991 because of environmental concerns (CEH report on resorcinol, 2000).

Global production of Resorcinol exceeded 39000 metric tonnes in 1995. The USA is the largest producer and consumer. Western Europe's demand is satisfied by imports from Japan and the US. A new Western European plant due on stream in 1999 will be constructed at an unidentified European location using environmentally friendly hydroperoxidation technology. Production of Resorcinol in Japan is primarily for export (66% in 1995).

In the US and Western Europe, Resorcinol is used primarily in the production of specialty adhesives and/or adhesive improvers for tires and wood products. In Japan, the largest markets for Resorcinol are rubber products and meta-aminophenol production. In developed regions of the world, smaller amounts of Resorcinol are also used as a chemical intermediate in the production of UV stabilizers, functional and textile dyes, pharmaceuticals, explosives and herbicides. Outside the US, Western Europe and Japan, the principal use or Resorcinol is in the production of adhesive resins for tires (CEH report on resorcinol, 2000).
Resorcinol may be released to the environment in waste effluents associated with coal gasification and conversion, coal-tar production, and shale oil processing and from the combustion of wood and tobacco. If released to the atmosphere, resorcinol is expected to degrade rapidly (estimated half-life of 1.9 hr) by reaction with photochemically produced hydroxyl radicals. Night-time reaction with nitrate radicals may also contribute to its atmospheric transformation. If released to soil or water, biodegradation is expected to be an important fate process based on the results of a number of biological screening studies which have suggested that resorcinol is readily biodegradable. By analogy to other phenol compounds, resorcinol may react relatively rapidly in sunlit natural water with photochemically produced oxidants such as hydroxyl and peroxy radicals. Resorcinol is expected to leach readily in soil; however, leaching may not be important if concurrent biodegradation occurs at a rapid rate.
Occupational exposure may occur through dermal contact or inhalation at sites where the compound is produced or used as a chemical intermediate, or at sites involved with coal conversion. Exposure to the general population may occur through the inhalation of wood and tobacco smoke and through direct dermal contact with topical medicinal products containing resorcinol (Spectrum laboratories 2000).

Vulnerable use and vulnerable groups
Exposure to Resorcinol primarily occurs at the production sites and in the waste phase and through use in pharmaceutical preparations on skin and cigarette smoke.

Environmental concentrations
Resorcinol was found as a pollutant in filtered ground water and surface water at a waste treatment plant. It has also been found in effluent resulting from production of coal-tar chemicals: no concentration given. The concentration of resorcinol in the condensate water from a coal gasification facility in Grand Forks ranged from not detected (no analytical detection limit reported) to 60 ppm between Jun 1980 and Apr 1982. A resorcinol level of 1000 ppm was detected in wastewater from a synthetic coal conversion process. Resorcinol has also been found in wood smoke and cigarette smoke (Spectrum laboratories 2000).

Legal status

Resorcinol is listed in Council Directive 67/548/EEC relating to the classification, packaging and labelling of dangerous substances, Commission Directive 91/322/EEC on the protection of workers from risks related to chemicals at work, Council directive 76/768 concerning the approximation of laws relating to cosmetic products, Commission directive 90/128/EEC relating to plastic materials and articles intended to come into contact with foodstuffs and Council regulation EEC/793/93 on the evaluation and control of the risks of existing substances.

Conclusion

Resorcinol is used in the manufacture of dyes, pharmaceuticals, tanning and cosmetics. Exposure may occur through use as a pharmaceutical or cosmetic on skin and by (wood or cigarette) smoke inhalation. Resorcinol is readily biodegradable and not bioaccumulated. The substance is only found in effluents and cigarette smoke. The substance is prioritised as high concern.

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Styrene

The substance was selected to be evaluated in the expert meeting because it is a HPV chemical which is produced in more than 1000 tonnes/year.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 3 and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1 Physico chemical properties of styrene

Water solubility	160-300 mg/l (fra97) (mackay 92b in fra97) 280-400 mg/l (verschueren 96 in fra97) 280 mg/l (2 riwa, 1998)
Vapour pressure	6-10 hPa at 20 C (mer89 in iuclid96 (v3)) 0.8666 kPa (6.45 mmHg) at 25°C (EHC26)
Henry coefficient	2.33 Pa.m3/mole (fra97) (mackay 92b in fra97) calc.
Log Koc	2.74 (mackay 92b in fra97) estimated
Log Kow	2.9 (fra97) (mackay 92b in fra97) HPLC 2.78-3.18 (1 riwa, 1998) 2.96-3.16 (OECD 107) (bas and ban80 in iuclid96 (k3 and k4))

Styrene is a colourless, viscous liquid with a pungent odour (EHC26).
Styrene is soluble in water, has a medium volatisation from water and a low log Kow.

The log BCF in gold fish (calc. 0.83-1.13) shows low bioaccumulation, which was expected on the basis of the log Kow (Sabljic, 1987 in fra97). The BCF for fish (Carassius auratus) is 13.5 (oga84 in iuclid96 (b6)).

Styrene is persistent in water (2-30 weeks).
The degradation half-lives in soil are 2-4 weeks, in air are 51 min to 7.3 h, in surface water 2 to 4 weeks and in ground water 4 to 30 weeks (howard 91 in fra97).
If released to the atmosphere, styrene will react rapidly with both hydroxyl radicals and ozone with a combined , calculated half-life of about 2.5 hours. If released to environmental bodies of water, styrene will volatize relatively rapidly and may be subject to biodegradation, but is not expected to hydrolyze. If released to soil it will biodegrade and leach with a low-to-moderate soil mobility (828 in DHC99).
Several tests have shown styrene to be readily biodegradable and not to bioaccumulate in fish (CEFIC, 1999).

The results of controlled laboratory studies on animals and human beings have shown that uptake of styrene is rapid and that it is widely distributed throughout the body. Uptake is mainly via the pulmonary and, to a lesser extent, the dermal and oral routes. Styrene is distributed through the whole body and stored in lipid depots. Its subsequent slow elimination from the tissue indicates a potential for bioaccumulation following repeated daily exposure. Styrene is biotransformed largely via the 7,8-epoxide by the mixed function oxidase system. The first step in the metabolic transformation of styrene is its oxidation, catalysed by cytochrome P-450 dependent monooxygenases, to oxirane derivatives in the aliphatic chain or in the aromatic ring (EHC26). The principal urinary metabolites are alpha-hydroxybenzeneacetic (mandelic) and phenylglyoxylic acids. Recent evidence suggests that the pattern of urinary metabolite excretion varies with mammalian species. Other minor metabolic pathways may also be important in the toxicological assessment of this compound. The elimination of styrene and its metabolites appears to involve a two-compartment kinetic model that becomes

monophasic in experimental animals at high exposure levels. This suggests the existence of saturable metabolic pathways (EHC26).
Removal from sewage treatment plant by powder coal dosing is >90% at 1-8 mg/l with a contacting time of 300h. Removal by active coal filtration is >90% (estimated) (riwa 1998).

Transport of styrene in the environment is considered to be very limited due to its volatility from soil and surface water and the rapid destruction in air (DT50 2.6 hours) (CEFIC, 1999).

Use, Exposure and emissions
Styrene is present in the coal and brauncoaltar. Technical it is primarily extracted by direct dehydration from ethylbenzene (Rompp 1992 in Bruhn 1998).

The first step in styrene production is the catalytic alkylation of benzene with ethylene, both raw materials being supplied primarily from the petroleum industries. The following two processes are in use for the production of styrene (US EPA, 1980 in EHC26): dehydrogenation of ethylbenzene and co-product with propylene oxide. Ethylbenzene is oxidized to the hydroperoxide, which is then reacted with propylene to yield the propylene oxide and a co-product, methyl phenyl carbinol. The carbinol is then dehydrated to styrene (US EPA, 1980 in EHC26). Expected impurities may include propylbenzene, isopropylbenzene, and alpha-methylstyrene (EHC26).

The Federal Republic of Germany, Japan, and the USA are the major producers (Tossavainen, 1978 in EHC26). In Europe it is produced in the Netherlands (Sow benelux, Shell), Germany (BASF, Huls, Rheinische olefinwerke), Belgium (BASF), France, Spain and Italy (RIWA, 1998). It is estimated that the world styrene consumption will grow at an average of 5.1% per year in the period 1982-90 reaching 13.6 million tonnes by 1990 (Chemical Market Review, 1981 in EHC26). Styrene is produced in 3,700,000 tonnes in Western Europe (cefic126 (p11)).

Table 2. Production of styrene

Country	Year (tonnes)	Production
Canada	1974	146 000
France	1976	270 000
Germany	1976	60 000
Italy	1976	325 000
Japan	1976	1 090 000
Mexico	1974	30 000
Spain	1976	60 000
USA	1976	2 864 000
Others	1976	approx. 1 000 000
World	1977	7 000 000

a From: Tossavainen (1978) in EHC26.

Styrene (ethenylbenzene) is a commercially important chemical used in the production of polymers, copolymers, and reinforced plastics (EHC26). It is primarily used for the production of polystyrene and styrene-copolymers with acrylonitril, butadiene, maleineanhydride acide. Styrene is next to ethylene and vinylchloride the most important monomere for the production of thermoplastics (rompp, 1992 in bruhn 1998). Styrene is used in closed systems resulting in inclusion into or onto matrix. Used in basic chemicals, in synthesis in chemical industry, in paints, lacquers and varnishes industry, in paper, pulp and board industry and in polymer industry. It is used as an intermediate for polystyrene, styrene-butadiene, rubber (latex) and monomer (iuclid96 (u1)). Styrene is used as a raw material for latex production in one site in Finland (iuclid96 (u5)). Styrene is used in the manufacture of plastics, synthetic rubbers, resins, and insulators. It is also important in the manufacture of some paints and it is

used in the preparation of acrylonitril-butadiene-styrene and styrene-acrylonitrile polymer resins. It is an important ingredient in the manufacture of styrenated polyesters, rubber-modified polystyrene, and copolymer resin systems used as a diluent to reduce viscosity of uncured resin systems. It is used with glass fibers in the construction of boats and in the synthesis of styrene-divinylbenzene copolymers as a matrix for ion exchange resins. Styrene is used as a cross-linking agent in polyester resins. It is a FDA-approved flavoring agent for ice cream and candy (828 in DHC99). In the USA in 1980 styrene is primarily used for polystyrene (45%), for acrylonitrile-butadiene-styrene plus (10%), for styrene-acrylonitrile resin and styrene-butadiene rubber (8%), for styrene-butadiene latex (6%), for unsaturated polyester (5%) and 4% miscellaneous and 17% export (EHC26).

Styrene is released into the environment by emissions and effluents from its production and its use in polymer manufacture. It has been found in exhaust from spark-ignition engines, oxy-acetylene flames, cigarette smoke and gasses emitted by pyrolyis of brake linings. Styrene is also emitted in automobile exhaust (how90 in iuclid96 (u2)). Significant amounts of styrene may be released to the environment from emissions generated by its production and use and from automobile exhaust (828 in DHC99).
In 1994 the emission to air is 1560 tonnes/year and to water 16,477 tonnes/year (mtc in DHC99). The emission for styrene is 15.5 tonnes/year in 1988 in the Netherlands (gre96).

According to Pervier et al. (1974), the major sources of styrene contamination of the environment are the petrochemical industries. Emissions from styrene production may result from vents on distillation columns and other processing equipment, storage tank losses, miscellaneous leaks and spills, waste-waters, and solid process wastes. However, losses from production are low in comparison with other petrochemical losses. Some styrene can also be emitted from polymerization processes (EHC26).
Styrene can be released into the environment during various disposal procedures, e.g., during the incineration of many types of styrene polymers. Styrene has been detected in hydrocarbon exhausts from spark-ignition engines (Flemming, 1970 in EHC26), in oxyacetylene and oxyethylene flames (Crittenden & Long, 1976 in EHC26), and in cigarette smoke.

Exposure mainly occurs in industries and operations using styrene, and industrial sources are the most likely cause of general population exposure. Other potential sources of general population exposure include motor vehicle exhaust, tobacco smoke, and other combustion/pyrolysis processes. Low-level exposure of the general population can occur through the ingestion of food products packaged in polystyrene containers. General population exposure levels are usually orders of magnitude lower than occupational exposure levels - though the latter vary considerably depending on the operations concerned. While some exposure occurs in styrene/polystyrene manufacturing plants, the highest levels of exposure are found in the industries and operations concerned with the fabrication and application of plastics. Thus, industrial processes, such as those in the reinforced plastics industry, require the greatest attention. In addition, clean-up and maintenance procedures in many related industries may result in significant exposures (EHC26).
The domestic use of polyester resins has increased potential exposure patterns. Exposure may occur when styrene is used as a solvent during the preparation of resin flooring (Gadalina et al., 1969; Kaznina, 1969 in EHC26); or through the use of styrene in various hobbies, crafts, and toys (Smirnova & Yatakova, 1966 in EHC26). It is not know in howfar these uses are still actual.

Vulnerable use and vulnerable groups
For the moment, based on the fact that domestic use in e.g. toys is possible and also that styrene is used as a flavoring agent for ice-cream and candy and in food packaging material (from which it leaches), there is an indication that styrene may present a specific risk to a vulnerable group of high risk situations. If these uses are abandonned there is no specific risk to these groups.

Environmental concentrations
While styrene has been detected in various US drinking waters, it was not detected in a ground water supply survey of 945 US finished water supplies which use groundwater sources. Styrene has been detected in various US chemical, textile, latex, oil refinery and industrial wastewater effluents. Styrene has been frequently detected in ambient air of source dominated locations and urban areas, has been

detected in the air of a national forest in Alabama, and has been detected in the vicinity of oil fires. Food packaged in polystyrene containers has been found to contain small amounts of styrene (828 in DHC99).
Schofield (1974) analysed hydrocarbon emissions from vehicles and found that 0.76% of total hydrocarbons was in the form of styrene in the exhaust of conventional engines, and 2.67% in the exhaust of rotary engines. Styrene has also been identified in cigarette smoke, reported levels ranging from 18 to 48 µg per cigarette (Johnstone et al., 1962; Baggett et al., 1974; Jermini et al., 1976 in EHC26).

Polystyrene and its copolymers such as acrylonitrile-butadiene-styrene (ABS), have been widely used as food packaging materials. Currently available analytical surveys of food and food packaging have shown that the styrene monomer migrates into food from both rigid and expanded polystyrene foam containers. According to Withey & Collins (1978), the lowest concentration of monomer in rigid containers was 700 ppm and, the highest was 3300 ppm. Other studies give figures of a similar order of magnitude (Hamidullin et al., 1968). The lowest concentration of monomer in expanded polystyrene foam was 87 ppm (Withey, 1976). The highest migration figure (245.5 ppb) was found in samples of sour cream contained in rigid polystyrene containers (Withey & Collins, 1978). Styrene leached from containers at 0.2 - 0.5 ppm conveyed a disagreeable odour and taste to dairy products (Jensen, 1972). However, styrene was not detected in milk stored in polystyrene containers for up to 8 days (detection limit 50 ppb) (Finley & White, 1967). In another study, styrene rates of leaching ranged from 0.0077, 0.0078, and 0.0078 µg/cm2 respectively, for foam cups containing water, tea, and coffee, to 0.036, 0.064, and 0.210 µg/cm2, respectively for foam, impact, and crystal polystyrene cups containing 8% ethanol (Varner & Breder, 1981a,b in EHC26).
In general, styrene concentrations in food are between 3 and 4 orders of magnitude lower than that in the package. It appears that the contribution to the total body burden of styrene via food is minimal (EHC26).

Occupational exposure to styrene can be classified according to the types of operations in which it is present. In polystyrene manufacture, occupational chemical exposure is mainly to styrene. In reinforced plastics applications, where styrene is a solvent-reactant for copolymerization, styrene is also the major air contaminant. In many of the applications, the operations involve potential skin contact with liquid styrene (EHC26).
In summary, occupational exposure to styrene varies considerably depending on the operations concerned. While styrene is present in detectable amounts in styrene/polystyrene manufacturing plants, the greatest exposures occur in the operations and industries that use unsaturated polyester resins dissolved in styrene. However, in all industrial operations using styrene, high levels of exposures occur during the clean-up and maintenance procedures (EHC26).

Pulmonary uptake of styrene concentrations of 67-164 mg/m3 (16-39 ppm), after a single breath or during exposures of up to 8 h, has been studied in human volunteers. Depending on exposure conditions, uptake varied from 45 to 66% (Bardodej et al., 1961; Fiserova-Bergerova & Teisinger, 1965; Bardodej & Bardodejova, 1970 in EHC26). Styrene is absorbed through human skin when applied in the form of a liquid, an aqueous solution, or a vapour (EHC26). A recent review of studies of the uptake of styrene in several animal species indicated that styrene is distributed rapidly throughout the body, is stored in lipid-rich tissues, and is extensively metabolized (Santodonato et al., 1980 in EHC26).

In a study by Engström (1978 in EHC26), 7 male subjects were exposed to a styrene concentration of 210 mg/m3 (50 ppm) during 30 min at rest and three, 30-min periods on a bicycle ergometer set at a work intensity of 50, 100, or 150 W. The mean uptake of styrene was 490 mg. Specimens of subcutaneous adipose tissue were taken before and after exposure and at 0.5, 2, 4, and 21 h after exposure. Mean concentrations in the adipose tissue, up to 21 h after exposure, were of the same magnitude (3.6 mg/kg). From the concentration of styrene still detectable l3 days after exposure, it was possible to estimate an elimination half-time of between 2.2 and 4.0 days (EHC26).

Ramsey & Young (1978 in EHC26) exposed volunteers to a styrene concentration of 336 mg/m3 (80 ppm) for 6 h and observed that the decay of styrene concentration in the blood following the exposure fitted a 2-compartment model. They speculated that accumulation of styrene would not occur following repeated exposure to concentrations up to 840 mg/m3 (200 ppm) (EHC26).

Engström et al. (1978a,b in EHC26) measured air concentrations of styrene in a polymerization plant and obtained subcutaneous adipose samples from 3 workers. The TWA air concentrations ranged from 32 to 84 mg/m3 (7.6 to 20.2 ppm) and the mean daily styrene uptake of the 3 volunteers was 193, 343, and 558 mg, respectively. Adipose tissue concentrations were 2.8, 4.0, and 8.1 mg/kg, respectively, at the beginning of the working week, and 4.7, 7.7, and 11.6 mg/kg, at the end (EHC26).

Table 3 Occurrence in the environment of styrene

Compartment	Year	Time	Location	Concentration	Unit	Reference (source)
Water			Rhine	<0.2	µg/l	22 Riwa, 1998
Water			Rhine	<0.1	µg/l	22 riwa, 1998
Water			Meuse	<0.2	µg/l	22 riwa, 1998
Water			Meuse	<0.1	µg/l	22 riwa, 1998
Water			Info-spec	0.06-0.8	µg/l	Riwa, 1998
Wellwater			Close to waste dump of styrene-butadiene plant	0.01-0.02	mg/l	Valenta, 1966 in EHC26
Wellwater			Close to site of styrene dump	0.1-0.2	mg/l	Grossman, 1970 in EHC26
Air			Urban atmosph. USA Southern California	2-15	ppb	Neligan , 1965 in EHC26
Air			Urban atmosph. Japan	0.2	ppb	Hoshika, 1977 in EHC26
Air			Air at butadiene-styrene rubber plant in Cszechoslovakia	17	ppb	Valenta, 1966 in EHC26
Air			Ambient air	1-5	ppb	Ece94a in iuclid96 (ca1)
Air			At contaminated site	15.5	ppb	Ece94b in iuclid96 (ca2)
Food			Pilsner beer	70	µg/kg	Ece94a in iuclid96 (ca1)
Food			Split peas and lentils	4-5	µg/kg	Ece94a in iuclid96 (ca1)
Food			White wine	1-3	µg/kg	Ece94a in iuclid96 (ca1)
Food			apples	<1	µg/kg	Ece94a in iuclid96 (ca1)

d.l= detection limit

Legal status
Styrene is listed on the Council Regulation 793/93/EEC 1.3. Priority list.

Conclusion

Styrene is used in closed systems in paints, paper, pulp, as polymer in polystyrene for hobbies, crafts and toys and as packaging material in food containers (from which it has been shown to leach). It is also used as a flavoring agent. Exposure may occur primarily through food (flavoring, packaging) and toys. Although styrene is readily biodegradable and does not bioaccumulate based on the possible exposure and because it is found in food and water, styrene is prioritised as high concern.

Because there no recent information it may be that the use of styrene may have changes so that the mentioned exposures may not occur any more. This has to be researched.

For the moment assumed is that styrene gives an indication for high risk for certain groups or situations and is also measured in the environment the final indication is: high concern. If certain uses are abandoned the indication may change to medium concern.

References

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Thiram

The substance was selected to be evaluated in the expert meeting because it is a HPV chemical which is produced in more than 1000 tonnes/year.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 3 and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1 Physico chemical properties of thiram

Water solubility	16.5 mg/l at 20 C (ucb85a in iuclid96 (s10)
Vapour pressure	2.3 mPa at 25 C (OECD 104)(ucb85a in iuclid96 (v11))
Log Koc	>0.8 (Ordelman, et al, 1993a) 2.8 (estimated based on water solubility; Kenaga 1980 in Ordelman, et al, 1993a)
Log Kow	1.82 (Ordelman, et al, 1993a) 1.73 (OECD107) (ucd83 in iuclid96(k10))

Thiram is not very soluble in water.
Thiram is hydrolysed with a half life of 4 days at pH 5.7, 47 days at pH 7 and 140 days at pH 8 and photolysed with a half life of <1 day (Ordelman, et al, 1993a).
Based on the log Kow bioaccumulation is not expected.

Dithiocarbamates are generally instabile compounds. Important metabolites formed at all dithiocarbamates are carbondisulfide (CS2) and sulfurhydrogen (H2S) (Van Leeuwen, 1986 in Ordelman et al, 1993a). Furthermore a distinction can be made between the metabolites of the ethylenebisthiocarbamates (maneb and zineb) and the mono- and dialkyldithiocarbamates (metam natrium, thiram). From the ethylenebisthiocarbamates a joint 1,2,4-dithiazole (DIDT) is formed. DIDT is presumed to be the active compound. Other metabolites of the ethylenebisthiocarbamates are ethylenediisothiocyanate (EDI) and ethylenethiourea (ETU). ETU can be metabolised further. DIDT and ETU cannot be formed in the absence of oxygen (Vonk, 1975 in Ordelman, et al, 1993a). The metabolisation of DIDT to EDI does not occur in the presence of zinc- or ironsulfate (Vonk, 1975 in Ordelman, et al, 1993a) in water but does occur in the presence of copperoxide (hunter and evans 1991 Ordelman, et al, 1993a). Thiram is metabolised to methylisothiocyanate (MITC) which is sometimes regarded as the active compound (Panman and Linders, 1990 a and a in Ordelman, et al, 1993a). MITC hydrolysed to dimethylthioureum (DMTU, DT50 = 10-19 days, pH=7).
Expected is that the most dithiocarbamates will be metabolised quickly in the environment and in aquatic organisms and therefore will not spread to greater water systems and will not biaccumulate or biomagnify. The BCF is calculated to be 91 (Ordelman et al 1993a).

Under aerobic conditions the half life in soil of thiram was found to be 2 days. At pH 7 the hydrolysis DT50 in water of thriam is 3.5 days: not persistent (CEFIC, 1999).

Use, Exposure and emissions
Thiram is produced in 10000-50000 tonnes in europe (iuclid96 (p3)).
In 1985 79 tonnes thiram was used in the Netherlands, 95 tonnes in 1988, 99 tonnes in 1991 and 94 tonnes in 1994 (Ordelman et al , 1993a). The turnover of thiram is increased with 25% from 1985 to 1991 (Ordelman, et al, 1993a). The use as a paint is limited and was estimated to be less than 5 tonnes in domestic use in 1985 (Ordelman, et al, 1993a). The most important production locations of dithiocarbamates are in Germany and in the Netherlands (Ordelman, et al, 1993a).

Thiram is used as a protection remedy leaf-fungicide with a repellent effect. It is used on fruit, strawberries, endive, latuce and used to treat corn against wild gluttony (perkow and ploss 1996 in bruhn 1998). Thiram is also used in the domestic and industrial area as a antifungicide and antibacterial paint. The most important use in the Netherlands is the protection of apples during storage.

Vulnerable use and vulnerable groups
Because thiram is used as a herbicide on food crops this could mean a certain risk. However thiram is expected to metabolize quickly in the environment. Thiram could also present a risk to agricultural workers applying the herbicide. Assumed is that these workers take the neccessary precautions using the substance. The metabolite MITC also is metabolized fairly quickly.

Environmental concentrations
Dithiocarbamates have not been measured in the different application areas in the Netherlands. From the available measurements follows that the different metabolites of dithiocarbamates have been found in surface water. ETU is found in Flevoland in the Netherlands in 46% of the measurements up till 0.9 µg/l. ETU is descended from maneb in this area. MITC is only found incidentally up to 0.13 µg/l. Dithiocarbamates (as CS2) have incidentally been found up to 7.5 µg/l. Dithiocarbamates are not measured in ground water in the Netherlands. Only ETU and MITC have been found. Especially in areas with bulb cultivation high levels of ETU occur in ground water of up to 42 µg/l. MITC has been found up to 2.5. In Flevoland CS2 (226 µg/l max) and ETU (max 75 µg/l) have been found in rain water. CS2 is not found in marine water in the Netherlands. In Zeeland in the Netherlands in 55% of the measurements low concentrations CS2 have been found in sediment. Dithiocarbamates have not been measured in organisms (Ordelman, et al 1993a).
No measurements of thiram in freshwater, marine water, rain water and groundwater have been done (Ordelman, 1996).

Legal status

Thiram is listed on the Priority pesticides list under Directive 91/414/EEC (and specified under Council Regulation 3600/92).

Conclusion
Thiram is used as a herbicide on food crops this could mean a certain concern for exposure. As thiram is metabolised quickly in the environment, actual levels of thiram are too low to affect human health. However the metabolite MITC substance should be researched, to find out if it could have endocrine effects.
On the basis of its metabolite MITC, thiram is considered as a substance of high concern.

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Toxaphene

The substance was selected to be evaluated in the expert meeting because it is a very persistent chemical.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 2 and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1 Physico chemical properties of toxaphene

Water solubility	0.4/3 (verschueren 96 in fra97) at 25 and 20 degree C 3 mg/l (26 riwa, 1998) 0.55 mg/l (gre in DHC99)
Vapour pressure	26.3-52.6 Pa at 25C (TNO, 1999)
Henry coefficient	0.073 Pa.m3/mole (ARS95 in fra97) 0.62 Pa.m3/mole (TNO, 1999)
Log Koc	5 (ARS95 in fra97) 3.18; 5.32 (Ritter, 1995 in TNO, 1999) 5.0 (De Geus, 1999 in TNO, 1999)
Log Kow	6.44/5.28 (verschueren 96 in fra97) 5.5 (1 riwa, 1998) 6.4 (26 riwa, 1998) 4.82 (gre in DHC99) 3.23-6.60 (Ritter, 1995 in TNO, 1999) 6.44 (De Geus, 1999 in TNO, 1999)

Toxaphene is a yellow (amber) waxy solid (TNO, 1999).
Toxaphene is poorly soluble in water and moderately volatile (TNO, 1999).

Based on the solubility and log Kow toxaphene is expected to medium to strong accumulation in organisms (fra97). The BCF for fish is 4000-60000 (rivm in DHC99). For toxaphene, bioconcentration factors of 400-1200 for shrimp, 6920 for algae, 8000 for oyster, and 9600 for aquatic snails have been reported (Richardson & Gangolli, 92-96 in TNO, 1999). In fathead minnows and channel catfish maximum bioconcentration factors were 69000 and 50000, respectively (Richardson & Gangolli, 92-96 in TNO, 1999). Toxaphene was bioaccumulated in fish 10000-69000 in freshwater fish species. Excretion was very slow: 56 days for 36% elimination. For the arctic cod a high BCF value of 2×10^6 has been reported (De Geus *et al*., 1999 in TNO, 1999). Swackhammer *et al*. (1998 in TNO, 1999) presented the most recent data for toxaphene in the Great lakes of North America for water, sediment and the foodweb. It was shown that toxaphene significantly biomagnifies in the foodweb. Mean lipid normalised log bioaccumulation factors (BAFs) for phytoplankton, zooplankton, Mysid shrimps, sculpin and lake trout amounted to 5.82, 6.53, 6.29, 6.58, and 6.96 (TNO, 1999)
The BCF of a toxaphene mixture is 3100-2,000,000 (experimental) (De Geus *et al*. 1999, PIC, 1999, Ritter *et al.,* 1995 in TNO, 1999). The BCF of octachlorobornane is 163000 for zooplankton and 133000-5660000 for fish (experimental) and 48000-65000 (calc. For fish) (Geyer *et al.* ,1999 in TNO, 1999). The BCF of nonachlorobornane is 290000 for zooplankton and 100000-680000 for fish and 85000-115000 (calc. For fish) (Geyer *et al.* ,1999 in TNO, 1999).
It can be concluded that toxaphene is highly accumulating. The bioconcentration potential can be expected to differ considerably among toxaphene mixtures of different composition. Experimental BCF values for toxaphene congeners are still scarce (TNO, 1999).

Semi-volatility confers a mobility through the atmosphere that allows relatively great amounts to enter the atmosphere and be transported over long distances. On the other hand this moderate volatility does not result in the substance remaining permanently in the atmosphere. Moreover, due to its persistence toxaphene is continually deposited and re-evaporated. It may volatilize from hot regions but will condense and tend to remain in colder regions. This has far-reaching implications for the global environment because volatilized residues disperse through the global atmosphere. This results in the compound being found almost everywhere, but also in the most remote areas far removed from any source of use (TNO, 1999). The evaporation of toxaphene out of the water is an important process also due to the slow degradation rate in water-sediment systems (TNO, 1999).

Furthermore these compounds are also very persistent and residues are found on a global scale. Atmospheric transport of toxaphene after volatilization into the atmosphere is probably the major environmental pathway to most of the oceans and surface waters of the world. Reported losses of toxaphene a few months after application range from 50-80% (Majewski *et al.,* 1999 in TNO, 1999). Current atmospheric concentrations are also partly caused by volatilization of persistent residues although this can not be quantified at present. Atmospheric deposition is not a once-only process and toxaphene can be re-introduced into the atmosphere for further dispersal again and again (Majewski *et al.,* 1999 in TNO, 1999). Pollutant transport time into the troposhere above the surface boundary layer is generally in the order of a few weeks to months. Once in the atmosphere the global wind circulation patterns control long-range transport. Although the mixing time is in the order of 1 or 2 years, limited transport between hemispheres does occur (Majewski 1999 in TNO, 1999).

The rate of hydrolysis of toxaphene is negligible (TNO, 1999).
Toxaphene formulations are relatively stable in water and soil but may be degraded by losing HCl or Cl_2 when prolonged exposed to sunlight, alkali or temperatures above 100 °C. According to Saleh (1991 in TNO, 1999), technical toxaphene does not undergo a serious change when exposed to sunlight. The stability with respect to UV light, and acid and alkaline treatment differs among toxaphene congeners (De Geus *et al*., 1999 in TNO, 1999). It is suggested that dechlorination of toxaphene occurs during photolysis and the dechlorination rate is nonachlorobornanes > octachlorobornanes > heptachlorobornanes (TNO, 1999).

The DT50 in sandy loam is 10 years (Menzie 72 in fra97) 10 days according to mtc in DHC99 . The half-lives in field dissipation is 9 to 500 days (ARS95 in fra97).The half-life of toxaphene in soil ranges from 100 days up to 12 years, depending on the soil type and climate (Ritter *et al.* 1995 in TNO, 1999). Microbial degradation in soil and sediment is enhanced by anaerobic conditions. Half-lives of 3 weeks (Richardson & Gangolli, 92-96 in TNO, 1999) and 6 weeks (EPA, 1999 in TNO, 1999) were reported for degradation of toxaphene in anaerobic soils. For (partly) aerobic soils half-lives between 0.8 and 14 yr were reported. Toxaphene will adsorb strongly to soil and sediment with a Koc of 2.1×10^5 (EPA, 1999 in TNO, 1999). Toxaphene will gradually evaporate from soil to the air despite its strong adsorption. Based on the (above-mentioned) physical-chemical properties it is not expected that toxaphene will leach to groundwater (EPA, 1999; PIC, 1999 in TNO, 1999). In studies groundwater toxaphene concentration exceeding 1 ng/L have not been observed (PIC, 1999 in TNO, 1999).

Fingerling *et al*. (1997 in TNO, 1999) studied the degradation of toxaphene components in UV irradiated air and flooded soil. They concluded that the contribution of abiotic processes is more important in atmosphere, water, and maybe part of the aquatic biota, whereas the contribution of microbial pathways is probably more important in soil. Angerhöfer et al. (1999 in TNO, 1999) found that residue patterns of UV irradiated toxaphene and toxaphene in fish samples are quite similar. Fish seem to be able to metabolise some toxaphene congeners like Parlar #44 and #62 and in contrast with the Parlar #26 and #50 congeners (TNO, 1999).
Boon et al. (1996 in TNO, 1999) studied the biotransformation of toxaphene with bioassays. They concluded that the *in-vitro* biotransformation capacity differed among taxonomic groups, with an increase in activity in the order sperm whale < seebirds & dolphins << seals. Residue patterns of toxaphene in these animals in the field situation confirmed these findings (TNO, 1999). Differences

among toxaphene congeners in biotic and abiotic degradation potentials may occur but is only scarcely investigated. This complicates the environmental risk assessment of toxaphene in a way analogous to PCBs.

Removal from sewage treatment plant by powder coal dosing is >90% at 2-33 mg/l with a contacting time 28 days. Active coal filtration contacting time 26 min with treatment of 950.000 m3 water (riwa, 1998). Removal from sewage treatment plant is >90% by active coal (riwa, 1998).

Use, Exposure and emissions

Toxaphene is a reproducible mixture of polychlorinated camphenes and comprises more than 180 components. The major part of technical toxaphene consists of chlorinated bornanes (ca. 75%) (TNO, 1999). Toxaphene is produced through the chlorination of camphene (gulden98).

In short, the process of producing toxaphene firstly involves the preparation of the raw camphene feed which is subsequently chlorinated with or without using UV light to produce toxaphene (De Geus *et al.*, 1999 in TNO, 1999). Chlorination of technical camphene purchased from commercial sources also takes place and other synthesis routes are used as well. Toxaphene has according to available information never been produced in the Netherlands and in the EU production is now discouraged by EU regulations. Information on emissions from production in the United States indicates heavily contaminated water systems which are adjacent to chemical plants that produced toxaphene for a sustained period. Chlorobornanes found in unmodified toxaphene were reported in fish, several miles from the contaminated site. However, it can be assumed that this type of contamination remains restricted to a local scale. Although no quantitative data such as emission factors are available, it is probably safe to say that the amounts released through application will by far outweigh emissions from production, moreover since production and formulation are not reported to take place within Western Europe (TNO, 1999).
At the level of the European Union use and marketing within the EU is forbidden as plant protection product (directive 83/181/EEC of 14/31983-OJ L91 p35). Furthermore toxaphene is now a PIC chemical (listed in the Rotterdam Convention) and is listed in ANNEX 1 of REG 2455/92 and is therefore subject to the Export Notification Procedure (TNO, 1999).
Also, toxaphene is addressed by the UN/ECE Protocol on Persistent Organic Pollutants. This Protocol focuses on a list of 16 substances that have been singled out according to risk criteria. The Protocol bans the production and use of toxaphene outright and has been signed by the majority of the countries in Europe. Full implementation of this Protocol will eliminate all remaining uses, trade, stockpiles and production of toxaphene in UN/ECE-Europe. At present (November 1999) The Russian Federation, Belarus, Turkey, Bosnia-Herzegovina, The former Yugoslav Republic of Macedonia and Yugoslavia have not yet signed this Protocol for various reasons (TNO, 1999). Toxaphene was and is still used as insecticide (TNO, 1999). In the Netherlands toxaphene has never been used at all and it was officially banned in 1968. Within the European Union, only Germany and to a smaller extent Italy and Spain have reported historic use. Germany totally banned the substance in 1981, while European Union legislation, which became effective in 1984, “prohibits to use or place on the market all plant protection products containing toxaphene with no remaining uses allowed”. Also in all EFTA Member States no toxaphene is used at present (TNO, 1999).
The situation in Central and Eastern Europe may be more complicated. At present in Estonia, Lithuania, Romania and the Slovak Republic national law bans toxaphene from (principal) use. Its use is according to national law restricted in the Czech Republic, The Former Yugoslav Republic Of Macedonia and the Russian Federation. The Ukrainian Ministry of Agriculture states that toxaphene is not in use. In Latvia import and usage is not permitted since 1994 but there are stockpiles of obsolete toxaphene. International reportings on emissions (e.g. EMEP) by Hungary suggest that there is no use of toxaphene. Almost no information is available for the present situation Poland, Belarus, Bulgaria, Slovenia, Moldova, Croatia, Bosnia Herzegovina and Yugoslavia. In 1991, Saleh (Saleh 1991) reported use for Romania, Hungary, Poland and the former Soviet Union. Use in these regions was also expected by other authors at the end of the 1980s (e.g. Voldner and Schroeder 1989 in TNO, 1999).

A global inventory of the registration status of toxaphene, under the United Nations Environmental Programme (UNEP) indicates that by June 1999 toxaphene was banned by 58 countries while in 12 countries use is (severly) restricted. 37 Countries stated that there were inconclusive data to determine the status and in 8 countries legislation was lacking. 33% of all countries failed to respond (TNO, 1999). However it should be kept in mind that the official registration status of a pesticide is never a 100% accurate indication for the usage since it might not be fully clear whether for instance 'restricted use' is significant or illegal or otherwise not administered amounts are used (TNO, 1999).
In the period 1991 to 1994 production of toxaphene was at least 1200 tonnes in Africa. Production was also reported during this period for Central America while in South America import of the substance took place. At least 131 tonnes was exported from Europe (including Central and Eastern Europe) in 1991 (UNEP 1996 in TNO, 1999).
In conclusion, it can be assumed that use of toxaphene does not take place in Western Europe but there might be minor use in Central and Eastern Europe and Russia for certain crops. Outside Europe use does not take place in the USA and Canada but does take place in Central and South America. Currently, the heaviest current use seems to be in certain African countries. In literature Ethiopia, Sudan, Tanzania and Uganda are mentioned more than once. No significant uses are reported for Asia though for many Asian countries recent data and legislation is lacking (TNO, 1999).
The total cumulative global usage accounted for amounted to 450.000 tonnes. The interpolated usage came to 1330.000 tonnes for 1950 to 1993 and 670.000 tonnes during 1970 to 1993. These are enormous quantities (compare 2.600.000 tonnes for DDT during 1950-1993). The regions for which the highest usage is recorded seem to be the (Southern) USA, the former Soviet Union and Central America. But also in Europe, at least 10.000 tonnes has been used in Germany (in the former GDR, during 1960-1980, heaviest use in the 70s) and there was also usage in Italy, Spain (both minor), Poland and other Central and Eastern European countries (Voldmet 1995 in TNO, 1999).
Chlorine bleaching of wood pulp in the paper and pulp industry produces chlorinated camphenes similar to toxaphene. However no evidence has been found of compounds identical to the main congeners in commercial toxaphene (De Geus *et al.* 1999 in TNO, 1999).

Toxaphene is a non-systemic contact and stomach insecticide with some acaricidal action. It has often been used in combination with other pesticides. Its primary application includes:
- the cotton growing industry
- the production of: cereal, grains, fruits, soybeans, nuts and vegetables
- the control of ticks and mites in livestock
- application as a piscicide

In general, emission of pesticides takes place during and after application. For a given pesticide emission to water systems can be calculated based on among others application rate, soil properties, tillage practise, the way in which the pesticide is applied, physical and chemical properties of the pesticide and meteorological conditions. The pathways by which pesticides enter the environment during and after application are discussed in more detail in (Teunissen-Ordelman *et al*. 1995 in TNO, 1999). However, since use does not take place in the Netherlands nor its direct surrounding, these emission mechanisms are not considered relevant for toxaphene in the Dutch water systems (TNO, 1999).
As will be further discussed in the following section, toxaphene is a typically semi-volatile compound, a characteristic that favours the long-range transport of this substance. It can move over great distances through the atmosphere. Volatilisation may occur from plant and soil surfaces following application as pesticide. Atmospheric long-range transboundary transport is probably the dominant emission pathway for toxaphene in the Netherlands. Other sources may include ocean currents and rivers (Voldner & Li 1995 in TNO, 1999)
Toxaphene was introduced in 1949 and has been widely used since than, especially on cotton. In the Netherlands, it has never been registered. However, toxaphene, being persistent and semi-volatile, can be regarded as a "global pollutant", and has been detected e.g. in European marine fish. It is subject to international initiatives regarding Persistent Organic Pollutants (POPs) (TNO, 1999).

Vulnerable use and vulnerable groups

Toxaphene is a insecticide used on food crops and is very persistent. It may therefore present a risk. It may also present a risk to agricultural workers applying the insecticide. However it is not used anymore in the EU countries. It is found in the environment in wildlife biota and in mothermilk in Canada. Although it is not used anymore in the EU through long-boundary transport and through fish (wild life biota) it may present a risk to vulnerable groups.

Environmental concentrations

Data for European freshwater systems (surface water, ground water, rain water) are not available to our knowledge. Information is available for the North American continent because of the regional heavy use of toxaphene in earlier decades. Swackhammer *et al*. (1998 in TNO, 1999) reported recent toxaphene concentrations in water from the Great North American Lakes ranging from 1.1 ng/l in Lake Superior to 0.17 ng/l in Lake Ontario. Swackhammer *et al.* also determined recent suspended particulate concentrations of toxaphene of 9.4 and 6.3 ng/l for Lake Michigan and Lake Ontario. Muir et al. (1997) dertermined toxaphene residues in water, sediment, zooplankton and fish in a Canadian lake. Residues of total toxaphene and individual toxaphene congeners with increasing trophic level within the foodweb. Water and sediment concentrations of total toxaphene amounted to 209 pg/L and 360 pg/g dw, respectively.
Toxaphene in rainwater is scarcely measured. Saleh (1991 in TNO, 1999) reported an average toxaphene level of 28 ng/L in rainfall at pristine sites in South Carolina in the period of July 1981. This was 80 times higher than any other orgnochlorine analysed in those samples.

Muir et al. (1997 in TNO, 1999) studied concentrations of 2 selected congeners and total toxaphene in sea water, zooplankton, fish and sea mammals in a Canadian arctic sea. Biomagnification was demonstrated with much higher residue levels in beluga (whale species) than in seals although both feed at the same trophic level. In the investigated system the water concentration of toxaphene ranged from 35 to 100 pg/L.
Toxaphene residues are demonstrated in fish from the North Sea, with an upward trend from the southern to the northern North Sea (De Geus et al., 1999 in TNO, 1999). The measured toxaphene residues in herring and mackerel from the North Sea and remote waters west and Northwest from Ireland and the Shetland islands exceeded the German tolerance level of 0.1 mg/kg on fat basis. Marine fish from Danish water had concentrations in the range 5 to 50 ng/g fat.
Toxaphene levels in fish from the Great Lakes and in Arctic marine mammals (up to 10 and 16 $\mu g\ g^{-1}$ lipid). Toxaphene concentrations in North sea fish are at least 10-fold lower than in fish from Arctic and Canadian waters and vary from 1 to 600 µg/kg wet weight (De Boer, 1997 in TNO, 1999).
Profiles for toxaphene congeners may vary between those for technical mixture, sediment, invertebrates, fish, mammals. Toxaphene profiles found in trout, shrimp and sediment in toxaphene treated lakes were complex and comparable. However mammals like seals are known to rapidly metabolise most chlorobornanes. Toxaphene profiles in fish vary considerable depending on source characteristic, geographic location, species and age (TNO, 1999).
The composition of toxaphene residues in environmental samples can differ widely. However Angerhöfer et al. (1999 in TNO, 1999) noted that in samples of fish species or fish products from North Atlantic and North sea toxaphene peak patterns are remarkably similar with only 20-25 dominant congeners. The main mass of these residues are represented by 6 compounds. Three indicator congeners for toxaphene were more often selected for analysis of in fish caught in several European coast water. These congeners are B[12012]-(202), B[12012]-(212) and B[30030]-(122). These 3 congeners comprised a major portion of the toxaphene residues in cod liver oil (25-30%) and fresh fish (8-12%) from Northern and Western European coastal waters. Highest residues were found in marine fish with moderate and high fat content (Wells and De Boer, 1997 in TNO, 1999).
According to a field study by Kidd et al. (1995 in TNO, 1999) biomagnification of toxaphene in long food chains can result in concentrations in fish which are hazardous to human health. The extent of biomagnification is expected to be higher at arctic and subarctic latitudes.

Table 2 Occurrence in the environment of toxaphene

Compartment	Year	Time	Location	Concentra-tion	Unit	Reference (source)
Wildlife biota	1994		Rhine Netherlands Zebra mussel Dreisena polymorpha (whole)	0.0047	µg/g ww total toxaphene	Hendriks, et al, 1998 en De Geus et al, 1999 in TNO, 1999
Wildlife biota	1994		Meuse Netherlands Zebra mussel Dreisena polymorpha (whole)	0.036	µg/g ww total toxaphene	Hendriks, et al, 1998 en De Geus et al, 1999 in TNO, 1999
Wildlife biota	1994		Ysselmeer Netherlands Zebra mussel Dreisena polymorpha (whole)	0.00074	µg/g ww total toxaphene	Hendriks, et al, 1998 en De Geus et al, 1999 in TNO, 1999
Wildlife biota	1994		Rhine Zebra mussel Dreisena polymorpha (whole)	0.012	µg/g ww total toxaphene	Hendriks, et al, 1998 en De Geus et al, 1999 in TNO, 1999
Wildlife biota	1994		Meuse Netherlands Eel Anguilla anguilla (fillet)	0.02	Mg/g ww total toxaphene	Hendriks, et al, 1998 en De Geus et al, 1999 in TNO, 1999
Wildlife biota	1989		Rhine Netherlands Eel Anguilla anguilla (muscle)	0.3	Mg/g lipid total toxaphene	Hendriks, et al, 1998 en De Geus et al, 1999 in TNO, 1999
Wildlife biota	1989		Yssel Netherlands Eel Anguilla anguilla (muscle)	0.09	µg/g lipid total toxaphene	Hendriks, et al, 1998 en De Geus et al, 1999 in TNO, 1999
Wildlife biota	1988		Gulf of Finland Atlantic cod Boreogadus saida (liver)	0.64	µg/g lipid total toxaphene	De Geus et al, 1999 in TNO, 1999
Wildlife biota	1989		Vester Tana Atlantic cod Boreogadus saida (liver)	0.54	µg/g lipid total toxaphene	De Geus et al, 1999 in TNO, 1999
Wildlife biota	1989		South North-Sea Atlantic cod Boreogadus saida (liver)	0.4	µg/g lipid total toxaphene	De Geus et al, 1999 in TNO, 1999
Wildlife biota	1989		Central North-Sea Atlantic cod Boreogadus saida (liver)	0.6	µg/g lipid total toxaphene	De Geus et al, 1999 in TNO, 1999
Wildlife biota	1989		Northern North-Sea Atlantic cod Boreogadus saida (liver)	1.0	µg/g lipid total toxaphene	De Geus et al, 1999 in TNO, 1999
Wildlife biota	1993 ?		Germany Atlantic cod Boreogadus saida (liver)	2.45	µg/g lipid total toxaphene	De Geus et al, 1999 in TNO, 1999
Wildlife biota	1993 ?		Germany Atlantic cod Boreogadus saida (liver)	2.73	µg/g lipid total toxaphene	De Geus et al, 1999 in TNO, 1999
Wildlife biota	-		North-Sea Atlantic cod Boreogadus saida (liver)	0.3	µg/g ww total toxaphene	De Geus et al, 1999 in TNO, 1999
Wildlife biota	1979		Galway Ireland Atlantic cod Boreogadus saida (spawn)	0.64	µg/g lipid total toxaphene	De Geus et al, 1999 in TNO, 1999
Wildlife biota	1979		Galway Ireland Atlantic cod Boreogadus saida (spawn)	0.26	µg/g wwtotal toxaphene	De Geus et al, 1999 in TNO, 1999
Wildlife biota	1993 ?		Norway Atlantic salmon Salmo salar (oil)	1.1	µg/g lipid total toxaphene	De Geus et al, 1999 in TNO, 1999
Wildlife biota	1993		Norway	0.54	µg/g lipid	De Geus et al, 1999 in

Compartment	Year	Time	Location	Concentra-tion	Unit	Reference (source)
	?		Atlantic salmon Salmo salar (oil)		total toxaphene	TNO, 1999
Wildlife biota	-		Ireland Hake Merluccius (liver)	0.9	µg/g ww total toxaphene	De Geus et al, 1999 in TNO, 1999
Wildlife biota	1978		Baltic Herring Clupea harengus (muscle)	13	µg/g lipid total toxaphene	De Geus et al, 1999 in TNO, 1999
Wildlife biota	1989		Southern North-Sea Herring Clupea harengus (muscle)	0.4	µg/g lipid total toxaphene	De Geus et al, 1999 in TNO, 1999
Wildlife biota	-		Skaggerak Herring Clupea harengus (muscle)	0.04	µg/g ww total toxaphene	De Geus et al, 1999 in TNO, 1999
Wildlife biota	1989		German Bight Plaice Pleuronessa platessa (liver)	0.1	µg/g lipid total toxaphene	De Geus et al, 1999 in TNO, 1999
Wildlife biota	1989		Skagerrak Plaice Pleuronessa platessa (muscle)	0.013	µg/g ww total toxaphene	De Geus et al, 1999 in TNO, 1999
Wildlife biota	-		North-Sea Twait shad Alosa fallax (muscle)	0.02	µg/g lipid total toxaphene	De Geus et al, 1999 in TNO, 1999
Wildlife biota	-		North-Sea White-beaked dolphin (blubber)	19	µg/g lipid total toxaphene	De Geus et al, 1999 in TNO, 1999
Food			Haring North-sea	1-20	µg/kg ww	Alder 97 in DHC99
Food	1991		Dab in Wadden-sea	1-10	µg/kg ww	De Boer &Wester 93 in DHC99
Food	1991-92		Fish in south North-sea	2-200	µg/kg ww	De Boer &Wester 93 in DHC99
Food	1990		Harbour porpoise blubber North-sea	6800	µg/kg ww	De Boer &Wester 93 in DHC99
Food	1990		Witsnuitdolfijn North-sea	19000	µg/kg ww	De Boer &Wester 93 in DHC99

d.l= detection limit

Legal status

Toxaphene is listed on the OSPAR candidate list and HELCOM priority list.

Conclusion

Toxaphene is used on food crops, is persistent and bioaccumulative and found in biota (fish=food) and human mother milk. Exposure through food is very likely because the substances are persistent and found in food. Toxaphene is already forbidden (as plant protection product) to be used in the EU but is found to be transported by long boundary transport through air and is still found in biota and mother milk. The substance is prioritised as having high concern for exposure.

References

DHC99: Dutch Health Council (1999). Endocrine-disrupters in the Netherlands.

Fra97: Franse & de Voogt, 1997 Oestrogene verbindingen in het Nederlands milieu, MTC report.

Gre96: Greve, (1996). (Dutch Health Council). Hormoon-verstorende stoffen in Nederland. Gebruik, emissie, milieuconcentraties en fysisch/chemische karakteristieken

Gülden, M., et al, (1998), Endocrinically active chemicals and their occurrence in surface waters, UBA-FB 97-068, Research report 102 04 279

RIWA (1998), Xeno-oestrogenen en drinkwater(bronnen).

TNO, 1999, Water System Evaluations – An analysis of possible problems in the aquatic environment, Toxaphene, Draft version of report no, 30622, for RIKZ.

Tributyltin

The substance was selected to be evaluated in the expert meeting because it is a HPV chemical, which is produced in more than 1000 tonnes/year, and because they are metals.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 1 and the Human health relevant endocrine disruption data as category 2.

Chemical characteristics

Table 1 Physico chemical properties of tributyltin

Substance	TBTO [A]	Tributyltin
Physical state	Colourless to yellow liquid	
Water solubility	0.75 mg/l (WHO, 1999) 1.5 mg/l (gre96) 1.9 mg/l (4 riwa) 71.2 mg/l at 20 C (OECD105) scher98 in iuclid96 (s5))	-
Vapour pressure	0.085-16 mPa at 20 C (blu84 in iuclid96 (v5))	-
Henry coefficient	0.02 Pa.m3/mole (gre96)	-
Log Koc	4.6	4.6
Log Kow	3.2-3.8 (mag83 and lau86 in iuclid96 (k5 and k6))	3.8 3.62 (1 riwa)

[A] In this document, the term TBTO is used when that specific chemical is intended. In the environment, however, tributyltin compounds are expected to exist mainly as tributyltin hydroxide, tributyltin chloride, and tributyltin carbonate. In those cases or when the identity of the specific chemical is not clear, the general term tributyltin is used (WHO, 1999).

Water solubility and lipophilicity are also dependent on pH and ion content of the water. For TBTO, for example, a pH-dependent water solubility of between 0.75 and 60 mg/l was measured. For tributyltin compounds in general, a water solubility of <1 to >200 mg/l has been calculated.

TBT dissociates in water and forms hydrated tributyltin cations (TBT+), which can react with various anions (e.g. OH-, Cl-, HCO3). The type and percentage of TBT species found in water is dependent on pH and the ion content of the water. In seawater, for example, a pH-dependent balance between TBT+ and TBTCl, TBTOH, TBTHCO3 has been found. At pH < 7, TBT+ and TBTCl are primarily present, at pH 8 a mixture of TBTCl, TBTOH and TBTCO3 (Gulden, 1998).

Bioaccumulation

Octanolwater distribution coefficients of log Kow =3.19 (pH 6) - 3.85 (pH 7.8) have been measured for TBTO and of log Kow = 3.2 (pH5.8) - 4.1 (pH 8) for TBTCI (Gulden, 1998) and is 3.54 for seawater (WHO, 1999).

The high log Kow indicates that TBTO has a bioaccumulative potential. This is confirmed by experimental bioconcentration coefficients reported of 100 - 30,000 for bacteria living in the outfall river, 8,600-30,000 for green algae (*Ankistrodesmus falcatus*), 5,500 for diatom (*Isochrysis galbano*), 2,000- 6,000, 2,300-11,400 and 2,400-7,800 for oyster (*Crassostrea gigas*) and 1,000 - 7,000 for blue mussel (*Mytilus edulis*), respectively (Japan, 1997). The BCF for fish (*Cyprinodon variegatus*) is 2600 (total fish) and in liver 20000-52000. For the mollusc *Ostrea edulis* the BCF is 1000-1500 and for the crustacean *Rhithropanopeus harrisii* 500-4400 (fed) and 100-200 (not fed) (bua88 in iuclid96 (b8-B15)). Another publication reported a range of biomagnification factors in marine mammals of 0.6~6.0 (Madhusree et al., 1997 in WHO, 1999).

Although it has been suggested that tributyltin accumulates in organisms because of its solubility in fat (IPCS, 1990), recent work suggests that this might not be the case. Although tributyltin residues in blubber of marine mammals have been reported (Iwata et al., 1994, 1995, 1997), levels were considerably higher in other tissues, notably liver (Iwata et al., 1994, 1995, 1997; Kannan et al., 1996, 1997, 1998; Kim et al., 1996a,b; Madhusree et al., 1997; Tanabe, 1998; Tanabc et al., 1998). Comparison of patterns of tributyltin residues with those of fat-soluble organochlorines in marine manunals showed marked differences. Unlike the organochlorines, tributyltin residues were the same in both sexes and remained constant after animals reached maturity. It has been suggested that transfer through milk to offspring, a marked trend with the organochlorines, does not occur with tributyltin. Cetaceans showed greater bioaccumulation than pinnipeds (Kim et al., 1996c). There has also been a report of accumulation in liver and kidney of seabirds (Guruge et al., 1997). Stäb et al. (1 996) recently determined organotin compounds in the food web of a shallow freshwater lake; in birds in the food web, the highest concentrations of organotin compounds were also in liver and kidney, not in subcutaneous fat. The various authors cited suggest protein binding in liver to be the major mechanism of bioaccumulation (WHO, 1999)

TBTO is adsorbed to the surface of particles in water, sediment and soil. The values given for sediment/water distribution coefficients vary widely (Fent, 1996: 0.34 - 64 x 10^4 BUA, 1988: 0.11 - 55.4x10^4 which is probably due to differing experimental conditions (e.g. pH, salt content, concentration, type, size and organic carbon content of the suspended particles). Thus, TBT is accumulated in sediment. Suspended particle type and content determine the percentage of particle-bound TBT in the water. Measurements by Kalbfull et af.(1991) in limnic harbors revealed a suspended particle bound content of TBT of between 9% and 94% (Gulden, 1998, WHO, 1999).

Degradation

Progressive disappearance of adsorbed TBTO is due to degradation, **not** desorption (WHO, 1999). The degradation of TBTO involves the splitting of the carbon-tin bond (IPCS, 1990). This can result from various processes - both physicochemical (hydrolysis and photodegradation and biological degradation by microorganisms and metabolism by higher organisms) - occurring simultaneously in the environment (WHO, 1999).

The Degradation pathway of tributyltin in the environment (stepwise debutylation) is shown as follows. Theoretically the degradation is completed by release of oxidized tin in water.

Degradation of TBTO proceeds by cleavage of carbon-tin bond and the mechanism of its cleavage is considered to be two-fold, physicochemical (hydrolysis, photodegradation) and biological (degradation by microorganisms, metabolism by higher living organisms).

Photodegradation of TBTO is theoretically possible. There are a number of different informations on the rate of photolysis depending on the conditions, giving $t_{1/2}$ from 11 days to more than 144 days. In distilled water, TBTO is degraded by natural, long - wave, or short - wave light with half-life times of 89, 18 and 1.1 days, respectively. Under natural conditions (lighting relationship, light adsorption in water) the photolytic degradation is, with values of more than three months, not very efficient.

Microorganisms, particularly bacteria have degrading activity, and the mechanism of degradation depends on the environmental conditions (temperature, pH and presence of easily degraded organic compounds). The DT50 in water is estimated to be >1 month (riwa, 1998). Expected removal from sewage treatment plant >90% by active coal (riwa, 1998).

The mechanism of biodegradation is proposed as follows:

Products of hydroxylation derivatives cleavage of carbon-tin binding (production of dibutyl derivatives) production of monobutyl derivative

$(Bu_3Sn)_2OBu_3SnOH$dibutyl derivativemonobutyl derivative

In another report the following mechanism of biodegradation is proposed:

R_3SnX $(R_3Sn)_2O$ $(R_3Sn)_2CO_3$

UV or microorganism
$(R_2SnO)_n$
UV or microorganism
$(RSnO^-)_n$
UV or microorganism
SnO_2

Although anaerobic degradation occurs, there is a lack of agreement as to its importance; some consider it to be slow, whereas others believe that it is more rapid than aerobic degradation. Species of bacteria, algae, and wood-degrading fungi have been identified that can degrade TBTO. Estimates of the halflife of tributyltin in the environment vary widely. The half-life in the water column ranges from a few days to weeks. In sediment TBT is slowly degraded. A half-life of 4- 5 months has been reported for the aerobic layer, and more than 500 days for the anaerobic layer (Gulden, 1998). Tributyltin may persist in sediments for several years (WHO, 1999).

Metabolisation
Little definitive information is available on the pharmacokinetics of TBTO. TBTO is absorbed from the gut (20-50%, depending on the vehicle) and via the skin of mammals (approximately 10%). Other data suggest absorption in the 1-5% range via the skin. TBTO can be transferred across the blood-brain barrier and from the placenta to the fetus. Following 14 days of oral administration, steady-state levels in tissue are reached after 3-4 weeks. Absorbed material is rapidly and widely distributed among tissues (principally the liver and kidney). Metabolism in mammals is rapid; metabolites are detectable in blood within 3 h of TBTO administration. The principal metabolite appears to be the hydroxybutyl compound, which is unstable and rapidly splits to form the dibutyl derivative and butanol (WHO, 1999).

Tributyltin metabolism also occurs in lower organisms, but it is slower, particularly in molluscs, than in mammals. The capacity for bioaccumulation is, therefore, much greater in lower organisms than in mammals. There are some recent preliminary data (Takahashi et al., 1998, in WHO, 1999) on the occurrence of total butyltin residues in human liver. The average concentration in four samples was 84 ng/g wet weight (range 59-96 ng/g in concentration of tributyltin was less than the detection limit of 2 ng/g. The concentration of dibutyltin was 79% of the total (WHO, 1999).

Use, Exposure and emissions

Tributyltin compounds have been registered as molluscicides, as antifoulants on boats, ships, quays, buoys, crab pots, fish nets, and cages, as wood preservatives, as slimicides on masonry, as desinfectants, and as biocides for cooling systems, power station cooling towers, pulp and paper mills, breweries, leather processing, and textile mills. TBT in antifouling paints was first marketed in a form that allowed free release of the compound. More recently, controlled-release paints, in which the TBT is incorporated in a co-polymer matrix, have become available. Rubber matrices have also been developed to give long-term slow release and lasting effectiveness for anti-fouling paints and molluscicides In this form, much of the TBT remains in the matrix of the rubber, though the effectiveness lasts for several years (EHC116). TBT is not used in agriculture because of high phytotoxicity (EHC116).

Production and consumption
The world consumption of tin in 1976 was estimated to be 200 x 10^3 tonnes, of which 28 x 10^3 tonnes was organotin. Approximately 40% of the total was consumed in the USA (Zuckerman et al., 1978). The United Kingdom Department of the Environment (1986) reported that the worldwide use of organotin in 1980 was 30 x 10^3 tonnes (EHC116).
This total was made up as follows:
* PVC stabilizers (dibutyl), approximately 20 x 10^3 tonnes;

* wood preservatives (tributyl), 3-4 x 10^3 tonnes;
* antifouling paints (tributyl), 2-3 x 10^3 tonnes;
* other uses of both di- and tri-butyltin, < 2 x 10^3 tonnes.

The total production in Europe was 1-10000 tonnes/year in 1987 (gre96). It is also reported that the annual world production of TBT compounds is estimated to be 4000 to 5000 tonnes (Organotin Environmental Programme Association (ORTEPA); personal communication to IPCS, 1989 in EHC116)). The production was 1300 tonnes/year in 1990 and 1994 in the Netherlands (RIWA, 1998 and gre96).

The total annual use (production and imports) of organotin compounds in Canada was reported by Thompson et al. (1985) to be in excess of 1 x 10^3 tonnes. The total annual production of TBTO in the Federal Republic of Germany is reported to be 2 x 10^3 tonnes, of which 70% is exported. National usage is as follows: 70% antifouling paints; 20% timber protection; 10% textile and leather protection; small amounts are also used as a preservative in dispersion paints and as a disinfecting agent. Annual tin emissions are reported to be less than 300 kg (TWG, 1988a). Annual TBT use in the Netherlands in 1985 was reported to be 1.5 x 10^4 kg for wood preservation and 10 x 10^4 kg for antifouling paints (TWG, 1988c). Organo-tin antifoulant use in Norway was 13.7 x 10^4 kg in 1986 for the treatment of nets and sea pens at approximately 600 fish farms (Linden, 1987). In Japan, usage was estimated at 1300 tonnes in 1987, of which two-thirds was used for antifouling paints on vessels and one-third for antifouling of nets in fish culture (EHC116).

A survey of total and retail sales of TBT-containing paints and antifouling preparations for nets was carried out in Finland in 1987. Of a total of 42 000 litres, 37 000 litres were sold retail. The concentration of TBT in the antifouling paints was 4-18%. The previous use of TBT as a slimicide or fungicide (estimated at 2.1 tonnes per year during the period 1968-1970) has been discontinued. The estimated sale of wood reservatives containing TBT was 130 tonnes in 1987; these contained between 0.9 and 1.8% of TBT. Champ & Pugh (1987) reported that about 300 TBT antifouling paints were registered in the USA in 1987, but only about 17 paints are now registered for use (US EPA; personal communication to IPCS, 1989 in EHC116). MAFF/HSE (1988) listed 345 different wood preservative formulations, 24 surface biocides and 215 antifouling paints containing TBT with registration approval for use in the United Kingdom under the Control of Pesticides Regulations. In 1989, the number of antifouling paints containing TBT registered for use in the United Kingdom had fallen to 148, with the number of wood preservatives and surface biocides remaining about the same (337 and 26 registered products, respectively) (MAFF/HSE, 1989 in EHC116).

Commission of the European Communities (EC) in 1991, and most non-EC members in Europe prohibited the use of TBT-based paints on vessels less than 25 m in length.
Since 1990 in the FRG, the use of all tinorganic compounds in antifouling paint for boats up to 25 meters long, i.e. in practice primarily sport boats, and for the preparation and conservation of water , e.g. cooling and processing water, has been forbidden (Chemical Ban Ordinance) (Gulden, 1998).

TBT is transferred to surface waters primarily through the leaching out of antifouling paint; TBT used as material protection also enters through municipal waste water (Gulden ,1998).

Vulnerable use and vulnerable groups
Recent preliminary data (Takahashi et al., 1998) suggest the potential for non-food sources of exposure for example, consumer products such as rubber gloves and baking sheets (WHO, 1999).
It is unknown in what extend TBT can be adsorbed from these non-food sources in to humans. Other routes of exposure may be through food-intake, mainly fish, shellfish and crustaceans.

Dutch scientists first recognized the biocidal properties of triorganotin compounds in the 1950s; major production and use of these substances dates from this period. It was found that the different triorganotin compounds have different toxicities to different organisms. Tributyltin compounds were

found to be the most toxic of the triorganotins to gram-positive bacteria and to fungi. They were also found to have biocidal properties to a wide spectrum of aquatic organisms (EHC116).
The enhancement of tributyltin concentrations in the surface microlayer may present a hazard to littoral organisms, neustonic species (including benthic invertebrate and fish larvae), and surface-feeding seabirds and wildfowl. Accumulation and low biodegradation of tributyltin in sediment may pose a hazard to aquatic organisms when these polluted sediments are disturbed by natural processes or dredging activities (WHO, 1999).

Environmental concentrations

In 1987 - 1990, before the ban on the use of antifouling paint containing TBT for boats less than 25 m long, the Bavarian lnstitution for Water Research, under the auspices of a research grant from the German Environmental Agency, studied TBT concentrations in water and sediments from various yacht harbors in German waters (Unterelbe, Wannsee, Tegelsee, Bodensee) (Kalbfut3 et al., 1991 in Gulden, 1998).

More recent data (collected up to the mid- 1990s) have documented a decline in tributyltin levels in the environment, presumably due to the restrictions placed on the use of antifouling paints on vessels (CEFIC, 1994; Ruiz et al., 1996; Stronkhorst, 1996; Tolosa et al., 1996; NIVA, 1997; dela Cruz & Molailder, 1998 in WHO, 1999).

There are a number of reports on the occurrence of tributyltin residues in marine organisms. Levels of total butyltin residues (the sum of detected tributyltin, dibutyltin, and monobutyltin) of 5-230 ng/g in muscle of fish (Kannan et al., 1995, 1996, 1997 in WHO, 1999), 300 ng/g in liver and kidney of marine birds (Guruge el: al., 1997), and 13-395 ng/g in muscle of marine mammals have been reported (Iwata et al., 1994, 1995, 1997; Kannan et al., 1997). In marine mammals, much higher total butyltin residues were reported for blubber (48-744 ng/g), kidney (25-3210 ng/g), and liver (40-11340 ng/g) (Iwata et al., 1994, 1995, 1997; Kannan et al., 1996, 1997, 1998; Kim et al., 1996a,b,c; Madhusree et al., 1997; Tanabe, 1998; Tanabe et al., 1998). Geographical comparisons showed greater accumulation of residues close to coasts compared with the open sea and in the vicinity of developed compared with developing countries (WHO, 1999).

Information on tributyltin concentrations in various media that are relevant to estimation of human exposure is extremely limited, being restricted to data from Japan. It is unknown if this information is representative of other areas, and additional investigation is desirable.
Tsuda et al. (1995) investigated the daily intakes of tributyltin compounds from meals in Shiga Prefecture, Japan. Daily intakes of TBTO determined by the duplicate-portion method were 4.7 ± 7.0 µg/day in 1991*(n* = 39) and 2.2 ± 2.2 µg/day in 1992 (n = 40). Using the market basket method, the daily intake was estimated at 6-9 µg/day in 1991 and 6-7 µg/day in 1992. The TBTO was found mostly in seafood (WHO, 1999).
Market basket studies in 10 local regions in Japan have shown that the national average daily intake of tributyltin (expressed as tributyltin chloride) was 3.7, 9.9, 5.4, 3.6, 2.9, 1.6, 1.5, and 2.3 µg/day in 1990, 1991, 1992, 1993, 1994, 1995, 1996, and 1997, respectively. Variation among the local regions reflects differences food intake pattems as well as differences in tributyltin levels in local fisheries (WHO, 1999).

Table 2 Occurrence in the environment of tributyltin

Compartment	Year	Substance	Location	Concentration average (max.)	Unit	Reference (source)
Water Sediment Fishes Shellfishes Birds	1994	TBT	Japan	0.000003 - 0.00003 (35/99) 0.0008 – 0.440 (87/102) 0.10 - 0.17 (15/70) 0.05 - 0.10 (6/30) - (0/5)	ppm	Japan, 1997
Water Sediment Fishes Shellfishes Birds	1994	TPT	Japan	0.000005 - 0.00001 (4/92) 0.001 – 0.26 (44/88) 0.03 - 0.28 (28/70) 0.03 - 0.04 (5/30) - (0/5)	ppm	Japan, 1997
Water	<1990	TBT	Harbors Germany	<0.005-0.93 Mean 0.2	µg/l	Gulden, 1998
Water	1989		Harbors Rhine near Mainz Germany	0.024 – 0.178	µg/l	Gulden, 1998
Water	1992	TBTO	Lakes and rivers (NL)	0.0013 (0.0013)	µg/l	Gre96
Water	1992	TBTO	North-sea coast (NL)	0.03 (0.09)	µg/l	Gre96
Water	1992	TBTO	Wadden-sea (NL)	0.015 (0.02)	µg/l	Gre96
Water		TBT	Rhine (NL)	<0.01	µg/l	67 riwa, 1998
Water		TBT	Meuse (NL)	<0.01	µg/l	67 riwa, 1998
Water		TBT	Stagnant waters (NL)	1	µg/l	67 riwa, 1998
Water		TBT	Rhine Mainz (Germany)	No detection		65 riwa, 1998
Water		Mono, dibutyltin	Rhine Mainz (Germany)	0.5-2.3	ng/l	65 riwa, 1998
Water	1995/96	TBT	Bayern Germany	<0.002-0.0029	µg/l	Gulden, 1998
Water	<1998	TBT	Coastal waters and estuaries	1-10	ng/l	WHO, 1999
Water	<1998	TBT	Marine ports	20-460	ng/l	WHO, 1999
Effluent	1996	TBT	Bavarian STPs Germany	28-180	ng/l	Gulden, 1998
Sediment		TBT	Rhine Lobith (NL)	<122	µg TBT/g dryweight	22 riwa
Sediment	<1990	TBT	Harbors Germany	11-7600	µg/kg ww	Gulden, 1998
Sediment	1989	TBT	Harbors Rhine near Mainz Germany	32-443	µg/kg	Gulden, 1998
Sediment	1995	TBT	Bayern, Germany	<10-23	µg/kg dwt	Gulden, 1998
Sediment	1992	TBT	Niedersachsen Germany	17-877	µg/kg dwt	Gulden, 1998
Suspended matter	1994	TBT	Nordrheinwestfalen Germany	77	µg/kg dwt	Gulden, 1998
Suspended matter	1995	TBT	Rivers Hessen Germany	<7-108 avg 20-50 (10/12)	µg/kg dwt	Gulden, 1998
Sediment	<1990	TBT	Harbors Germany	11-7600	µg/kg ww	Gulden, 1998
Sediment	1989	TBT	Harbors Rhine	32-443	µg/kg dwt	Gulden,

Compartment	Year	Substanc e	Location	Concentration average (max.)	Unit	Reference (source)
			near Mainz Germany			1998
Sediment	<1998	TBT	Coastal water and estuaries	Most < 100 some >1000	µg/kg dwt	WHO, 1999
Sediment	<1998	TBT	Port Sweden	10 940	µg/kg dwt	WHO, 1999
Wildlife biota	<1998	TBT	Biota	0.01 – 3	mg/kg	WHO, 1998

d.l= detection limit

Legal status

Tributyltin is listed on the OSPAR candidate list and HELCOM priority list.

Conclusion

Human exposure may take place through food intake (mainly seafood) and potentially from non-food consumer products such as rubber gloves and baking sheets (WHO, 1999). It is unknown in what extend TBT can be adsorbed from these non-food sources in to humans.

In the environment tributyltin compounds were found to be the most toxic of the triorganotins to gram-positive bacteria and to fungi. They were also found to have biocidal properties to a wide spectrum of aquatic organisms (EHC116). The enhancement of tributyltin concentrations in the surface microlayer may present a hazard to littoral organisms, neustonic species (including benthic invertebrate and fish larvae), and surface-feeding seabirds and wildfowl. Accumulation and low biodegradation of tributyltin in sediment may pose a hazard to aquatic organisms when these polluted sediments are disturbed by natural processes or dredging activities (WHO, 1999).

TBT is considered as persistent in the environment. Although concentrations in the waterphase are decreasing due to restricted use, TBT is still measured in water, sediment, suspended solids and wildlife, indicating a high concern for exposure.

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Tri-n-propyltin

The substance was selected to be evaluated in the expert meeting because it is a metal.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 1 and the Human health relevant endocrine disruption data as category 3.

Chemical characteristics

Table 1 Physico chemical properties of tri-n-propyltin

Water solubility	233.4 mg/l (Syr, 1996)
Vapour pressure	1.97 (Syr, 1996)
Log Kow	2.13 (Syr, 1996)

Tri-n-propyltin is relatively soluble in water. It is rapidly biodegradable (Syr, 1996)

Use, Exposure and emissions
No information available

Vulnerable use and vulnerable groups
No information available

Environmental concentrations
No information available

Legal status
tri-n-propyltin is referred Council Directive 67/548/EEC relating to the classification and labelling of dangerous substances.

Conclusion
Due to lack of information, it is worst case assumed that there is a high concern for exposure.

References

Triphenyltin

The substance was selected to be evaluated in the expert meeting because it is a metal.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 1 and the Human health relevant endocrine disruption data as category 3.

Chemical characteristics

Table 1 Physico chemical properties of triphenyltin

	Triphenyltin hydroxide (TPTH)	Triphenyltin acetate (TPTA)	Triphenyl tin chloride (TPTCl)	Triphenyltin TPT
Water solubility	1 mg/litre at pH 7 at 20C (WHO, 1999)	9 mg/l at 20C,pH 5 (WHO, 1999)	40 mg/l at 20C (WHO, 1999)	0.0026 mg/l (gre96)
Vapour pressure	0.047 mPa (50'C) (WHO, 1999)	1.9 mPa (60 'C) (WHO, 1999)	0.021 mPa	-
Henry coefficient				0.00675 Pa.m3/mole (gre96)
Log Koc				3.67 (gre96)
Log Kow	3.43	3.43	-	3.8 (gre96)

Physical and chemical properties of triphenyltin compounds vary depending upon the anion linked to tin. At ambient temperatures in the pH range of 3-8, TPTA and TPTCl are hydrolysed to TPTH within 1 min; as a consequence, the results of most studies with TPTA or TPTCl can be applied to TPTH. Triphenyltin compounds are colourless solids with low vapour pressures «2 mPa at 50°C). The compounds are lipophilic and have low water solubility (typically a few mg/litre at neutral pH).

Hydrolysis of triphenyltin compounds in water leads to the formation of principally TPTH and various hydrated oxides (Beurkle, 1985). It has been demonstrated that the presence of chloride from seawater lowers the solubility of triphenyltin compounds by reaction with the hydrated cation to form the covalent organotin chloride (Ozcan & Good, 1980 in WHO, 1999).

Bioaccumulation

Bioconcentration factors in fish exposed to maintained concentrations of triphenyltins are high, which might be expected from the log Kow > 3. Peak BCF were reached 56 and 112 days after the start of exposure ranging from 3500 to 4700 for whole fish. The active substance depurated relatively slowly (Agrevo, 1999).

An extensive study on the presence of nine organotin compounds in a freshwater food-web (zebra mussel, eel, roach bream, pike, perch, pike perch, and cormorant) revealed that phenyltin concentrations in benthic species were higher than butyltin concentrations in lower trophic levels (Staeb et al., 1996). This suggests that triphenyltin is to a large extent taken up from the sediment by benthic organisms. At higher trophic levels, net bioaccumulation of triphenyltin compounds was greater than that of tributyltins resulting in relatively higher triphenyltin concentrations. With birds, the highest concentrations of organotins were in liver and kidney and not in subcutaneous fat, which shows that organotins accumulate via mechanisms different from those of traditional lipophilic compounds. BCFs in daphnids did not exceed 300 (Filenko & Isakova, 1979). In fish, BCFs ranged from 257 to 4100. The highest value (4100) was estimated for filefish (*Rudarius ercodes*) cultivated in water containing 148 ng triphenyltin/litre for 56 days (Yamada & Taka- yanagi, 1992). When *Lymnaea stagnalis* (a freshwater same snail) was exposed to 2 µg TPTH/litre for 5 weeks, tin accumulated to the greatest extent in the intestinal sac, to a level of 65.1 mg/kg (i.e., BCF of 32500;

Van der Maas et al., 1972). Tissue concentrations of triphenyltin in common carp (*Cyprinus carpio*) exposed to 5.6 µg TPTCI/litre for 10 days, which reached a plateau after 7 days, were exarnined. The BCFs were highest in the kidney (2090), followed by liver (912), muscle (269), and gall bladder concentrations (257) (Tsuda et al., 1987).

Half-lives of triphenyltin in mussels *(Mytilus cdulis)* taken in the summer of 1989 in Yokohama (a busy port, heavily contaminated with triphenyltin) and Urayasu (a river mouth, about 10 times less polluted than Yokohama) in Japan were estimated to be 139 and 127 days, respectively (Shiraishi & Soma, 1992). Biological half-lives of triphenyltin in short-necked clams *(Tapes Amygdalaljaponica)* and guppy *(Poecilia reticulata)* were estimated to be approximately 30 days and 48 days, respectively (Takeuchi et al., 1989; Tas et al., 1990). The ecological half-life of triphenyltin in gastropods was estimated to be 347 days (Mensink et al., 1996).

In plants, no translocation occurs from treated leaves (FAO, 1991 a). TPTA and TPTCI are spontaneously hydrolysed to form TPTH. Phenyl groups are split off from TPTH to form diphenyl and monophenyl compounds. Both parent compound and metabolites conjugate to form glycosides or glutathione conjugates.

Degradation

Degradation of triphenyltin occurs through sequential dephenylation resulting from cleavage of the tin-carbon bond through biological, ultraviolet irradiation, chemical, or thermal mechanisms; biological cleavage and cleavage by ultraviolet irradiation are considered to be the most significant processes. Abiotic factors, such as elevated temperatures, increased intensity of sunlight, and aerobic conditions, seem to enhance triphenyltin degradation in the environment (CICAD National Committee, 1997).

Aerobic degradation of triphenyltins in soil in laboratory studies occur with half lives of approx. 20 days. Anaerobic degradation occurs more slowly, with an estimated half live of 40 days (Agrevo, 1999).

The adsorption/desorption studies and the column leaching studies indicate that triphenyltins are of low mobility. In sediment water studies conducted in the dark, the rate at which triphenyltins partition out of water and into sediment was rapid (1.4 days), indicating that in practice triphenyltins are unlikely to remain in the water column (Agrevo, 1999).

The persistence of TPTA and TPTH depends on soil type and pH. TPTH is strongly adsorbed to sediment and soil, and little desorption occurs. Therefore, uptake into plants via roots may be expected to be extremely low.

14C-labelled TPTA in soil degraded to inorganic tin with evolution of 14C-labelled carbon dioxide. Similar experiments on sterile soil showed insignificant evolution of labelled carbon dioxide, which suggests that degradation can be attributed to microorganisms (Bames et al., 1971 in WHO, 1999). Soil respiration was slightly enhanced after treatment with TPTA, indicating that there were no adverse effects on aerobic microorganisms (Suess & Eben, 1973 in WHO, 1999).

A half-life of 1-3 months has been reported for TPTH in sandy and silt loam soils and 126 days in flooded silt loam (US EPA, 1987). The half-life of triphenyltin in water was estimated to be several days in June and 2-3 weeks in November (Soderquist & Crosby, 1980 in WHO, 1999).

Metabolism and distribution

Several studies have shown that TPTH orally administered to rats is eliminated mainly via the faeces, with smaller amounts in the urine. Metabolites found in faeces included di- and monophenyltin as well as a significant portion of non-extractable bound residues (the sulfate conjugates of hydroquinone, catechol, and phenol). In faeces, the major substance present was unchanged parent compound.

TPTA was rapidly and completely hydrolysed to TPTH at pH 3-8 and 23-24°C (Beurkle, 1985 in WHO, 1999).

Seven days after oral administration to rats, TPTH residues (approximately 3% of the administered dose) were distributed mainly in the kidneys, followed by liver, brain, and heart (Eckert et al., 1989; Kellner & Eckert, 1989 in WHO, 1999). Similar results were obtained after chronic exposure for 104 weeks (Dom & Wemer, 1989; Tennekes et al., 1989a in WHO, 1999).

Species differences in the metabolism of triphenyltin were investigated by Ohhira & Matsui (1996). Dearylation of triphenyltin was slower in hamsters than in rats, and pancreatic accumulation of triphenyltin was higher in hamsters. There was a good correlation between tin concentrations in the pancreas and plasma glucose levels, indicating that triphenyltin-induced hyperglycaemia depends upon the amount of tin compounds absorbed into the pancreas. Most of the tin compounds in the brains of both species were triphenyltin.

Percutaneously absorbed TPTA in guinea-pigs was distributed to the highest extent in the liver, followed by the adrenal glands, kidneys, brain, spinal cord, and pancreas (Nagamatsu et al., 1978 in WHO, 1999). Triphenyltin, diphenyltin, and monophenyltin were detected in faeces in a ratio of 15:6:2. The biological half-life of triphenyltin was estimated to be 9.4 days.

Strong adsorption of triphenyltin to soil suggests that organisms in treated soil may not be widely affected. The fact that soil respiration was not affected significantly suggests that there were no adverse effects on aerobic microorganisms (WHO, 1999).

Use, Exposure and emissions

Triphenyltin acetate is used as a non-systemic fungicide primarily used for the control of potato blight (phytophthera) (heu99 in cefic577 (u10)). Furthermore it is used as a preventive action on sugar beets, hops, and celery (FAO, 1991a), and on rice against fungal diseases, algae, and molluscs (WHO, 1999).

The major manufacturers and suppliers of triphenyltin in Europe are Hoechst Schering AgrEvo GmbH, Elf-Atochem Agri SA and Griffin LIC (supply only, not manufacture) (Agrevo, 1999).

For each triphenyltin acetate and triphenyltin hydroxide production volumes in Europe are 500 to 1000 tonnes per year. Main markets are the Netherlands, Germany and the UK. 377 tonnes of triphenyltinacetate and 11 tonnes of triphenyltinhydroxide was used in the Netherlands in 1985, 341 and 16 tonnes in 1988 resp. and 203 and 16 tonnes in 1991 resp. (Ordelman et al , 1993).
1200 tonnes of triphenyltin is produced in 1994. The emission is 2.6 tonnes Sn/year in 1988 (gre96). Triphenyltin is used for industrial purposes (gre96).

In the past triphenyltin compounds also have been used extensively as algicides and molluscicides in antifouling products since the 1960s (HSE, 1992). In that period triphenyltins entered the environment through their use in antifouling paints for boats and fishnets. Use of triorganotins in antifouling paints has been restricted in many countries because of their catastrophic effects on the oyster industry and more general effects on the aquatic ecosystem (WHO, 1999).

In Japan use of triphenyltin compounds for antifouling paints decreased from 4835 tonnes in 1983 to 346 tonnes (formulation basis) in 1989 (Sugita, 1992). Their use for antifouling paints was 40 tonnes (active ingredient) in 1989 and stopped after 1990 in Japan (MITI, 1998). About 120-140 tonnes (active ingredient) were produced each year between 1994 and 1996 in Japan for export (MITI, 1998 in WHO, 1999).

Vulnerable use and vulnerable groups
TPT is used as fungicide in food crops, which may be a route of exposure for humans. Other routes of exposure may be through intake of fish, shellfish and crustaceans.

Like tributyltin enhancement of triphenyltin concentrations in the surface microlayer may present a hazard to littoral organisms, neustonic species (including benthic invertebrate and fish larvae), and surface-feeding seabirds and wildfowl. Accumulation and low biodegradation of triphenyltin in sediment may pose a hazard to aquatic organisms when these polluted sediments are disturbed by natural processes or dredging activities.

Environmental concentrations
Temporal variations of phenyltin concentrations in zebra mussels (*Dreissena polymorpha*) were studied at two locations near potato fields during and after the triphenyltin fungicide spraying season in the Netherlands (Staeb et al., 1995). Phenyltin concentrations in zebra mussels were high in the period before and during harvesting but not during the spraying season, which suggests that phenyltin compounds in some foliage ended up in the water and were taken up by the mussels. Although higher concentrations were detected in locations near areas of spray operation, marinas, and harbours, the widespread presence of triphenyltin residues in mussels collected in 56 locations all over the Netherlands suggests the contribution of transport via the air.

Biological monitoring of triphenyltin concentrations in fish from coastal areas of Japan showed that concentrations in fish, mussels and birds decreased between 1989 and 1995 (Japan Environment Agency, 1996 in WHO, 1999).
The finding of triphenyltin in coastal fish, as well as in open-ocean or pelagic fish, is suggestive of biomagnification through the foodchain. High levels in clams and oysters showed that direct uptake from water or sediment also plays an important role for these species.

Zebra mussels were used as a biomonitor to evaluate organotin pollution in Dutch fresh waters (Staeb et al., 1995 in WHO, 1999). High concentrations (1700-3200 ng tin/g dry weight) were found near locations where triphenyltin fungicide had been sprayed. Degradation products (di- and monophenyltins) were also detected in nearly all mussels.

In pecan orchards (Georgia, USA) where triphenyltin fungicides were sprayed, triphenyltin concentra tions in foliage and soils were 8.5-37 µg/g dry weight and 1.2-12 µg/g dry weight, respectively (Kannan Lee, 1996). Although triphenyltin was absent in surface soil where the fungicide had been sprayed 8-10 times a year until 2 years earlier, monophenyltin was detected at approximately the same concentration as in recently sprayed orchards.

Fish (bluegill [*Lepomis macrochirus*], largemouth bass [*Micropterus salmoides*], and channelcatfish [*Ictalurus punctatus*]) from a pond near a recently sprayed orchard contained predominantly monophenyltin (with the highest concentration of 22 µg/g wet weight in the liver of catfish) in addition to smaller amounts of triphenyltin and diphenyltin.

No data are available on occupational exposure to triphenyltin compounds. There are also no data on levels of triphenyltin in indoor or ambient air or in drinkingwater.

In supervised trials of triphenyltin formulations (wettable powder; 54%; 216-324 g active ingredient/ha) on potatoes in Germany, residues ranged from 0.3 mg/kg to less than the detection limit (0.01 mg/kg) 7 days after application (FAO, 199 la). Supervised trials of triphenyltin formulations (wettable powder; 50 or 54%; 216-324 g active ingredient/ha) in Germany on sugar beets showed residues ranging from 0. 1 to 1.9 mg/kg in leaves and less than the detection limit (0.05 mg/kg) in beets 35 days after application. In supervised trials of triphenyltin formulations (wettable powder) on rice in the USA, residues ranged from less than the detection limit (0.01 mg/kg) to 0.03 mg/kg 22-23 days after application (57.5%; 536 g active ingredient/ha, twice) and from less than the detection limit

(0.01 mg/kg) in milled rice or bran 22-46 days after application (47.5%; 250 or 500 g active ingredient/ha) (FAO, 199 la).
When 14C-labelled TPTH was administered orally to dairy cows over a period of 60 days at doses of 1.13, 5.61, or 22.44 mg triphenyltin/kg diet (dry matter), residues were 0.08, 0.31, and 0.9 g/kg in meat and 0.006, 0.026, and 0.41 mg/kg in milk, corresponding to transfer factors of 0.038-0.068 in meat and 0.004-0.006 in milk (Smith, 1981).
Triphenyltin levels were measured in fish, clams, and shrimps obtained from the Tokyo Central Fish Wholesale Market from April 1988 to March 1991. Levels were higher in cultured fish and in fish from coastal or bay areas than in pelagic fish (mean concentration 0.048 µg/g) (Takeuchi et al., 1991). Freshwater fish were relatively uncontaminated. Fish obtained from bay or inshore areas were the most contaminated; the highest concentration measured in 82 samples of four fish species was >1.0 µg triphenyltin/g muscle (mean 0.317 µg/g). Triphenyltin levels in clams and shrimps ranged from 0 to 0.83 µg/g edible portion (mean 0. 113 µg/g). Triphenyltin intake from pelagic fish was estimated based on analyses of fish samples in 1988-1991 by a market basket study in Tokyo as 3.15 µg (mean concentration of 0.048 µg/g times 65.6 g intake of pelagic fish by a Japanese person per day). Although tributyltin was used more abundantly than triphenyltin in antifouling paints, residue levels in fish and shellfish were mostly comparable, with several differences among fish groups.
National market basket studies, including the above study, have estimated daily intakes of triphenyltin per 50-kg person in Japan (expressed as TPTCl) to be 4..3, 10.4, 2.7, 0.6, 1.2, 1.4, 0.7, and 2.7 µg in 1990, 1991, 1992, 1993, 1994, 1995, 1996, and 1997, respectively (NIHS, 1998). Triphenyltin compounds were found mostly in seafood. As about a twofold difference was observed between the above estimated daily intakes (averages of 10 local laboratories, including the Shiga Prefecture) and estimated intakes in the Shiga Prefecture (Tsuda et al., 1995 in WHO, 1999), this implies that differences in food intake patterns or some other factor may influence estimates of daily intake. This fact and coincidental contamination with tributyltin must be taken into account in any risk estimation for exposure by the oral route.
Another report of a market basket survey estimated the intake of triphenyltin from raw and processed seafood in Nagasaki Prefecture (a southern part of Japan) in 1989-1991 to be 8.51 µg/day per person (Baba et al., 1991). TPTCl concentrations in fish, shellfish, seaweed, canned fish/shellfish, fish paste product, and salted/dried fish were 274, 80, 21, 12, 16, and 2.2 ng/g (averages), respectively. Cooking did not reduce the triphenyltin content of fish and shellfish samples.

Table 2 Occurrence in the environment of triphenyltin

Compartment	Year	Location	Concentration	Unit	Reference (source)
Water	<1992	Lakes and rivers	0.001-0.01	µg/l	Gre96
Water	1988-91	30 loc. Estuaries and bays Japan	2.7-8.0	ng/l	Japan, 1983-1996 in WHO, 1999
Water	1992-95	30 loc. Estuaries and bays Japan	2.5-3.0	ng/l	Japan, 1983-1996 in WHO, 1999
Water	1991	Tokyo bay, Japan	25.1 (G.Mean)	ng/l	Takeuchi, 1991 in WHO, 1999
Water	1993	Tokyo bay, Japan	1.8 (G.Mean)	ng/l	Takeuchi, 1991 in WHO, 1999
Water	1988-90	Freshwater marinas Switzerland	191 (Max)	ng/l	Fent, 1991-95 in WHO, 1999
Water	1993	Westinder lake system Nehterlands	< 5 d.l.	ng/l	Staeb, 1996 in WHO, 1996
Sediment	1993	Westinder lake system Nehterlands	2906	µg/kg	Staeb, 1996 in WHO, 1996
Sediment	1988-91	30 loc. Estuaries and bays Japan	3.3-7.8	µg/kg	Japan, 1983-1996 in WHO, 1999
Sediment	1992-95	30 loc. Estuaries and bays Japan	1.5-2.3	µg/kg	Japan, 1983-1996 in WHO, 1999
Sediment	1991	Tokyo bay, Japan	4.3 (G.Mean)	µg/kg	Takeuchi, 1991 in WHO, 1999
Sediment	1993	Tokyo bay, Japan	0.19 (G.Mean)	µg/kg	Takeuchi, 1991 in

Compartment	Year	Location	Concentration	Unit	Reference (source)
					WHO, 1999
Sediment	1988-90	Freshwater marinas Switzerland	107 (Max)	µg/kg dwt	Fent, 1991-95 in WHO, 1999
Wildlife biota	1995	Mussels Dreisena Marina Switzerland	3.88 (Max.)	mg/kg wwt	Fent, 1991-95 in WHO, 1999
Wildlife biota	1995	Mussels Mytilus Marina Spain	0.31 (Max.)	mg/kg dwt	Morcillo, 1997 in WHO, 1999
Wildlife biota	1995	Thais snails Marina Spain	0.24 (Max.)	mg/kg dwt	Morcillo, 1997 in WHO, 1999
Wildlife biota	1989	Fish muscle in coastal area Japan	2.6 (Max.) (40/65) 20/65 <20 µg/kg	mg/kg wwt	Japan, 1996 in WHO, 1999
Wildlife biota	1995	Fish muscle in coastal area Japan	0.25 (Max.) (21/70) 49/70 < 20 µg/kg	mg/kg wwt	Japan, 1996 in WHO, 1999
Wildlife biota	1989	Mussels in coastal area Japan	0.45 (Max.) (17/25) 7/25 < 20 µg/kg	mg/kg wwt	Japan, 1996 in WHO, 1999
Wildlife biota	1995	Mussels in coastal area Japan	<0.02 (d.l.) (35/35)	mg/kg wwt	Japan, 1996 in WHO, 1999
Wildlife biota	1989	Birds in coastal area Japan	0.05 (Max.) (5/10) 5/10 < 20 µg/kg	mg/kg wwt	Japan, 1996 in WHO, 1999
Wildlife biota	1995	Birds in coastal area Japan	<0.02 (d.l.) (10/10)	mg/kg wwt	Japan, 1996 in WHO, 1999
Wildlife biota		Fish near recently sprayed orchard USA	22 (d.l.) (10/10)	mg/kg wwt liver	WHO, 1999
Food	<1990	Potatoes in market	0.013-0.016 (3/25) 22/25 <0.013	mg/kg	ACP, 1990 in WHO, 1999

d.l= detection limit

Legal status
No information available on the legal status of Triphenyltin.

Conclusion
Since 1991 human exposure to triphenyltin is decreasing due to restrictions in the use as antifouling agent. TPT is used as fungicide in several crops, which may be a route of exposure for consumers. Other routes of exposure may be through intake of fish, shellfish and crustaceans.

Like tributyltin enhancement of triphenyltin concentrations in the surface microlayer may present a hazard to littoral organisms, neustonic species (including benthic invertebrate and fish larvae), and surface-feeding seabirds and wildfowl. Accumulation and low biodegradation of triphenyltin in sediment may pose a hazard to aquatic organisms when these polluted sediments are disturbed by natural processes or dredging activities.

TPT is considered as persistent in the environment. Although concentrations in the waterphase are decreasing due to restricted use, TBT is still measured in water, sediment and wildlife, indicating a high concern for exposure.

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Vinclozolin

The substance was selected to be evaluated in the expert meeting because it is a HPV chemical which is produced in more than 1000 tonnes/year.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 3 and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1 Physico chemical properties of vinclozolin

Water solubility	3 mg/l (gre96)
Vapour pressure	0.01 Pa (Teunissen-Ordelman, 1995).
Henry coefficient	1 Pa.m3/mole (gre96)
Log Koc	3 (gre96)
Log Kow	3.1 (gre96)

Vinclozolin is poorly soluble in water.
The limit value or MTR is >0.09 µg/l in the Netherlands (gre96).
The half-life of vinclozolin in water is 0.08 to 35 days (gre96).
The half life for hydrolyses is 3.8 days at pH 13, 22-70 days at pH 3, 0.9 to 2 days at pH 6 and 0.25 u at pH 9. Estimated at pH 6,7,8 it is 2-35 hours. Photolysis half life in surface water is approx. 1 day under the influence of sun light (Teunissen-Ordelman, 1995).
Since bioconcentration coefficient of vinclozolin is as small as 36, bioconcentration in the aquatic environment is anticipated not to occur (Japan, 1997). Log BCF (calc.) is 2.05 – 2.2. (Teunissen-Ordelman, 1995). Vinclozolin is not considered to persist in the environment because it is degraded in water and soil (Japan, 1997).
Little information is available on the environmental behaviour of vinclozolin (Perkow and Ploss, 1996 in Gulden 1998). It is not persistent, but is hydrolyzed in soil and water; under laboratory conditions a halflife of 26 hours in water at pH 7, 20 C, has been calculated (Szeto et al., 1989a in Gulden 1998). Primary hydrolysis products of vinclozolin include 3,5-dichlorophenyl-carbaminic acid-(1-carboxy-1-methyl)-allylester (M1) arid N-(3,5-dichlorophenyl)-2-hydroxy~2methyl-3-butteric acid amide (M2); 3,5-dichloroanitine (M3) has also been found, albeit in lesser amounts (Szeto et al., 1989a,c in Gulden 1998). The formation of M1 from vinclozolin is reversible. The formation of M1 is favored under basic conditions, while under acidic conditions the reverse reaction is favored (Szeto et al., 1989a in Gulden 1998). Vinclozolin is also rapidly metabolized to M1 and/or M2 in mammals (rats), and is then eliminated as glucuronide in urine or bile (references in Kelce et al., 1995a in Gulden 1998).

Vinclozolin is not taken up by plants through their leaves and is generally easily removed from the leaves by a light rain. Plants can, however, take up the metabolite M1 through their roots and transport it from there to the leaves, where it can be transformed back into vinclozolin (Szeto et al., 1989b in Gulden 1998).

The rates od degradation of the active ingredient and the metabolites in soil were determined in three laboratory studies. Resulting DT50 values were 1-42 days for vinclozolin, 1.5 – 82 days for m1 and 2-19 days for m2. In a field dissipation study the sum of vinclozolin and all metabolites containing the 3,5-dichloroaniline structural element was analysed. This resulted in DT50 values of 15 days at three locations (CEFIC, 1999).

Use, Exposure and emissions

Vinclozolin was introduced by BASF AG in 1975, is available under the tradename Ronilan, and has been commercially available since 1983 in plant protective agents. Vinclozolin is used as a fungicide for fruit, grapes, vegetables, hops, rape and ornamental plants (Gulden, 1998).

Vinclozolin is not produced in the Netherlands (Teunissen-Ordelman, 1995). However 1-10 tonnes/year is formulated in the Netherlands in 1986 but is not emitted because there was an waste water treatment plant (WWTP) (Teunissen-Ordelman, 1995). Vinclozolin is produced in Ludwigshafen in Germany (Teunissen-Ordelman, 1995).

The production volume of vinclozolin is in the range of 500-1000 metric tonnes/year (CEFIC 113, 1999).

56 tonnes vinclozolin was used in the Netherlands in 1985, 54 tonnes in 1988 and 38 tonnes in 1991 (Ordelman et al , 1993). The emission is 0.8 tonnes/year in 1991 (gre96).

Vinclozolin is a fungicide widely used on fruits in the US (879 in DHC99).

Vinclozolin is used for plant treatment in several cultures, for soil treatment and storage treatment (flowers) and limited for plunging treatments of bulbs. The most important use in the Netherlands is in the Onion culture (Teunissen-Ordelman, 1995).

Vulnerable use and vulnerable groups

Vinclozolin is used as a herbicide on food crops this could mean a certain risk but vinclozolin is not persistent. However its metabolites are more persistent. Vinclozolin may also present a risk to agricultural workers applying the herbicide and to workers at the production plant. Assumed is that these workers take the neccessary precautions using the substance.

Environmental concentrations

Vinclozolin is found in freshwater in 1992 and 1993, in rain water in 1988, 1989, 1990/91 and 1992. Vinclozolin is not measured in marine water and ground water vinclozolin has been measured but not been found (Ordelman, 1996).

In early 1994, 28 water treatment plant effluents flowing into the Nidda, a tributary of the Rhine, were sampled a total of 188 times. Vinclozolin was found in only one single sample at a concentration of 0.2 µg/l (Seel et al., 1996 in Gulden 1998).

Vinclozolin was not found at all in measurable concentrations in measurements in German inland waters, collected from the Federal States. The detection limits were between 0.01 and 0.5 µg/l. Studies on the occurrence of the antiandrogenically active vinclozolin degradation products, Ml and M2, which are more water soluble and stable than vinclozolin, have not been done.

Table 2 Occurrence in the environment of vinclozolin

Compartment	Year	Location	Concentration	Unit	Reference (source)
Water	1992	Lakes and rivers	0.3	µg/l	Gre96
Water	1988	Westland Netherlands	0.056 average 0.54 max	µg/l	Greve 1989a in Teunissen-Ordelman, 1995
Water	1989-90	Westland Netherlands	0.025 average 0.40 max	µg/l	Luttik 1991 in Teunissen-Ordelman, 1995
Water	1989-90	Westland Netherlands	0.1 average 0.49 max	µg/l	Lourens 1992 in Teunissen-Ordelman, 1995
Water	1989	Rijnland Netherlands	0.004 average 0.02 max	µg/l	HS Rijnland 1992 in Teunissen-Ordelman, 1995
Water	1987-88	't Langeveld Netherlands	0.6 average 3.8 max	µg/l	Greve 1989b in Teunissen-Ordelman, 1995
Water	1989-90	Hogeveense polder Netherlands	0.34 average 3.5 max	µg/l	Baumann 1991 in Teunissen-Ordelman, 1995
Water	1988	Haarlemmer-meerpolder Netherlands	0.017 average 0.1 max	µg/l	Greve 1989c in Teunissen-Ordelman, 1995
Water	1991	Flevoland Netherlands	0.15 average 0.2 max	µg/l	Van Boom en Keijzer 1993 in Teunissen-Ordelman, 1995
Water	1992	Delfland Netherlands	0.044 average	µg/l	Geenen en vd Geest 1995 in

Compartment	Year	Location	Concentration	Unit	Reference (source)
			0.32 max		Teunissen-Ordelman, 1995
Water	1993	Flevoland Netherlands	< dl	µg/l	Geenen en vd Geest 1995 in Teunissen-Ordelman, 1995
Water	1992	Drenthe Netherlands	0.55 average 0.55 max	µg/l	Geenen en vd Geest 1995 in Teunissen-Ordelman, 1995
Water	1992	Holl.Eilanden en Waarden Netherlands	< dl	µg/l	Geenen en vd Geest 1995 in Teunissen-Ordelman, 1995
Water	1993	Holl.Eilanden en Waarden Netherlands	< dl	µg/l	Geenen en vd Geest 1995 in Teunissen-Ordelman, 1995
Water	1991	Rhine Lobith Netherlands	< dl	µg/l	Forschungsvorhaben 1992 in Teunissen-Ordelman, 1995
Water	1990	Rhine Lobith Netherlands	< dl	µg/l	Forschungsvorhaben 1991 in Teunissen-Ordelman, 1995
Ground water	1992	Netherlands	< dl	µg/l	Janssen en Hopman 1993a in Teunissen-Ordelman, 1995
Ground water	1991	Netherlands	< dl	µg/l	Hpman en Janssen 1992 in Teunissen-Ordelman, 1995
Ground water	1992	Vierlingsbeek Netherlands	< dl	µg/l	Janssen 1993b in Teunissen-Ordelman, 1995
Ground water	1990	Noor-brabant Netherlands	< dl	µg/l	Lagas 1990b in Teunissen-Ordelman, 1995
Ground water	1990	Zuid-holland Netherlands	< dl	µg/l	Lagas 1990c in Teunissen-Ordelman, 1995
Rain water	1988-89	Boschpolder Netherlands	0.033 average 0.072 max.	µg/l	Slijkhuis 1990 in Teunissen-Ordelman, 1995
Rain water	1990-91	Fleverwaard Netherlands	0.2 average 0.6 max.	µg/l	Van Boom en Keijzer 1993 in Teunissen-Ordelman, 1995
Rain water	1990-91	Westland Netherlands	0.026 average 0.13 max.	µg/l	Prov. Zuid-Holland 1991 in Teunissen-Ordelman, 1995
Rain water	1991-92	Zuid-Holland Netherlands	0.044 average 0.26 max.	µg/l	Prov. Zuid-Holland 1994 in Teunissen-Ordelman, 1995

d.l= detection limit

Legal status

Vinclozolin is listed on the OSPAR candidate list and the Priority pesticides list under Directive 91/414/EEC (and specified under Council Regulation 3600/92).

Conclusion

Vinclozolin is a fungicide used on food crops. Human exposure may be expected by food because vinclozolin is inherently biodegradable. Vinclozolin is not bioaccumulative. Vinclozolin is measured in the environment (in water systems). This substance is prioritised as high concern.

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Zineb

The substance was selected to be evaluated in the expert meeting because it is a HPV chemical which is produced in more than 1000 tonnes/year.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 3 and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1 Physico chemical properties of zineb

Water solubility	10 mg/l (4 riwa, 1998)
Vapour pressure	-
Henry coefficient	0.0000027 Pa.m3/mole
Log Koc	3.1 (estimated based on water solubility; Kenaga 1980 in Ordelman, et al, 1993a)
Log Kow	1.3 (30 riwa, 1998)

Zineb is not very soluble in water.
Based on the log Kow no bioaccumulation is expected.
The half-life for zine in water is 0.2 to 16 days (gre96).
The DT50 in water is 0.25 to 17 days (4 riwa) and in soil 3 days (30 riwa, 1998).
Zineb is hydrolysed with a half life of 0.4 days at pH 5.7, 5 days at pH 7 and 18 days at pH 8. The half life in freshwater/sediment is 31-33 days (Ordelman, et al, 1993a).
Removal from sewage treatment plant is expected to be >90% by active coal (riwa, 1998). The limit value for zineb is 0.6 µg/l in the Netherlands (gre96).

Dithiocarbamates are generally instabile compounds. Important metabolites formed at all dithiocarbamates are carbondisulfide (CS2) and sulfurhydrogen (H2S) (Van Leeuwen, 1986 in Ordelman et al, 1993a). Furthermore a distinction can be made between the metabolites of the ethylenebisthiocarbamates (maneb and zineb) and the mono- and dialkyldithiocarbamates (metam natrium, thiram). From the ethylenebisthiocarbamates a joint 1,2,4-dithiazole (DIDT) is formed. DIDT is presumed to be the active compound. Other metabolites of the ethylenebisthiocarbamates are ethylenediisothiocyanate (EDI) and ethylenethiourea (ETU). ETU can be metabolised further. DIDT and ETU cannot be formed in the absence of oxygen (Vonk, 1975 in Ordelman, et al, 1993a). The metabolisation of DIDT to EDI does not occur in the presence of zinc- or ironsulfate (Vonk, 1975 in Ordelman, et al, 1993a) in water but does occur in the presence of copperoxide (hunter and evans 1991 Ordelman, et al, 1993a). The most important metabolite is ETU (13 riwa, 1998).
Expected is that the most dithiocarbamates will be metabolised quickly in the environment and in aquatic organisms and therefore will not spread to greater water systems and will not bioaccumulate or biomagnify. The BCF is measured at 270 and calculated to be 169 (Ordelman et al 1993a).

Use, Exposure and emissions
Zineb is produced as follows: ethylenediamine and sulfurcarbon are combined together with a 50% sodium hydroxide solution and decalcinated water (1 Ordelman et al 1993a). To the reaction product a zinc chloride solution is added after which zineb is formed (2 Ordelman, et al, 1993a). Zineb is produced in the Netherlands in Rotterdam in 640 tonnes/year in 1986 (Riza 1992 in Ordelman et al, 1993a). Before the waste water enters the sewage treatment plant it is first lead through an installation to regain the zinc. It was expected in 1993 that the industrial emissions in 1995 would be reduced with 90% compared to 1985 (Riza 1992 in Ordelman et al, 1993a). At formulation 60-150 tonnes and 300-1000 zineb are used in 1984 and 1986 resp. with an emission of <0.001 tonnes/year at 2 locations.
In 1985 190 tonnes of zineb was used in the Netherlands, 184 tonnes in 1988, 138 tonnes in 1991 and 125 tonnes in 1994 (Ordelman et al , 1993a). The emission is 1 tonne/year in 1994. It is allowed to be

used in the Netherlands (gre96). It is produced in more than 50 tonnes/year. It is produced in the Netherlands (Elf Atochem Agri bv), Germany (BASF), France, Spain and Intaly (RIWA, 1998).

Zineb is used as a protective leaf fungicide which inhibits the oxidative carboxylation of brenztraubensaure and the related inhibition of respiration in mitochondrions which inhibits the growth of the fungus (perkow and ploss 1996 in bruhn 1998). Zineb was allowed to be used in Germany until 12-97 but is not extended until feb. 1998 (bruhn 1998). Zineb is also used as a wood preservant (RIWA, 1998).
Zineb is registered as a general use pesticide by the US EPA. It is used as a fungicide and an insecticide to protect fruit and vegetable crops from a wide range of foliar and other diseases. In july 1987 the EPA announced the initiation of a special review of the ethylene bisdithiocarbamates (EBDCs), a class of chemicals to which zineb belongs. This special review was initiated because of concerns raised by laboratory tests on rats and mice. As part of the Special Review, EPA reviewed data from market basket surveys and concluded that actual levels of EBDC residues on produce purchased by consumers are too low to affect human health. Many home garden uses of EBDCs have been cancelled because the EPA has assumed that home users of tehse pestcides do not wear protective clothing during application (829 in DHC99). It is available in the US as wettable powder and dust formulations (gr99).
The most important use in the Netherlands is to protect field beans and tulip bulbs (Ordelman et al, 1993a).

Vulnerable use and vulnerable groups
Because zineb is used as a herbicide on food crops this could mean a certain risk. However zineb is metabolized quickly in the environment. The EPA (see above) concludes that actual levels of zineb are too low to affect human health. Maneb could also present a risk to agricultural workers applying the herbicide. Assumed is that these workers take the neccessary precautions using the substance. However the metabolite ETU could present a risk but this substance is not evaluated as an endocrine disruptor. This substance should be researched, to find out if it could have endocrine effects.

Environmental concentrations
Dithiocarbamates have not been measured in the different application areas in the Netherlands. From the available measurements follows that the different metabolites of dithiocarbamates have been found in surface water. ETU is found in Flevoland in the Netherlands in 46% of the measurements up till 0.9 µg/l. ETU is descended from maneb in this area. MITC is only found incidentally up to 0.13 µg/l. Dithiocarbamates (as CS2) have incidentally been found up to 7.5 µg/l. Dithiocarbamates are not measured in ground water in the Netherlands. Only ETU and MITC have been found. Especially in areas with bulb cultivation high levels of ETU occur in ground water of up to 42 µg/l. MTIC has been found up to 2.5. In Flevoland CS2 (226 µg/l max) and ETU (max 75 µg/l) have been found in rain water. CS2 is not found in marine water in the Netherlands. In Zeeland in the Netherlands in 55% of the measurements low concentrations CS2 have been found in sediment. Dithiocarbamates have not been measured in organisms (Ordelman, et al 1993a).
No measements of zineb in freshwater, marine water, rain water and groundwater have been done (Ordelman, 1996).

Legal status
Zineb is listed on the Priority pesticides list under Directive 91/414/EEC (and specified under Council Regulation 3600/92).

Conclusion
Zineb is used as a herbicide on food crops this could mean a certain concern for exposure. As zineb is metabolised quickly in the environment, actual levels of zineb are too low to affect human health.

However the metabolite ETU could present a risk but this substance is not evaluated as an endocrine disruptor. This substance should be researched, to find out if it could have endocrine effects.

On the basis of its metabolite ETU, zineb is considered as a substance of high concern.

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Amitrole

The substance was selected to be evaluated in the expert meeting because it is a HPV chemical which is produced in more than 1000 tonnes/year.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 3 and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1 Physico chemical properties of Amitrole

Water solubility	280 g/l at 25ºC (wor91 in EHC158) (21 RIWA, 1998)
Vapour pressure	55 nPa at 20ºC (wor91 in EHC158)
Henry coefficient	< 3E-12 (gre in DHC99)
Log Kow	-0.969 at 23ºC (OECD 107) (CFP in IUCLID96 (k1)) -1 (21 RIWA, 1998) 0.85 (1 RIWA, 1998) -0.15 (gre in DHC99)

Amitrole is readily soluble in water. Based on the log Kow no bioaccumulation is expected. The photodegradation half-life in distilled water is more than one year (Wor91 in EHC158). Photodegradation does occur in the presence of the photo-sensitizer humic acid potassium salt, reducing the half-life to 7.5 h (Jen87 in EHC158).

Amitrole has a low vapour pressure and therefore does not enter the atmosphere.
Amitrole is not volatile. Soil and water are the only compartments of concern regarding environmental fate. Direct spray on a non-covered soil (worst case scenario) may lead to soil contamination and leaching, run-off and drift can affect ground or surface waters. The DT50 in laboratory in standard German soil is 2.4- 46 days and in field in UK loamy soil DT50 is 15 to 21 days (CEFIC, 1999). Amitrole is moderately mobile in laboratory studies and rapidly degraded inot natural compounds in soil and no leaching to ground water is expected (CEFIC, 1999).

Amitrole is absorbed to soil particles and organic matter by proton association (Nea69/70 in EHC158). The binding is reversible and not strong, even in favourable acid conditions. Measured log Kow values classify amitrole as “highly mobile” in soils with a pH>5 and “medium to highly mobile” at lower pH (And89 in EHC158). The leaching through experimental soil columns differs but generally movement is most readily seen in sand (Day61, Zan81, Wel87 in EHC158). With increasing organic matter content the mobility is reduced. Degradation in soils is usually fairly rapid but varies with the soil type and temperature. Microbial degradation is probably the major route of amitrole breakdown. Abiotic mechanisms, including the action of free radicals, may also lead to degradation. In laboratory studies amitrole is degraded to CO2 with a half-life of 2-30 days (EHC158 in EHC158). In the field this may take longer and half-life in test clay was about 100 days (Wel87 in EHC158). The degradation products of amitrole are tightly bound to soil. Because amitrole is rapidly degraded to these products, the leaching potential will be low. Amitrole is resistant to hydrolysis and oxidising agents (Sit85 in EHC158).

After oral administration of amitrole it is readily absorbed from the gastrointestinal tract of mammals and rapidly excreted from the body (mainly as parent compound) mainly through urine. The majority of secretion takes place during the first 24 hours. Two minor metabolites are detected in urine because of metabolic transformation. When inhaled a similar rapid excretion through urine takes place (EHC158).

Bioaccumulation studies in flow through test systems with Lepomis macrochirus and Ictalurus punctatus showed that in fish exposed to 1 mg/l a slight bioaccumulation occurred after 21 days (1.7 - 3 times the concentration in water). When returned to untreated water the amitrole concentration in fish organs decreased rapidly (iwa78 in EHC158).
28 day BCF is 246 for Lepomis macrochirus at a concentration of 1 mg/l (CFP78 in iuclid96 (b1)).
With a high water solubility, a very low log Kow and non-persistence in animals means that there will be no bioaccumulation or biomagnification (through food chains) (EHC158).

The DT50 in soil is 0.7 days (21 RIWA, 1998).
The DT50 is 40 days (gre in DHC99).
Expected removal from sewage treatment plant is 20% (RIWA, 1998).

Use, Exposure and emissions

Amitrole is used as a defoliant, a herbicide, a reagent in photography and a plant growth regulator (gr99). Amitrole is used as a non-selective herbicide against deep-rooting weeds and in combination with other herbicide used as a total herbicide (perkow and ploss 1996 in bruhn 1998). In Germany it may not be used for aerial spraying from 1-9 to 30-4 in more than 4 kg/ha (bruhn 1998).

Amitrole does not occur naturally. Amitrole is used as a herbicide with a wide spectrum of activity and appears to act by inhibiting the formation of chlorophyll (Car75 in EHC158). It is primarily used as a herbicide and as a brush killer. It is also used as a non-selective pre-emergent herbicide on fallow land before planting kale, maize, oilseed rape, potatoes and wheat and in other non-crop situations. It is commonly used around orchard trees, on fallow land, along roadsides and railway lines, or for pond weed control. Approved uses of amitrole on soil are either for non-crop land prior to sowing, or for inter-row weed control in tree and vine crops, where contact with food plants is avoided. It is also used for the control of ponds weeds and is especially effective in the control of water hyacinth (Wor91 in EHC158). Amitrole has also been used as a cotton defoliant in some countries (Has69 in EHC158).
Amitrole is not to be used on food crops (EHC158).
Amitrole is manufactured by the condensation of formic acid with aminoguanidine bicarbonate in an inert solvent at 100-200 C (gr99).
Amitrole is produced in 1900 tonnes in Europe (CEFIC105 (p10)).
40-50 tonnes amitrole was used in 1991. The emission was 1.2 tonnes/year (gre96)
Amitrol is produced in Germany (Bayer), France (CFPI and elf atochem). Particles containing amitrole may be released from production plants. (RIWA).

Vulnerable use and vulnerable groups
Under normal circumstances of occupational exposure, it is unlikely that amitrole induces thyroid effects in humans (EHC158).
Amitrole does not present a significant risk to human health when manufactured and used according to good handling procedure. Current restrictions on its use in most countries, particularly its restriction to non-crop use, will ensure minimum human exposure (EHC158).
Annual monitoring of thyroid function is recommended for workers regularly involved with amitrole, both at the formulation or application stages. Also should epidemiological studies be continued on workers exposed to amitrole (EHC158).

Amitrole could present a risk to agricultural workers applying the herbicide. Assumed is that these workers take the neccessary precautions using the substance. Therefore is no indication that amitrole present a specific risk to vulnerable groups or creates high risk situations.

Environmental concentrations
Atmospheric levels of 0-100 mg/m3 have been measured close to one plant (Ala84 in EHC158). The use of amitrole in waterways and watersheds has led to transitory water concentrations of up to 150

µg/l. Concentration fall rapidly to non-detectable (< 2µg/l) levels in running water within two hours. Application to ponds gave an initial water concentration of 1.3 mg/l falling to 80 µg/l after 27 weeks. Close to a production plant, river concentrations ranged from 0.5 to 2 mg/l.
No residues of amitrole have been detected in food following recommended use. Spraying of ground cover around fruit trees did not lead to residues in apples. Wild growing fruit in the vicinity of control areas can develop residues.
There have been no reports of amitrole in drinking water.

Limits for residues in food and water should be maintained at low levels (<0.02 mg/kg in raw agricultural commodities of plant origin).

Amitrole has been measured in many compartments in the environment.

Table 2 Occurrence in the environment of amitrole

Compartment	Year	Time	Location	Concentration	Unit	Reference (source)
Water	-	Initial level at application	Pond water	1.34	Mg/kg	Grz66 in EHC158
Water	-	Level at 11 day after application	Pond water	1.03	Mg/kg	Grz66 in EHC158
Water	-	Level at 278 weeks after application	Pond water	0.08	Mg/kg	Grz66 in EHC158
Water	-	30 min after beginning of application	Watershed in Oregon	155	µg/l	Mar68 in EHC158
Water	-	End of application	Watershed in Oregon	26	µg/l	Mar68 in EHC158
Water	-	6 days after application	Watershed in Oregon	<2 (d.l)	µg/l	Mar68 in EHC158
Water	-	2h after treatment	Flowing water canals 7.2 km downstream	1	µg/l	Dem70 in EHC158
Water	-	-	River downstream discharge of aeration pond in vicinity of production plant	0.5 – 2	Mg/l	Ala84 in EHC158
Water	-	-	Aeration pond in vicinity of production plant	50 – 200	Mg/l	Ala84 in EHC158
Water	-	-	13 sampling points in France	<0.1 (d.l)	µg/l	Legra91 in EHC158
Water	1984	-	24 water samples in Japan	<4 (d.l)	µg/l	Jea87 in EHC158
Water	-	-	Meuse	<0.1	µg/l	22 RIWA, 1998
Sediment	1984	-	24 bottom sediments in Japan	<5-20 (d.l)	µg/kg	Jea87 in EHC158
Air	-	-	Production plant	0-100	Mg/m3	EHC 158
Soil	-	At maximum application rate to control terrestrial weeds	-	Up to 20	Mg a.i. /kg dry soil	EHC158
Food	-	-	A wide range of food crops	<0.05 (d.l)	Mg/kg	EHC 158
Food	-	-	Apples in Tasmania and New South	<0.01 (d.l)	Mg/kg	Moo68/69/70 in EHC158

Compartment	Year	Time	Location	Concentration	Unit	Reference (source)
			Wales			
Food	-	3 months after ground cover application	Apples in West Virginia	< 0.025 (d.l)	Mg/kg	Schu64 in EHC158
Food	-	3 months after direct application on fruit or foliage	Apples in West Virginia	> 0.025 (d.l)	Mg/kg	Schu64 in EHC158
Food			White and black grapes	0.01-1	Mg/kg	Iuclid96 (cf2)
Soil	-	13 days after spraying 3.5 kg a.i./ha	Near railway	0.67 en 2	Mg/kg	Dor88 in EHC158

d.l= detection limit

Legal status

Amitrol is listed in Annex 1D of the Third North Sea Conference, the OSPAR candidate list and the Priority pesticides list under Directive 91/414/EEC (and specified under Council Regulation 3600/92).

Conclusion

Amitrole gives no indication for high risk for certain groups or situations but because it is measured in the many compartments of the environment, the final indication is: medium risk.
Amitrole is a herbicide used alongside roads. Exposure is expected through direct exposure by contact of playing children with soil and plants alongside roads. It is not persistent and not bioaccumulative and is found in the environment primarily in water systems. It is prioritised as medium concern.

References

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Forschungsbericht 216 02 001/08.

CEFIC105: CEFIC material for expert meeting on endocrine disruptors

DHC99: Dutch Health Council (1999). Endocrine-disrupters in the Netherlands.

EHC158: WHO, 1994, Environmental Health Criteria, Amitrole, IPCS series.

Gre96: Greve, 1996 (Dutch Health Council). Hormoon-verstorende stoffen in Nederland. Gebruik, emissie, milieuconcentraties en fysisch/chemische karakteristieken.

RIWA, 1998 Xeno-oestrogenen en drinkwater(bronnen)

Nitrofen

The substance was selected to be evaluated in the expert meeting because it is a HPV chemical which is produced in more than 1000 tonnes/year.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 3 and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1 Physico chemical properties of nitrofen

Water solubility	<1 mg/l (keith in DHC99) 0.7-1.2 mg/l (Worthing 1987) 5 mg/l (hoe94 in iuclid96 (s2))
Vapour pressure	1.06 mPa at 40 C (Worthing 1987)
Log Kow	3.4 (1 riwa, 1998) 5.5 calc (hoe91 in iuclid96 (k2)) 4.61 exp. (Syr 1996)

Nitrofen is poorly soluble in water. Based on the log Kow bioaccumulation is expected.
When applied to soil, it will photolyze in surface layers (65% degradation in 1 week) and biodegrade (99% degradation in 50 days). Bioconcentration in fish and aquatic organisms will be appreciable. In the atmosphere, nitrofen would exist primarily absorbed to particulate matter and in aerosols from spraying operations. It will be subject to gravitational settling and rapidly photolyse (828 in DHC99).
The removal from sewage treatment plant is expected to be >90% with active coal (riwa, 1998).
33 day BCF in fish (*Gambusia affinis*) is 1546, in midge (*Culex quinquefasciatus*) 3188, in mollusc (Physa spec.) 2770 and in algae (*Oedogonium cardiacum*) 405 (oya88 in iuclid96 (b2)).

Use, Exposure and emissions
Nitrofen is produced in Germany (Chemie AG Bitterfeld-Wolfen). It is not allowed to be used in the Netherlands (RIWA, 1998). From 10-11-1992 nitrofen is forbidden to be used in Germany (bruhn 1998).
Nitrofen is on Annex 1 and 2 of the EU council regulation 2455/92 which prohibits all plant protection products containing nitrofen as an active ingredient, to be used or placed on the market. Nitrofen is also in EC directives 76/769/EC and 79/117/EC (ISPRA, 2000).

Nitrofen is used as a herbicide in vegetable cultures (perkow and ploss 1996 in bruhn 1998).
Nitrofen is a contact herbicide for pre and post emergence control of annual grasses and broadleafed weeds on a variety of food and ornamental crops and on rights-of-way, but it has not been used around homes and gardens (828 in DHC99). Nitrofen is used as a herbicide on many vegetables, a number of broad-leaved and grass weeds, cereals, rice, sugar beet, some ornamentals, broccoli, cauliflower, cabbage, brussel sprouts, onions, garlic and celery. It is also used in nurseries for roses and chrysanthemums.
Nitrofen may be released to the environment during its production and use as a pre- and post-emergence herbicide. However nitrofen is no longer manufactured or sold in the US and Canada because of possible mutagenic and carcinogenic effects, although it has been used in other countries (828 in DHC99).
Exposure to nitrofen would be primarily occupational via dermal contact, with agricultural workers who formulate and apply the herbicide or handle treated soil and crops being especially at risk (828 in DHC99).

Vulnerable use and vulnerable groups
Because nitrofen is used as a herbicide on food crops this could mean a certain risk. Nitrofen could also present a risk to agricultural workers applying the herbicide. Assumed is that these workers take the neccessary precautions using the substance.

Environmental concentrations

There are no measurements of nitrofen in the environment.

Legal status

Nitrofen is listed on the HELCOM Priority list, and Directive 76/769/EEC including banned substances (shortlist derived from DG ENV);

Conclusion

Nitrofen is a herbicide used on food crops. Human exposure may be expected by food but because this substance is restricted in the EU, exposure is less likely. Nitrofen is inherently biodegradable and bioaccumulative and not measured in the environment (in water systems). This substance is prioritised as medium concern.

References

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RIWA, Xeno-oestrogenen en drinkwater(bronnen).

4-tert Octylphenol and nonylphenol

These substances were selected for evaluation in the expert meeting because they are HPV chemicals, which are produced in more than 1000 tonnes/year.

Expert evaluation

On both substances the Wildlife relevant endocrine disruption data are evaluated as category 1 and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1 Physico chemical properties of 4-tert octylphenol and nonylphenol

	Octylphenol	Nonylphenol
Water solubility	12.6 mg/l at 20 C (ahe93 in iuclid96 (s7)) insoluble (gre in DHC99)	Not significant (5 mg/l for technical nonylphenol) (gre96; fra97) Poorly soluble (riwa) 11 mg/l at 20 C (OECD 105) hue88 in iuclid96 (s8))
Vapour pressure	0.21 Pa at 20 C estimated (hue89 in iuclid96 (v8))	10 Pa (KEMI in fra97) <1 Pa at 20 C (hue94 in iuclid96 (v9)).
Log Kow	4.18 (ahel, 1993 in fra97) 3.7 measured (mclee81 in iuclid96 (k6)) 4.2 (OECD107) (mclee81 in iuclid96 (k7))	4.48 (NP1-3 approx. 4.2) 2.9-4.2 (3.3 for technical nonylphenol) (gre96;fra97) 3.4 (58 riwa, 1998) 4.2-6.4 (shiu 1994 in sepa98) 3.28 (OECD107) hue89 in iuclid96 (k8))
Henry's law coefficient		435 Pa.m3/mole (fra97)

Nonyl and octylphenol are mainly derived from alkylphenolethoxylates (APEOs). The solubility in water of APEO decreases with decreasing number of ethoxylates. The result is that AP and lower oligomers tend to move to a lipophylic environment. Like sediment and biota. With a decrease in alkyl chain lengths, the log Kow decreases (shiu 1994 in sepa98). The compound is expected to accumulate in aquatic organisms based on estimated BCF-value of 6000 (52, 53, 54, 55, 56 in fraunhofer, 1999).

It is estimated that approx. 50% of all nonylphenolethoxylates in untreated wastewater will be adsorbed to the sludge in a wastewater treatment plant. Removal from sewage treatment plant expected to be >90% by active coal (riwa, 1998).

If released into the atmosphere octylphenol and nonylphenol are degraded by reaction with photochemically produced hydroxyl radicals with an estimated half-life of about 9 hours. An estimated Koc-value of 18.000 suggests that the substances will be immobile in soil and sediment. Rapid infiltration studies indicate, however, that the substances have some mobility in sandy or gravy soils and are able to reach groundwater although in lower concentrations as the original effluent. Biodegradation may be an important fate process in the water column (52, 53, 54, 55, 56 in Fraunhofer, 1999). Octylphenol is inherently biodegradable (Bla95/nay92 in cefic13 (cw1)).

In the sewer and in the sludge treatment plant water soluble oligomers (EO>8) are degraded to insoluble AP or AP1EO or AP2EO, which adsorb to the sludge. In anaerobic sludge further degradation of NP1EO and NP2EO to NP is observed (Giger, 1984 in Belfroid et al., 2000).

In the entire wastewater treatment process Ahel et al. (1994) estimate that, relative to the total content of 4-nonylphenol compounds,
- 40% is completely degraded,
- 20% is absorbed by the sludge, and

- 40 to 45% is not retained and enters the effluent.

The total 4-nonylphenol compounds absorbed by the sludge consisted of 95% 4-nonylphenol and 5% NP1EO and NP2EO.

Degradation in sewage treatment plants is temperature-dependent. Whereas 89% of APEOs were degraded in a sewage treatment plant in the summer (corresponding to 0.21 mg/l in the effluent), only 68% were degraded in the winter (0.49 mg/l in the effluent). It is thought that biodegradation occurs to a decisive extent only at temperatures above 15°C (CES 1993 in Oko-Institut, 1997).

In soil 4-nonylphenol is biologically mineralised under aerobic conditions. More than 80% is degraded in the first month, degradation then proceeds more slowly with 10% of the initial amount still being detectable after one year (Warhurst 1995). In comparison, Küchier (1996) found that soil concentrations of 0.6 mg/kg 4-nonylphenol were biodegraded within 4 weeks to below the limit of detection. However, it is likely that degradation in the soil is not always complete (Oko-Institut, 1997). By contrast, it is not degraded under anaerobic conditions (for example, in digested sludge), but rather increases in concentration due to the anaerobic degradation of other 4-nonylphenol compounds.

In rats nonoxynol-9 (NP with 9 ethoxy groups) is metabolised to Nonylphenol (NP) and subjected to glucuronidation. Radiolabelled nonoxynol-9 has been shown to be cleared from rat liver and kidneys within 48 h (nimrod and benzon 1996 in sepa98). Degradation and metabolism studies are mainly performed with straight chain alkyl whereas the technical products consist of mainly branched alkyl chains.

4-Nonylphenol is eliminated relatively rapidly from the human body. Accumulation in particular tissues or organs has not been observed in humans. This diminishes the likelihood of chronic toxic effects (Oko-Institut, 1997).

Use, Exposure and emissions

Octyl- and nonylphenol are widely used for raw materials of detergents, emulsifiers, wetting and dispersion agents in e.g. paints, anti-oxidants of plastic, pesticide (fungicide), and its supplemental agent (foaming agent) and stabilizer of polyvinyl chloride. Alkylphenols are also used as additives in lubricating oil, as spermicides in contraceptice foams (platt 1978 in sepa98).

Polyoxy ethylene alkylphenol, which is synthesized by the reaction of alkylphenol and ethylene oxide, is a non-ionic surfactant. The surfactant products on the market are complicated mixtures of isomers of alkyl groups and various oxyethylenes with different oxyethylene numbers. Eighty percent of Alkylphenol used on the market is predominantly nonylphenol over the world and octylphenol accounts for another 20%. The numbers of oxyethylene radicals are between 1 and 50 depending on their use.

The amounts of Polyoxyethylene nonylphenol used in England are annually 13,500 t. However, the household use was prohibited in 1976. In Germany, Nonylphenol was used in an amount of 13,500 t in 1986 in addition to the use for Polyoxyethylene-nonylphenol. Namely, as the raw material of tris-nonylphenylphosphite for anti-oxidation agent for plastic 1,200 t is used and its Ba or Ca salt for stabilizer of vinyl chloride amounts to 250 t. In Japan total amounts of production were 50,501 t in 1992, 41,218 t in 1993, 46,961 t in 1944 and 53,953 t in 1995 respectively. (Japan, 1997).

Nonyl- and octylphenol are stable biodegradation products of alkylphenolpolyethoxylates (APEOs). It is produced 10000-50000 tonnes (iuclid96 (p2)). The approximate EU production is 6800 tonnes (cefic13 (p1)). Other information reports a production of 693 to 1003 tonnes/year for the total alkylphenols (Westra, 1995 in fra97). Octylphenol is produced in more than 50 tonnes/year (RIWA, 1998). In 1995 in Germany nonylphenol is produced in 23,100 tonnes and emitted in >200 tonnes (Ecorapport DI in DHC99).

It is produced in the Netherlands (Dr W Kolk Nederland BV), Germany (Akzo, Henkel, Rewo Chemische Werke), France, Italy, Norway, Spain, the UK and Sweden (RIWA, 1998).

Nonylphenol was shown to be released from polystyrene and PVC products (soto 1991 and Junk, 1974 in sepa98 and Temanord, 1996).

Vulnerable use and vulnerable groups
The main routes of exposure to NP are direct exposure due to release from polystyrene and PVC products and through the consumption of contaminated food. For octylphenol no information is available on its release from plastics.

There is no information to in what extend nonylphenol is released from polystyrene or PVC. However, polystyrene bottles are used for baby feeding. Indicating that there might be a concern for exposure of a vulnerable group. Nonylphenol (and probably also octylphenol) does not accumulate in humans. Combined with the possible exposure this indicates a medium concern.

In the environment NP and OP are mainly found in sediments. Vulnerable groups are mainly sediment living organisms and predators.

Environmental concentrations
In the Fraunhofer report (1999) octylphenol is measured in sediment with a median concentration of 1.30 µg/l (mean 1.31 µg/l) based on 15 data from 15 stations (all these data were above the determination limit). In RIZA/RIKZ (in DHC99) octylphenol has not been found in environmental compartments in the Netherlands.

NP and NPEO with 1-3 ethoxy groups are detected in sewage systems effluents and NP has been determined in sediment near sources. Short chain APs are also released from the deposits of oil-shale from Kothla-Jrave in Estonia (pers.comm Ake Bergman in sepa98). In Sweden NP has been detected in recipients close to sources such as downstream from a sewage treatment plant in Gota Alv (160 ppb in water) and at a concentration of 2 ppb in sea water in an industrially affected area at the Swedish west coast (Malmqvist and Duus, temanord1996 in sepa98). So far reported data on environmental levels are from industrially affected areas (sepa98).
In Switzerland the use of 4-nonylphenol in textile cleaning agents was prohibited in 1986. This prohibition led to a decrease of the 4-nonylphenol content from a high of 1.3 g/kg dry weight in 1982 to 0.2 g/kg dry weight in 1995 in sewage sludges from the Kanton Zürich (Giger 1995).

In the mussel, *Mytilus edulis*, NP (4-nonylphenol) concentrations in the range 0.2 to 0.4 mg/kg w.w. have been measured. In freshwater macrophytic algae NP, NP1EO and NP2EO in the concentration ranges 2.5 to 38.0 mg/kg d.w., 0.9 to 4.7 mg kg-' d.w. and 0.6 to 4.3 mg/kg d.w., respectively, have been found. The concentrations in Fresh-water fish of NP, NP1EO and NP2EO have been determined at significantly lower concentrations in fresh-water fish than in fresh-water algae. NP, NP1EO and NP2EO have been detected in the concentration ranges <0.03 to 1.6 mg/kg d.w., 0.06 to 7.0 mg/kg d.w. and <0.03 to 3.0 mg/kg d.w. Concentrations of NP were in general higher in liver (0.98 to 1.4 mg/kg d.w.) ~ in muscle tissue (0.15 to 0.38 mg/kg d.w.).

NP, NP1EO and NP2EO have also been detected in wild fowl, i.e. the duck, *Anas boscas*. highest concentrations were observed in muscle tissue, where NP, NP1EO and NP2EO were measured at concentrations of 1.2, 2.1 and 0.35 mg/kg d.w., respectively.

Table 2 Occurrence in the environment of 4-tert octylphenol and nonylphenol

Chemical	Compartment	Year	Location	Concentration	Unit	Reference (source)
OP	Water	<1978 Winter	Delaware river USA	1-2	µg/l	Shel78 in iuclid96 (cw1)
OP	Water	<1978 Summer	Delaware river USA	0.2-2	µg/l	Shel78 in iuclid96 (cw1)
OP	Water	<1992	Surface water	0.6	µg/l	Bla95/nay92 in cefic13 (cw1)
OP	Sediment	<1997		2.2 90 Perc. 17/17	µg/kg	Fraunhofer, 1999
NP	Water	<1992	Lakes and rivers in UK	0.2-12	µg/l	Gre96; RIZA nota in fra97
NP	Water	<1996	Westerschelde	1-10	µg/l	Leonards 1996 in Fra97
NP	Water	<1996	Maas	10	µg/l	Leonards 1996 in Fra97
NP	Water	<1995	UK river mouth	<0.1	µg/l	Blackburn, 1995 in fra97
NP	Water		Meuse	0.1-<1	µg/l	22 riwa, 1998
NP	Water		Germany surface water	0.04-1000	µg/l	122 riwa, 1998
NP	Water		Germany industrial effluent	4000	µg/l	122 riwa, 1998
NP	Water		USA rivers	0.2	µg/l	Nay92 in rikz96 (CW7)
NP	Water		UK/Aire/river	180	µg/l	Bla95 in rikz96 (cw8)
NP	Water		Switzerland/Glatt/ river	45	µg/l	Ahe93 in rikz96 (cw9)
NP	Water		Netherlands/Meuse/r iver	10	µg/l	Rikz96 (cw10)
NP	Water			< dl-0.14	µg/l	RIZA/RIKZ Loes in DHC99
NP	Sediment		Netherlands	1	µg/l	Rikz95 in rikz96 (cse1)
NP	Sediment			240-870	µg/kg ww	RIZA/RIKZ Loes in DHC99
NP	Suspended matter			210-620	µg/kg ww	RIZA/RIKZ Loes in DHC99
NP	Wildlife biota			Not found		RIZA/RIKZ Loes in DHC99
NP	Wildlife biota	1991	Seabird feathers	9.7	µg/kg	Vethaak, 1996 in fra97
NP	Wildlife biota		Musca spec.	1	mg/kg	Wah90/mar90 in rikz96 (cwl2)
NP	Wildlife biota		Macroalgae	1	mg/kg	Wah90/mar90 in rikz96 (cwl3)
NP	Wildlife biota		Freshwater fish	2	mg/kg	ahe93 in rikz96 (cwl1)

d.l= detection limit

Legal status

No data available on the legal status of nonylphenol. 4-tert-Octylphenol is listed in Annex 1D of the Third North Sea Conference and the OSPAR candidate list.

Conclusion

There are some indications that nonylphenol is released from polystyrene and PVC. If this is the case, then there is a concern for vulnerable groups like bottle feeding babies. There is no information to in what extend nonylphenol is released from polystyrene or PVC. However, polystyrene bottles are used for baby feeding. Indicating that there might be a concern for exposure of a vulnerable group.

Nonylphenol (and probably also octylphenol) does not accumulate in humans. Combined with the possible exposure this indicates a medium concern.

Nonylphenol and in a lesser extend octylphenol (branched) are released in the environment through the degradation of alkylphenolethoxylates (APEOs) in the sewer and sludge treatment plants. APEOs are mainly adsorbed to sediment and suspended solids.

Nonyl- and octyl are inherently biodegradable and are measured in water sediment, suspended solids and wildlife, indicating a medium concern for exposure.

Fraunhofer has proposed to include the group "octylphenols - nonylphenols", covering all isomers of the C8 and C9 alkyl chains – in a priority list (Fraunhofer, 1999).

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4-Nitrotoluene

The substance was selected to be evaluated in the expert meeting because it is a HPV chemical which is produced in more than 1000 tonnes/year.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 3 and the Human health relevant endocrine disruption data as category 1.

Chemical characteristics

Table 1 Physico chemical properties of 4-nitrotoluene

Water solubility	Poorly soluble (26 riwa, 1998) 345 mg/l at 20 C (bay—in iuclid96 (s12))
Vapour pressure	0.13 hPa at 20C (aue85 in iuclid96 (v13))
Log Kow	2.37 (1 riwa, 1998)

4-Nitrotoluene is poorly soluble in water. Based on the log Kow 4-nitrotoluene is not expected to bioaccumulate. 4-Nitrotoluene has been shown to be inherently biodegradable (Bayer report 1973 in CEFIC 1999) in a closed bottle test and a Zahn-Weller test (Hoecst report 1982 in CEFIC, 1999).
The half life of photochemical degradation in water is 5.9 hours (sim86 in cefic360 (cw6)).
The 42 day BCF is 3.7-7.2 for Cyprinus carpioat 0.1 mg/l. At 0.01 mg/l the BCF is 4.5 to 8 (OECD305C)(citi92 in iuclid96 (b15)).

Use, Exposure and emissions
Nitrotoluene is produced in the EU approx 20000 tonnes (cefic360 (p8)). Nitrotoluene is used as intermediate for chemical synthesis (closed system) According to CEFIC information there is no consumer exposure and release to the aquatic environment is low (cefic360 (u8)).
Nitrotoluene is used in the varnish industry. It is produced in more than 50 tonnes/year (RIWA, 1998).
p-Nitrotoluene is an middle product for pigments, synthetics, pharmaceuticals, fragrants, explosives (TNT) and poly-urethane preproducts (Falbe and regitz 1991 in bruhn 1998).
Removal from sewage treatment plant by ozon (13-20 mg/l), with a high half-life of 100-400 min dependable on pH (low at high pH). Removal from sewage treatment plant is expected to be >90% by active coal (riwa, 1998).

Vulnerable use and vulnerable groups

Based on the available information nitrotoluene is not expected to present a risk to vulnerable groups or create high risk situations. Conclusion: no indication for high risk group or situation.

Environmental concentrations

4-nitrotoluene is not measured in the environment. However o-nitrotoluene is found in surface water.

Table 2 Occurrence in the environment of 4-nitrotoluene

Compartment	Year	Substance	Location	Concentration	Unit	Reference (source)
Water		o-nitrotoluene	Surface water	<0.1-1	µg/l	22 riwa, 1998

d.l= detection limit

Legal status

Nitrotoluene is referred to in Council directive 67/548/EEC concerning the classification, packaging and labelling of dangerous substances and council regulation EEC/793/93 on the evaluation and control of risks of existing substances.

Conclusion

Nitrotoluene is used as an intermediate in the varnish industry, pharmaceuticals and fragrants. It is used in closed system, which indicates no exposure. Although it is inherently biodegradable it is not bioaccumulated. Nitrotoluene is prioritised as low concern.

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Tetrabutyltin

The substance was selected to be evaluated in the expert meeting because it is a HPV chemical which is produced in more than 1000 tonnes/year and a metal.

Expert evaluation

The Wildlife relevant endocrine disruption data are evaluated as category 1 and the Human health relevant endocrine disruption data as category 2.

Chemical characteristics

Table 1 Physico chemical properties of tetrabutyltin

Physical state	Colourless liquid
Water solubility	8.8 at 20 C (OECD 105)(wit88 in iuclid96 (s6))
Vapour pressure	0.14 Pa at 25 C (OECD 104)(wit89 in iuclid96 (v7))
Log Kow	

tetrabutyltin is poorly soluble in water.

Use, Exposure and emissions
Tetrabutyltin is used as intermediate for the production of mono-, di, and tributyltin. Production volumes of tetrabutyltin have been estimated at 400 tonnes a year in the Netherlands in 1989 (Evers et al., 1996).

Vulnerable use and vulnerable groups
As tetrabutyltin mainly is used as intermediate for the production of other organotin compounds, human exposure will be primarily occupational. Tetrabutyltin might be a contaminant in other organotin compounds, probably resulting in a lower consumer exposure than tributyltin.

Environmental exposure to tetrabutyltin might be caused by industrial spills or by contaminants in other organotin products.

Environmental concentrations
No data available

Legal status
No information on legal status

Conclusion
Due to the restricted use of tetrabutyltin there is a relatively low concern for exposure to humans and organisms. It should be checked if there are indeed only limited applications of tetrabutyltin.

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