Hazardous Substances Series

Phthalates

OSPAR Commission
2005 (2006 Update)

The Convention for the Protection of the Marine Environment of the North-East Atlantic (the "OSPAR Convention") was opened for signature at the Ministerial Meeting of the former Oslo and Paris Commissions in Paris on 22 September 1992. The Convention entered into force on 25 March 1998. It has been ratified by Belgium, Denmark, Finland, France, Germany, Iceland, Ireland, Luxembourg, Netherlands, Norway, Portugal, Sweden, Switzerland and the United Kingdom and approved by the European Community and Spain.

La Convention pour la protection du milieu marin de l'Atlantique du Nord-Est, dite Convention OSPAR, a été ouverte à la signature à la réunion ministérielle des anciennes Commissions d'Oslo et de Paris, à Paris le 22 septembre 1992. La Convention est entrée en vigueur le 25 mars 1998. La Convention a été ratifiée par l'Allemagne, la Belgique, le Danemark, la Finlande, la France, l'Irlande, l'Islande, le Luxembourg, la Norvège, les Pays-Bas, le Portugal, le Royaume-Uni de Grande Bretagne et d'Irlande du Nord, la Suède et la Suisse et approuvée par la Communauté européenne et l'Espagne.

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Executive Summary/*Récapitulatif*

Phthalates are a family of industrial chemicals used as softeners, adhesives or solvents by a variety of industries. They are mainly used in the polymer industry as plasticizer in PVC and to a lesser extent in the non-polymer industry for different consumer products (sealants, paints, printing inks, cosmetics, coatings of different products such as cars, coils, cables or fabrics etc.). This document examines five selected compounds of phthalates: di-n-butyl (DBP), butylbenzyl (BBP), di(2-ethylhexyl (DEHP), di(isonyl) (DINP) and di(isodecyl) (DIDP) phthalate. DBP and DEHP were added to the OSPAR List of Chemicals for Priority Action in 1998 as part of the group "certain phthalates" which also covers BBP, DINP and DIDP.

Les phtalates forment une famille de produits chimiques industriels, utilisés comme adoucisseurs, adhésifs ou solvants dans toute une série d'industries. Ils sont surtout utilisés dans l'industrie des polymères comme plastifiants du PVC et, dans une moindre mesure, en dehors de l'industrie des polymères, pour différents produits de grande consommation (produits d'étanchéité, peintures, encres d'imprimerie, produits cosmétiques, revêtements de divers produits tels qu'automobiles, bobines, câbles ou tissus, etc.). Le présent document aborde cinq composés sélectionnés des phtalates : le di-n-butyl (DBP), le butylbenzyl (BBP), le di(2-éthylhexyl (DEHP), le di(isonyl) (DINP) et le di(isodecyl) (DIDP) phtalate. Le DBP et le DEHP ont été inscrits en 1998 sur la Liste OSPAR des produits chimiques devant faire l'objet de mesures prioritaires, à titre de partie intégrante du groupe dit "certains phtalates" qui englobe aussi le BBP, le DINP et le DIDP.

DBP is produced at three production sites in the EU at a level of 26 000 t in 1998 of which 18 000 t were consumed in the EU. Emissions from diffuse sources include, for example, cleaning of road tankers used for transport of DBP in the EU, end use of plasticized PVC or use of adhesives. DBP has been found in waste water effluents, rivers and estuarine and offshore waters as well as marine sediments. DBP is not a PBT substance according to the OSPAR DYNAMEC or EU-TGD PBT criteria. There is a potential risk for ecotoxic effects on aquatic species in the marine environment at a local scale. At regional level, risks to the marine environment are expected to be negligible. DBP has, however, potential for endocrine disrupting effects. The risk of these effects in marine mammals is probably low since DBP is readily degradable.

Le DBP est fabriqué dans trois sites de production implantés dans l'Union européenne, à raison de 26 000 t en 1998, dont 18 000 t ont été consommées dans l'Union européenne. Les émissions de sources diffuses comprennent, par exemple, le nettoyage des citernes des camions transportant du DBP dans l'Union européenne, l'utilisation du PVC plastifié sous forme de produit final ou l'utilisation d'adhésifs. La présence de DBP a été observée dans les effluents des eaux usées, dans des cours d'eau et dans des eaux estuariennes et du large ainsi que dans des sédiments marins. Selon les critères fixés par le mécanisme DYNAMEC d'OSPAR ou les critères de PBT du Document d'orientation technique de l'Union européenne, le DBP n'est pas une substance PBT. Il existe à l'échelon local un risque potentiel d'effets écotoxiques pour les espèces aquatiques en milieu marin. Au niveau régional, les risques pour le milieu marin devraient être négligeables. Cependant, le DBP possède un potentiel de perturbation du système endocrinien. Le risque de ce type de perturbation chez les mammifères marins est probablement faible car le DBP est directement dégradable.

BBP is produced in the EU, in quantities of 45 000 t/year in 1994-1997. Approximately 36 000 t/year are consumed in the EU. BBP is emitted to waste water, directly discharged to the aquatic environment or reaches the sea through riverine inputs. Among different sources, concentrations of BBP in waste water samples from kindergardens (washing and abrasion of PVC floors) and car wash facilities were highest. BBP does not meet the OSPAR DYNAMEC or EU-TGD PBT criteria but there might be a low risk of ecotoxic effects in the marine environment for organisms in the water column. There might also be a risk of potential for endocrine disrupting effects. In the EU, the risk assessment under Regulation 793/93 is still ongoing.

De 1994 à 1997, du BBP était fabriqué dans l'Union européenne à raison de 45 000 t/an. Environ 36 000 tonnes sont consommées tous les ans dans l'Union européenne. Du BBP est émis dans les eaux usées, directement déchargé dans le milieu aquatique ou atteint la mer par le biais des apports fluviaux. Parmi les diverses sources, les teneurs en BBP dans les échantillons d'eaux usées des écoles maternelles (lavage et abrasion des sols en PVC), ainsi que dans les appareils de lavage des voitures sont les plus fortes. Bien que le BBP ne réponde pas aux critères PBT du mécanisme DYNAMEC d'OSPAR ni à aux critères PBT du Document d'orientation technique de l'Union européenne, il existe un faible risque d'effets écotoxiques dans le milieu marin, ceci pour les organismes vivant dans la colonne d'eau. Il se peut qu'il y ait aussi un risque de perturbation du système endocrinien. L'évaluation des risques faite en vertu du Règlement 793/93 se poursuit dans le cadre de l'Union européenne.

The consumption of DEHP in the EU was 476 000 t/year in 1997. Most products in the internal market containing DEHP are produced in the EU. Due to the large quantities used annually and the patterns of use

in many articles with long service life, large amounts of DEHP are diffusely spread in the environment and is therefore found in all environmental compartments, including remote areas. DEHP is not considered a PBT substance (according to the OSPAR DYNAMEC or EU-TGD PBT criteria) although it is a borderline case. It is not considered persistent, but has a potential for bioaccumulation which does not meet the EU-TGD B-criterion but exceeds the OSPAR DYNAMEC criterion for bioaccumulation. It has also potential of reprotoxicity for mammalian species. At given environmental concentrations, there is no apparent risk for marine organisms, in particular in open marine waters. However, there might be potential endocrine disrupting effects. DEHP is listed as priority substances under the Water Framework Directive (Annex X) and is subject to a review for identification as a possible "priority hazardous substance". Risk assessment under Regulation 793/93 is still ongoing.

Dans l'Union européenne, la consommation de DEHP a atteint 476 000 t/an en 1997. La plupart des produits contenant du DEHP et se trouvant sur le marché intérieur sont fabriqués dans l'Union européenne. Du fait des grandes quantités consommées tous les ans, ainsi que des profils d'utilisation de nombreux articles à longue durée utile, de grandes quantités de DEHP sont diffusées dans l'environnement, et l'on en trouve donc dans tous les compartiments de l'environnement, y compris dans des zones lointaines. Le DEHP n'est pas considéré comme une substance PBT (suivant les critères de PBT du mécanisme DYNAMEC d'OSPAR ou du Document d'orientation technique (DOT) de l'Union européenne), en dépit du fait qu'il s'agisse d'un cas limite. Il n'est pas considéré comme persistant mais possède un potentiel de bioaccumulation qui ne répond pas au critère B du DOT de l'Union européenne, mais dépasse en revanche le critère de bioaccumulation du mécanisme DYNAMEC d'OSPAR. Il présente également un potentiel de reprotoxicité pour les espèces mammaliennes. A des teneurs déterminées dans l'environnement il n'y a pas de risque apparent pour les organismes marins, notamment dans les eaux marines de haute mer. Il se peut toutefois qu'il ait des effets potentiels de perturbation du système endocrinien. Le DEHP figure sur la liste des substances prioritaires de la Directive cadre relative à l'eau (Annexe X) et fait l'objet d'une étude en vue d'un classement éventuel parmi les « substances dangereuses prioritaires ». L'évaluation des risques, effectuée en vertu du Règlement 793/93, se poursuit.

DINP and DIDP are produced in the EU where their total consumption was 107 000 t/year for DINP and 199 480 t/year for DIDP in 1994. DINP and DIDP are not PBT substances according to OSPAR DYNAMEC or EU-TGD criteria and there is no indication of potential for endocrine disruption. Both substances have a potential for food chain transfer in marine mammals but their current use patterns and concentrations in the environment suggest that no long-term effects and no endocrine disrupting effect in the aquatic environment are expected. Neither of the substances qualifies as chemicals for priority action.

Du DINP et du DIDP sont fabriqués dans l'Union européenne, où leur consommation totale a atteint en 1994 107 000 t/an dans le cas du DINP et 199 480 t/an dans celui du DIDP. Le DINP et le DIDP ne sont pas des substances PBT selon les critères DYNAMEC d'OSPAR ni du DOT de l'Union européenne, et rien n'indique un potentiel de perturbation endocrinienne. Ces deux substances peuvent être transférées chez les mammifères marins par la chaîne alimentaire, mais leurs profils actuels d'utilisation et leurs teneurs dans l'environnement donnent à penser qu'il ne devrait se produire aucun effet à long terme ni de phénomène de perturbation endocrinienne dans le milieu aquatique. Ni l'une ni l'autre de ces substances ne présente de propriétés qui conduiraient à les classer parmi les produits chimiques devant faire l'objet de mesures prioritaires.

The actions recommended for DBP, BBP and DEHP in this Background Document are: to address their potential of endocrine disruption as part of a general approach to endocrine disruptors and to follow-up the work carried out to this end in the OECD and EU; Contracting Parties to support ongoing risk assessments in the EU; to assess the need for further reductions from the various sources and the practicability of such reduction and, in that light, to review regulations and control measures; to urge the relevant forums (e.g. OECD and EU) to start preparing an overview of possible reduction measures, including the identification of alternatives to the use of DEHP; to observe consumption rates, in particular of DEHP; to re-evaluate the risks posed by phthalates when further information becomes available and to review the need for measures; to communicate this Background Document to the European Commission and to other appropriate international organizations which deal with hazardous substances, namely the OECD.

Les actions recommandées dans le présent document de fond dans le cas du DBP, du BBP et du DEHP sont les suivantes : étudier leur potentiel de perturbation endocrinienne à titre de partie intégrante de la stratégie générale visant les perturbateurs endocriniens, ainsi qu'à titre de suivi des travaux effectués à cette fin à l'OCDE et dans l'Union européenne ; les Parties contractantes apporteraient leur soutien aux évaluations des risques en cours dans l'Union européenne ; apprécier la nécessité de nouvelles réductions des apports des diverses sources ainsi que la faisabilité de ces réductions, et, à la lumière des conclusions, revoir les règlements et les mesures de lutte ; presser les instances pertinentes (p.ex. OCDE et Union européenne) de démarrer l'élaboration d'une synthèse des mesures de réduction possibles, dont la

détermination des alternatives à l'utilisation du DEHP ; observer les taux de consommation, notamment du DEHP ; réévaluer les risques que présentent les phtalates lorsque de nouveaux éléments d'information sortiront, et reconsidérer si des mesures s'imposent ; communiquer le présent document de fond à la Commission européenne et aux autres organisations internationales compétentes traitant des substances dangereuses, à savoir l'OCDE.

A monitoring strategy for phthalates is annexed to this background document.

Une stratégie de surveillance sur les phtalates est annexée à ce document de fond.

1. Review

The risk of ecotoxic effects in the marine environment has been reviewed for 5 selected phthalate esters:

Di-n-butyl phthalate: A $PNEC_{aquatic(marine)}$ of 1 µg/l is proposed. Measured concentrations in marine waters are in the range 0,007-4,8 µg/l a potential risk of ecotoxic effects in the marine environment might therefore be possible but mainly on a local scale. Only low risk of ecotoxic effects on organisms in marine sediments is foreseen. DBP is not a PBT chemical according to the OSPAR or the EUPBT criteria, however DBP is a potential endocrine disrupter. It is therefore suggested that any potential risk of DBP in this regard that are not covered in the present assessment should be evaluated in the context of a general approach towards endocrine disrupting substances.

Butylbenzyl phthalate: The measured concentrations in marine waters are in the range 0,006-1,8 µg/l, which is slightly lower than the proposed $PNEC_{aquatic(marine)}$ at 0,75 µg/l. $PNEC_{sediment(marine)}$ is 0,152 mg/kg dwt and measured concentrations in marine sediments range from <0,006-0,220 mg/kg dwt. It can thus not be excluded that a risk to organisms living in the marine water column as well as marine sediment dwellers may occur at a local level. BBP is not a PBT chemical according to the OSPAR or the EU PBT criteria, however BBP is a potential endocrine disrupter. It is therefore suggested that any potential risk of BBP in this regard that are not covered in the present assessment should be evaluated in the context of a general approach towards endocrine disrupting substances.

Di(2-ethylhexyl) phthalate: The measured concentrations in marine waters are in the range 0,0001-0,375 µg/l. On the basis of VTG measurements in female fathead minnow, a $PNEC_{aquatic(marine)}$ is estimated to be 0,3 µg/l. Thus potential effects might occur. $PNEC_{sediment(marine)}$ is 20 mg/kg dwt and measured concentrations in marine sediments range from 0,007-16 mg/kg dwt. Risks to marine sediment dwellers may not be expected at a local level. DEHP is not a PBT chemical according to the OSPAR or the EU PBT criteria, however DEHP is a potential endocrine disrupter. It is therefore suggested that any potential risk of DEHP in this regard that are not covered in the present assessment should be evaluated in the context of a general approach towards endocrine disrupting substances.

Di(isononyl) phthalate: No risk of direct ecotoxic effects in the pelagic or the sediment compartment is foreseen. Further DINP is not a PBT chemical according to the OSPAR or the EU PBT criteria. With present consumption of DINP and taking the low toxicity of DINP into account no risk is foreseen for secondary poisoning via the food chain.

Di(isodecyl) phthalate: No risk of direct ecotoxic effects in the pelagic or the sediment compartment is foreseen and DIDP is not a PBT chemical according to the OSPAR or the EU PBT criteria With present consumption of DIDP and taking the toxicity of DIDP into account no risk is foreseen for secondary poisoning via the food chain.

2. Introduction

The purpose of the present review is to describe the emission of selected phthalates to the marine environment as well as their potential ecotoxic effects in order to make recommendations regarding the risk of a selected group of phthalates.

The review comprises the following phthalates:

- Di-n-butyl phthalate (DBP), CAS No. 84-74-2
- Butylbenzyl phthalate (BBP), CAS No. 85-68-7
- Di(2-ethylhexyl) phthalate (DEHP), CAS No. 117-81-7
- Di(isononyl) phthalate (DINP), CAS Nos 28553-12-0 and 68515-48-0
- Di(isodecyl) phthalate (DIDP), CAS Nos 26761-40-0 and 68515-49-1

3. Dibutylphthalate

3.1 Production, use and emission

3.1.1 Production

In 1998, dibutylphthalate (DBP) was produced at three production sites in the EU. The total production was 26 000 tonnes in 1998, of which 8000 tonnes were exported outside the EU. There was no import of DBP. The production of DBP has decreased during the nineties from approx. 49 000 t/year in 1994 to 26 000 in 1998 (EU RA DBP 2004).

3.1.2 Use

In 1998, the total consumption of DBP within the EU was 18 000 t/year as estimated from the total production and the amount exported outside the EU (Table. 3.1). The quantitative usage of DBP for the different use categories within the EU is estimated using the total consumption in 1998 and the percentage distribution data from 1997 (EU RA DBP 2004). The estimates are given in Table 3.1.

Table 3.1. Estimated quantitative usage distribution of DBP within the EU in 1998 based on the percentage distribution from 1997 (EU RA DBP 2004)

Industrial and use category	Distribution in 1997 (%)	Amount used within the EU in 1998 (t/year)
Polymers industry as softeners e.g. plasticizer in PVC	74	11 000
Adhesives in paper and packaging, wood building and automobile industry	13	2000
Pulp, paper and board industry as softener in printing inks	9	1300
Miscellaneous other applications as softener/solvent (e.g. sealants, nitrocellulose paints, film coatings glass fibres and cosmetics)	3	500

The data in Table 3.1 show that the most extensive usage of DBP is in the polymers industry, in which the substance is incorporated into plastic in order to increase its workability and distensibility (EU RA DBP 2004).

3.1.3 Emissions from diffuse sources

The main sources of the diffuse releases of DBP to the aquatic environment are (EU RA DBP 2004):

- cleaning of road tankers used for the distribution of DBP in the EU
- end use of plasticised PVC
- use of adhesives

Estimates for DBP released from these sources are summarised by the Netherlands (EU RA DBP 2004). The estimates using the production and consumption data from 1998 and the doses from diffuse sources according to the EU RA DBP (2004) are given in Table 3.2. The amount released from the diffuse sources corresponds to approx. 93% of the total losses of DBP (diffuse and point sources).

Table 3.2. Estimated diffuse emissions of DBP to water

Source	Total production/consumption of DBP	Losses of DBP in the EU	
		(%)	(t/year)
Cleaning of road tankers	26 000[1]	0,05	13
PVC products	11 000[2]	5	550
Adhesives	2 000		300

1) Total production of DBP in 1998
2) Total consumption of DBP for PVC

SOURCE INVESTIGATION

Vikelsøe et al (1998) investigated the emission of phthalates by analysis of waste water from car wash centres, a hospital, a laundry, a kindergarten and a WWTP in Denmark in 1996-1997. The results of the analyses showed emissions of DBP from car wash and the hospital while the concentrations of DBP in samples from the laundry and kindergarten were below the detection limits. The concentration intervals of the analyzed wastewater samples are given in Table 3.3.

Table 3.3. Concentration intervals of DBP in waste water samples from diffuse sources in Denmark (Vikelsøe et al. 1998)

Diffuse sources	Car wash 26 samples	Hospital 12 samples	Laundry 2 samples	Kindergarten 1 sample
DBP concentration in (µg/l) waste water	30-810	<60–118	<60	<1000

The release in the EU due to car washing was estimated assuming approx. $120 \cdot 10^6$ cars in the EU and two car washes per month (EU RA DINP 2003). Using the average release per wash of 10 mg found in the investigation of Vikelsøe et al. (1998), the release was estimated at 29 t/year.

An analysis of the phthalates emission into the environment in Denmark was performed in 1992-1993 and reported by Hoffmann (1996). The results from an experimental study of the release of phthalates from washing of textiles with PVC printing showed a release of different phthalates from not detectable (diphentylphthalate) up to a level of 12 000 µg per kg textiles per wash. The largest concentration was seen for DBP. The released DBP corresponded to 0,03-0,3% of the total amount of phthalates in the inks used for printing. In Denmark, the yearly release of DBP from washing of textiles is estimated by use of the data given by Hoffmann (1996):

120 t ink · 0,35 t/kg phthalates/t ink · (0,03-0,3%)/wash · 20 washes/year = 0,25-2,5 t DBP/year

With the assumption that the Danish use of textiles with PVC printing is applicable to the rest of the EU, the total emission will be:

(383 mill. inhabitants in the EU)/(5,3 mill. inhabitants in DK) · (0,25-2,5) t/year = 18-180 t/year

A recent study by Larsen et al. (2000) showed a lower release of total phthalates from washing of two textiles with PVC printing. The release was 1330 and 4300 µg phthalates per kg textile per wash for a textile of cotton and polyesters, respectively. The DBP release accounted for 710 and 1100 µg/kg in the textile of cotton and polyester, respectively. Using the results from this study and the same assumption as above the total emission in the EU will be 1,08-16,7 t/year or approx. 8,9 t/year. Furthermore, the study showed a phthalates release of 45, 130, 584 and 2860 µg/kg from textiles without PVC printing. The two lowest values were from polyester textiles containing phthalate carriers. It was not possible to explain the larger releases coming from cotton textiles.

The release from washing of textiles with PVC printing in the EU is assumed to be within 8,9 to 100 t/year or approx. 54 t/year.

The total consumption of phthalates in the Danish PVC glue production was approx. 190 t/year in 1992 (Hoffman 1996). The phthalates used were primarily DBP (63%) and BBP (37%). Based on these data and on an estimated release to waste water regarding the private use of glue of 0,75-15 t phthalates/year, the calculation of the emission of DBP originating from PVC glue to the waste water in the EU gave approx. 359 (34-683) t/year.

In 28 months laboratory studies under accelerated landfill conditions (Mersiowsky et al. 1999), DBP was detected in the leachate. Concentrations of the same order of magnitude were found for DEHP and BBP. In the EU-RAR (2001), an overall loss of DEHP to waste water from landfills was estimated at 15 t/a. Based on the relative consumption levels of DBP and DEHP, a loss of approx. 1 t/a can be estimated for DBP.

EFFLUENTS FROM MUNICIPAL WASTEWATER TREATMENT PLANTS

An overview of the DBP concentrations found in effluents from municipal wastewater treatment plants (WWTPs) is given in Table 3.4. The data are primarily from the EU RA DBP (2004), supplemented by data from measurements in Denmark (Grüttner et al. 1995) and the review given by the Nordic Council of Ministers (1993; 1996).

Table 3.4. DBP concentrations in the effluent from WWTPs

Location	Concentration (µg/l)	Reference
Three WWTPs, Norway	<0,06-1,54	EU RA DBP 2004
Five WWTPs, the Netherlands	<0,09-4,6	EU RA DBP 2004
WWTPs, Sweden	0,1-2,0	EU RA DBP 2004
Five urban WWTPs, France 1998	<2	EU RA DBP 2004
WWTP U.K., 1984	6,0	EU RA DBP 2004
WWTPs, Denmark	<0,5-<12	Grüttner et al. 1995
WWTPs, Sweden	1,2–10,8	Nordic Council of Ministers 1993; 1996
International data	n.d.–54	Nordic Council of Ministers 1996

n.d.: not detected

In 1998, the total amount of effluent from the WWTPs in Denmark was 802 mill. m^3 per year corresponding to 151 m^3 per inhabitant per year (Danish EPA 2000). Assuming the same amount of effluent waste water per inhabitant per year in the EU and a DBP concentration in the WWTP effluents of 3 µg/l, the total amount of DBP discharged from WWTPs in the EU (383 mill. inhabitants) to the aquatic environment will be 173 t/year.

STORM WATER

At two different locations in Denmark, an investigation from 1995 to 1996 of organic micropollutants in storm water showed DBP concentrations between <1,5 and 3,8 µg/l in 11 samples of storm water. The mean value of the concentrations was 1,3 µg/l. The total amount of storm water, i.e. run-off of rainwater, melted snow from roads and other paved or coated surfaces, discharged to recipients or WWTPs in Denmark are approx. 250 mill. m^3 per year. Approx. 60% of the storm water is expected to be discharged directly into an aquatic recipient (Danish EPA 1997). Based on these data, the following DBP emissions could be estimated:

- Total DBP emission through WWTPs: 0,1 t/year
- DBP emitted directly into the aquatic recipient: 0,2 t/year

Using the above conversing factor (72,3) for the ratio between emission in Denmark and the EU, the emission of DBP from storm water into the aquatic recipient and the WWTPs will be 14,1 and 9,4 t/year, respectively.

ESTIMATION OF THE DBP DISCHARGE FROM RIVER WATER TO THE MARINE ENVIRONMENT

The discharge of DBP from use in EU via rivers into the marine environment was estimated by use of the following assumptions:

- The yearly rain excess in the EU is assumed to correspond to the volume of river water discharged into the marine environment.
- The total rain excess in the EU is $710{,}6 \cdot 10^9$ m^3/year as estimated from rain excess data and surface area given by ECETOC (1994).
- Average concentrations in river water as appear from monitoring data, the calculated regional concentration (PECregional) and the PECregional based on monitoring data estimated in the EU RA DBP 2004 are used.

The concentrations of DBP in the water of the European rivers are approx. 0,1 to 1[1] µg/l (EU RA DBP 2004). This corresponds to a DBP discharge from rivers in the EU of 71-711 t/year. Concentrations as high as 2,2 µg/l have been measured in an upper estuary (sal. < 2%), which would correspond to a DBP discharge from rivers in the EU of 1563 t/year. Point sources as well as diffuse sources will contribute to this discharge. The discharge from diffuse sources accounts for approx. 93% of the total discharge of DBP (EU RA DBP 2004). PECregional has been estimated to 0,4 µg/l resulting in an estimated volume of 284 t/year discharged to the marine environment.

[1] $PEC_{regional}$ based on the upper limit of the range of average measured data EU RA DBP (2004).

It should be noted that these approaches for estimating emissions to the marine are intrinsically conservative, as direct discharges to the marine environment from cities, industries, etc. located along the coasts are not taken into account.

SUMMARY

A summary of the estimated release of DBP from the different diffuse sources above is given in Table 3.5.

Table 3.5. Summary of estimates of release of DBP to aquatic environments

Diffuse source	Emissions to surface and waste water (cf. EU RA DBP) (t/year)	Emissions to waste water (t/year)	Discharged to the aquatic environment (t/year)	Discharged with river water into the sea (t/year)
Total diffuse emission	863			
Car wash		29		
Textile wash		54		
Glue (private use)		359		
Storm water overflow		9,4	14,1	
WWTP emissions			173	
Rivers (based on monitoring data)				817 (71-1560) 391 (71-711)
Rivers (based on PECregional)				284
Σ	863	451	188	284-817 (391)

The amount of DBP in river waters corresponds to 1,1 - 4,50 % of the total consumption in the EU. It is assumed that this amount is emitted to the marine environment. However, estimates based on river concentrations (monitored or modelled) do not take direct discharge from industry or settlements situated in coastal regions into account and have to be added to this amount.

3.2 Concentrations in the marine environment

3.2.1 Estimated concentrations

The PEC at a regional scale is calculated (EU RA DBP 2004) by use of the EUSES model and input data based on the sum of released DBP from diffuse and point sources as well. When all distribution activities of DBP were assumed to take place within one region, the total daily release to water will be 315 kg (i.e. 115 t/year). Calculation based on this figure resulted in a regional PEC in fresh water at 0,4 µg/l and in sediment at 89 µg/kg dw.

The DBP concentration in the effluents from WWTPs varied from less than 0,06 to 54 µg/l, but with most measured data below 10 µg/l (Table 3.4). Assuming an initial dilution of 10 in the marine environment, the DBP concentration in a marine environment close to a WWTP effluent will be <0,006 to 5,4 µg/l.

3.2.2 Measured concentrations

Measured concentrations of DBP in the marine environment are given in Tables 3.6 and 3.7.

Table 3.6. DBP concentrations in marine water

Location	Concentration (µg/l)	Reference
Marine/estuarine areas	0,007-3,4	EU RA DBP 2004
Oslofjord, Drammensfjord, Grenlandsfjord, Iddefjord (seawater), Norway, 1995/96	<0,060	EU RA DBP 2004
Gulf of Mexico	0,034-0,093	SIME 99/3/21-E 1999
Kiel bight, Baltic area	0,059-0,20	SIME 99/3/21-E 1999
Crough Estuary, UK (salt water)	0,024-0,058	SIME 99/3/21-E 1999
Estuaries, UK (salt water)	0,012-4,8	SIME 99/3/21-E 1999
Estuaries, Germany (salt water)	(0)->0,5	SIME 99/3/21-E 1999
Mersey Estuary (saltwater , <2% salinity)	0,114-2,12	SIME 99/3/21-E 1999
Terra Nova Bay, Antarctica	0,025-0,373	Nordic Council of Ministers 1996
Sea water	0,046–3,4	Nordic Council of Ministers 1996
Estuaries	0,011–4,8	Nordic Council of Ministers 1996
Roskilde Fjord, Denmark 1998	0 – 0,043	Vikelsøe et al. 2001
Estuaries, The Netherlands	0 – 0,5	Belfroid et al. 1999

Table 3.7. DBP concentrations in marine sediment

Location	Concentration (mg/kg dw)	Reference
Oslofjord, Drammensfjord, Grenlandsfjord, Iddefjord (seawater), Norway, 1995/96	<0,020-0,102 (surface and 15 cm deep)	EU RA DBP 2004
Marine sediments Denmark, 1996-97	0,035-2,4	SIME 99/3/21-E 1999
Gulf of Mexico	<0,0001-0,0153	SIME 99/3/21-E 1999
Crough Estuary, UK	0,0122-0,0145 ww	SIME 99/3/21-E 1999
San Lois Pass	0,060	SIME 99/3/21-E 1999
Ems estuary	0,0255-0,0484 ww	SIME 99/3/21-E 1999
Helgoland bight	0,00525-0,0163 ww	SIME 99/3/21-E 1999
Terra Nova Bay & Toss Sea, Antarctica	0,005-0,037	Nordic Council of Ministers 1996
Marine & coastal sediments	<0,015-0,093	Nordic Council of Ministers 1996
Marine & coastal sediments	0,003-0,008 ww	Nordic Council of Ministers 1996
Roskilde Fjord, Denmark, 1998	0,043 - 0,143	Vikelsøe et al. 2001
Western Scheldt, The Netherlands, 1997	0,098	Belfroid et al. 1999
Suspended matter North Sea Channel, Ijmuiden, The Netherlands, 1997	1,488	Belfroid et al. 1999

ww = per wet weight

A summary of the concentrations shows that DBP is found in:

- estuary at 0,007-4,8 µg/l in water and 0,012-2,4 mg/kg ww in sediment;
- seas and oceans at an interval between 0,034-3,4 µg/l in water and <0,0001-0,32 mg/kg dw in sediment.

3.2.3 Conclusions

There was only very few details in the available literature concerning the diffuse emission of DBP. PVC products were found to be the main source of release to fresh water. Source investigations showed that wash of textiles with PVC printing and private use of glue resulted in a possible release to WWTPs of 54 and 359 t/year, respectively. Furthermore, it was estimated that an amount of 188 t/year was discharged into the

fresh and marine water environments from storm water and WWTPs. The total emission to the marine environment with river water was estimated to about 800 t/year corresponding to 4,5% of the annual consumption in the EU. Alternatively the total emission to the marine environment with river water was estimated to 300-400 t/year (1,5 – 2,2%) based on the modelled PECregional and the PECregional based on the average of the upper range of measured data respectively. However, estimates based on river concentrations (monitored or modelled) do not take direct discharge from industry or settlements situated in coastal regions into account and have to be added to this amount.

Measurements in the marine environment (estuary and ocean water) show concentrations of 0,007-4,8 µg/l in water and <0,0001-2,4 mg/kg dw in sediments.

3.3 Environmental properties

3.3.1 Physico-chemical properties

Physico-chemical properties are reported in the EU RA DBP (2004):

- Molecular weight = 278 g/mol
- Melting point = –69°C
- Boiling point = 340°C
- Density = 1,045 g/ml
- Vapour pressure = $9,7 \cdot 10^{-5}$ hPa at 25°C
- Water solubility = 10 mg/l
- log P_{ow} = 4,57

3.3.2 Degradation

Wolfe et al. (1980) measured the hydrolysis rate constant of DBP and estimated a half-life of 22 years at alkaline conditions. The hydrolysis half-life at neutral pH and 25°C is estimated to 3,4 years.

In the aquatic environment, only insignificant photodegradation is expected (Staples et al. 1997).

DBP is readily biodegradable in the OECD 301B test (Scholz et al. 1997). This result is confirmed by several other tests. For risk assessment purposes a degradation half-life in fresh surface water of 15 days is used (EU RA DBP 2004). A similar half-life can be expected in marine areas with relative high concentrations of suspended matter e.g. in estuaries. In marine areas where this is not the case a somewhat longer half-life may be expected, a half-life of 50 days is recommended by the EU Technical Guidance Document on risk assessment (EU, 2003). Under anaerobic conditions, ultimate biodegradation may be reached after long incubation periods (Staples et al. 1997, EU RA DBP 2004).

In sewage treatment plants, a removal of 91% is estimated of which 58% is degraded (EU RA DBP 2004).

Neither the OSPAR screening criterion for persistency nor the EU P criterion is therefore fulfilled, as the substance is considered readily biodegradable.

3.3.3 Bioaccumulation

The high K_{ow} of DBP indicates that the substance has a potential for bioaccumulation. However, the actual degree of bioaccumulation *in vivo* will be determined by the metabolisation and the elimination rate of the substance. For phthalates it is known that an important biotransformation pathway is the formation of the mono-ester and the subsequent formation of phthalic acid. In the review by Staples et al. (1996) and in the EU RA DBP (2004), several bioaccumulation studies with algae, crustaceans, fish and insects are referred. The bioconcentration factors determined on DBP for a number of organisms are presented in Table 3.8. When available, both BCF values including potential metabolites and BCF values based on the parent compound alone are given. For chemicals such as phthalates esters that are subject to metabolism, BCFs based on total radioactivity are expected to be higher than BCFs based on parent compound as radiolabelled metabolites may contribute significantly to total radioactivity. The main concern regards the mono-ester MBP. However, BCF values based on total ^{14}C measurements may overestimate the total BCF for DBP and MBP as other metabolites will exist as well as some of radiolabelled ^{14}C will be incorporated into the tissue after full metabolisation. The mono-ester is expected to have a low bioaccumulation potential as the log Kow is 2,8.

In the case where the toxicity of the metabolites i.e. the mono-ester may be of concern it should be considered to evaluate the potential bioaccumulation of the substance on the basis of the total BCF value including metabolites.

The BCF values found for the parent compound DBP is however much lower than those obtained on the basis of total ^{14}C measurements. The EU RA DBP (2004) recommends using the BCF value of 1,8 for the carp, however with some reservations because this study has several shortcomings. These reservations should be taken into account when evaluating the risk for secondary poisoning.

Table 3.8. Bioconcentration factors (BCF) of DBP for various aquatic organisms

Species	Exposure conc. (µg/l)	F/S	Duration (days)	BCF (including potential metabolites)	BCF (based on parent compound)	Reference
Selenastrum capricornutum	2,0e+08	F	1		5475 1324	Casserly et al. 1983 (Quoted in Staples et al. 1997)
Water flea *Daphnia magna*	0,08	F	14	5000		Mayer and Sanders 1973
Artemia sp.	2700-13 900	S	-	345		Hudson et al. 1981
Grass shrimp *Palaemonetes kadiakensis*	0,08	S	3	750		Sanders et al. 1973
Scud *Gammarus pseudolimnaeus*	0,1	F	14	6700		Mayer and Sanders 1973
Scud *G. pulex*	100	F	10	185		Thuren and Woin 1988
Midge *Chironomus plumosus*	0,18	F	7	714		Mayer and Sanders 1973
Fathead minnow *Pimephales promelas*	4,83	F	11	2068	167	Call et al. 1983
Fathead minnow *P. promelas*	34,8	F	11	2125	172	Call et al. 1983
Carp *Cyprinus carpio*	10-50	F	28		1,8	Hüls 1997 (quoted in EU RA DBP 2004)

Due to the ready biodegradability of DBP it is not expected that DBP has a potential for food chain transfer in the marine environment.

SUMMARY – BIOACCUMULATION

DBP may be bioaccumulated in aquatic biota, as demonstrated by the use of the ^{14}C technique, which include metabolites and incorporation into the tissue, however, studies on the basis of the parent compound points at a low bioaccumulation potential for fish. A BCF value of 1,8 was chosen in the EU RA DBP (2004). Neither the OSPAR screening criterion for bioaccumulation (BCF>500) nor the EU B criterion (BCF>2000) is therefore fulfilled.

3.3.4 Aquatic toxicity

MICROORGANISMS

The toxicity studies with microorganisms are summarised in Table 3.9. The table contains data on both bacteria and protozoa.

Table 3.9. Toxicity of DBP to microorganisms

Test species	(F/S)	N/M	Test duration	Endpoint	EC50 or LC50 (mg/l)	NOEC (LOEC) (mg/l)	Reference
Protozoa *Tetrahymena pyriformis*	F	N	24 h	Growth inhibition		1 (10)	Yoshizawa et al. 1977
Protozoa *T. pyriformis*	F	N	24 h	Reproductive inhibition	2,2		Yshioka et al. 1985
Protozoa *T. pyriformis*	F	N	48 h	Growth inhibition	7,0		Jaworska et al. 1995
Bacteria *Pseudomonas putida*	F	M	0,5 h	Respiration inhibition		(2500)	BASF AG, 1989
Bacteria *Photo-bacterium phosphoreum*	S	N	0,5 h	Light inhibition	10,8-23,2		Kaiser and Palabrica 1991
Laboratory Digester culture	F	M	5-193 h	Anaerobic meta-bolic inhibition		100	Johnson and Young 1983

F: Fresh water; S: Salt water; N: Nominal concentration; M: Measured concentration; NOEC: No Observed Effect Concentration; LOEC: Lowest Observed Effect Concentration.

In the test with *Pseudomonas putida*, the effect of DBP was found at concentrations far above the water solubility of the substance.

TOXICITY TO ALGAE

The toxicity studies with DBP for freshwater and marine algae are summarised in Table 3.10.

Table 3.10. Toxicity of DBP to algae

Test species	(F/S)	N/M	Test duration	Endpoint	EC50 or LC50 (mg/l)	NOEC (LOEC) (mg/l)	Reference
Chlorella pyrenoidosa	F	M	96 h	Growth inhibition	>13		Yan et al. 1995
Scenedesmus subspicatus	F	N	48 h	Cell multiplication inhibition	3,5		Kuhn and Pattard 1990
S. subspicatus	F	N	48 h	Growth rate	9,0		Kuhn and Pattard 1990
S. subspicatus	F	M	72 h	Cell growth	1,2	0,5	Scholz 1995
S. subspicatus	F	M	72 h	Growth rate	2,0	0,5	Scholz 1995
Chlorella emersoni	F	N	7 d	Growth and photosynthesis		2,78	Melin and Egneus 1983
Selenastrum capricornutum	F	M	10 d static	Cell number	0,40	0,21	Melnick and Schiller 1982
S. capricornutum	F	N	7 d	Growth and photosynthesis		2,78	Melin and Egneus 1983
S. subspicatus	F	M	72 h	Growth rate		1,2	Hüels 1995 (Quoted from the EU RA DBP, 2004)
Dunaliella parva	S	N	8 d	Cell aggregation		0,2	Acey et al. 1987

F: Fresh water; S: Salt water; N: Nominal concentration; M: Measured concentration; NOEC: No Observed Effect Concentration; LOEC: Lowest Observed Effect Concentration.

TOXICITY TO INVERTEBRATES

The short-term toxicity data on DBP for freshwater and marine invertebrates are presented in Table 3.11 and the long-term toxicity data on DBP for freshwater and marine invertebrates are presented in Table 3.12.

Table 3.11. Short-term toxicity of DBP to aquatic invertebrates

Test species	(F/S)	N/M	Test duration	Endpoint	EC50 or LC50 (mg/l)	NOEC (LOEC) (mg/l)	Reference
Midge *Chironomus plumosus*	F	N	48 h	Mortality	0,76 (0,52-1,10)		Suggatt and Foote 1981
Midge *C. plumosus*	F	M	48 h	Mortality	5,4 (3,5-7,5)		Mayer and Ellersieck 1986
Water flea *Daphnia magna*	F	M	48 h	Mortality	3,7		Call et al. 1983
Water flea *D. magna*	F	M	48 h static	Mortality	3,0	1,7	Adams et al. 1995
Water flea *D. magna*	F	N	48 h static	Mortality	5,2 (4,7-5,6)		McCarthy and Whitmore 1985
Water flea *D. magna*	F	M	48 h	Immobilization	3,4	1,3 (10,0)	Scholz 1994
Scud *Gammarus pseudolimnaeus*	F	N	96 h	Mortality	2,10		Sanders et al. 1973
Brine shrimp *Artemia salina*	S	N	24 h	Mortality	8,0		Hudson et al. 1981
Mysid shrimp *Mysidopsis bahia*	S	M	96 h static	Mortality	0,5	0,26	Adams et al. 1995
Copepod *Nitocra spinipes*	S	N	96 h	Mortality	1,7 (1,3-2,2)		Linden et al. 1979

F: Fresh water; S: Salt water; N: Nominal concentration; M: Measured concentration; NOEC: No Observed Effect Concentration; LOEC: Lowest Observed Effect Concentration.

Table 3.12. Long-term toxicity of DBP to aquatic invertebrates

Test species	(F/S)	N/M	Test duration	Endpoint	EC50 or LC50 (mg/l)	NOEC (LOEC) (mg/l)	Reference
Water flea *Daphnia magna*	F	M	21 d	Survival/ reproduction	0,20 (0,16-0,25)	0,11 (0,20)	DeFoe et al. 1990
Water flea *D. magna*	F	N	16 d	Fecundity		0,56 (1,8)	McCarthy and Whitmore 1985
Planarian *Dugesia japonica*	F	N	7 d	Head regeneratiom	0,54		Yoshioka et al. 1986
Scud *Gammarus pulex*	F	N	25 d	Locomotor activity		0,10 (0,50)	Thuren and Woin 1991
Estaurine Microcosm	S	M	2 weeks	Abundance and diversity		0,04 (0,34)	Tagatz et al. 1986

F: Fresh water; S: Salt water; N: Nominal concentration; M: Measured concentration; NOEC: No Observed Effect Concentration; LOEC: Lowest Observed Effect Concentration.

TOXICITY TO FISH

The short-term toxicity data on DBP for freshwater and marine fish are presented in Table 3.13 and the long-term toxicity data on DBP for freshwater and marine fish are presented in Table 3.14.

Table 3.13. Short-term toxicity data on DBP for fish

Test species	(F/S)	N/M	Test duration	Endpoint	EC50 or LC50 (mg/l)	NOEC (LOEC) (mg/l)	Reference
Gold fish *Carassius auratus*	F	N	NA	Change in heart rate		0,5 (1,0)	Pfuderer and Francis 1975
Channel catfish *Ictalurus punctatus*	F	M	96 h flow through	Mortality	0,46 (0,40-0,53)		Mayer and Ellersieck 1986
Bluegill sunfish *Lepomis macrochirus*	F	M	96 h static	Mortality	0,48	0,42	Adams et al. 1995
Bluegill sunfish *L. macrochirus*	F	N	96 h	Mortality	0,73 (0,42-1,28)		Mayer and Sanders 1973
Bluegill sunfish *L. macrochirus*	F	N	96 h	Mortality	1,2 (1,0-1,4)		Buccafusco et al. 1981
Bluegill sunfish *L. macrochirus*	F	N	96 h flow through	Mortality	1,55 (1,38-1,74)		Mayer and Ellersieck 1986
Carp *Cyprinus carpio*	F	N	43 h	Mortality (fed compound)		74-159 mg/kg	Loeb and Kelly 1963
Rainbow trout *Oncorhynchus mykiss*	F	M	96 h flow through	Mortality	1,6 (1,1-2,2)	0,5	Adams et al. 1995
Rainbow trout *O. mykiss*	F	N	96 h flow through	Mortality	1,48 (1,3-1,67)		Mayer and Ellersieck 1986
Yellow perch *Perca flavescens*	F	M	96 h flow through	Mortality	0,35 (0,28-0,44)		Mayer and Ellersieck 1986
Fathead minnow *Pimephales promelas*	F	N	96 h	Mortality	1,30 (0,31-5,45)		Mayer and Sanders 1973
Fathead minnow *P. promelas*	F	N	96 h flow through	Mortality	3,95 (3,47-4,5)		Mayer and Ellersieck 1986
Fathead minnow *P. promelas*	F	M	96 h flow through	Mortality	0,85 (0,72-1,0)		DeFoe et al. 1990
Fathead minnow *P. promelas*	F	M	144 h flow through	Mortality	0,92 (0,71-1,2)	0,32	Adams et al. 1995
Zebra fish *Brachydanio rerio*	F	M	96 h static	Mortality	2,2 (1,3-2,5)	1,3	Scholz 1994

F: Fresh water; S: Salt water; N: Nominal concentration; M: Measured concentration; NOEC: No Observed Effect Concentration; LOEC: Lowest Observed Effect Concentration.

Table 3.14. Long-term toxicity data on DBP for fish

Test species	(F/S)	N/M	Test duration	Endpoint	EC50 or LC50 (mg/l)	NOEC (LOEC) (mg/l)	Reference
Rainbow trout *Oncorhynchus mykiss*	F	M	90 d (60 d post hatch)	Growth/ survival		0,1 (0,19)	Rhodes et al. 1995
Fathead minnow *Pimephales promelas*	F	N	20 d flow through	Hatchability		0,56 (1,0)	McCarthy and Whitmore 1985

F: Fresh water; S: Salt water; N: Nominal concentration; M: Measured concentration; NOEC: No Observed Effect Concentration; LOEC: Lowest Observed Effect Concentration.

TOXICITY TO SEDIMENT-LIVING ORGANISMS

As phthalates tend to accumulate in sediments, toxicity to organisms living in or on the sediments is essential. No valid data on the toxicity of DBP to sediment-dwelling organisms are available. Only a multi-species experiment has been carried out, in which effects of DBP on benthic estuarine communities was studied at concentrations of 10, 100 and 1000 mg/kg (Tagatz et al. 1986). Laboratory and field colonized sand-filled boxes containing 40-58 species from 7-9 phyla were exposed for 8 weeks. Measured concentrations in sediments were 4-48% of the nominal concentrations at the end of the experiment. In laboratory studies, it was found that the annelids *Mediomastus californiensis*, mollusks *Acteocina canaliculata*, arthropods *Corophium acherusicum* and echinoderms *Leptosynopta spp*. were most common. Significant reductions in benthic diversity resulted from the 1000 mg/kg DBP in sediment exposures, with echinoderms being the most affected. In the field experiments, molluscs were the only taxon to be significantly affected. Only at the highest exposure concentration, effects on the community structure were observed. As the study is poorly reported and only sum parameters could be studied (low number per species), the NOEC derived from this study will not be used for the estimation of a PNEC (EU RA DBP 2004). However, the study may be used to analysis whether marine sediment dwellers could be expected to be particularly sensitive towards DBP. The experiments by Tagatz et al. (1986) have been reevaluated by Ammann et al. (1997), by using consistent statistical approaches. Based on the nominal exposure concentrations, NOECs and LOECs were re-evaluated. For the laboratory experiment, a NOEC value of < 10 mg/kg was determined. The most sensitive taxa were annelida. For the field experiment, the NOEC was 10 mg/kg and the most sensitive taxa were mollusca and phoronida. Based on measured concentrations (geometric mean of initial concentration of 10 mg/kg and final concentration of 4,1 mg/kg), a NOEC of 6,4 mg/kg can be derived.

MAMMALIAN TOXICITY

The CMR Working Group agreed in May 2000 to classify DBP for reprotoxicity Cat. 2; R61 and reprotoxocity Cat. 3; R62 i.e. for effects on development and fertility.

ENDOCRINE DISRUPTION

Jobling et al. (1995) studied the estrogenic effects of a range of chemicals, including DBP, commonly found in sewage effluents. Using cytosolic extract from liver of rainbow trout (*Oncorhynchus mykiss*), Jobling et al. (1995) documented that DBP binds to the receptor, inhibiting the binding of natural estradiol. DBP also showed mitogenic effect on the *in-vitro* growth of human breast cancer cell at test concentrations of 2,78 mg/l. In transiently transfected breast cancer cells, DBP was reported to affect the transcriptional activity of the estrogen receptor (Jobling et al. 1995). DBP concentrations in the range of 2,8 to 27,8 mg/l stimulated the activity. In a study by Harris et al. (1997), DBP was found to be estrogenically active using a recombinant yeast screening. The relative potency of DBP was approx. $1 \cdot 10^6$ times less than 17β-estradiol. In a study by Wine et al. (1997), levels of 52-794 mg/kg DBP were daily dosed to male and female rats. In conclusion, this study showed that DBP is a reproductive/developmental toxicant in rats exposed both as adults and during development. In a two-generation reproduction study with mice, effects were observed at dose levels of 0,17 g/kg bw/day. No effects were observed at 0,39 g/kg bw/day (Lamb et al. 1997).

Sharpe et al. (1995) assessed whether exposure of male rats to xenoestrogens during gestation and during the first three weeks after birth affects the size of their testes and sperm production in adult life. No effects of DBP were described.

The vitellogenin induction in rainbow trout after intraperitoneal injection of DBP was investigated by Christensen et al. (2000). No significant vitellogenin response was seen when DBP was dosed 3 times, on

day 0, 6 and 12, during an 18-d exposure scheme at concentrations of 500 and 1000 mg/kg body weight (wwt).

DBP has shown estrogen receptor affinity in *in vitro* assays. Several long-term tests with fish and invertebrates are available, but specific endpoints with respect to estrogenic activity were not studied in these tests (EU RA DBP 2004).

SUMMARY - EFFECT

The effect concentrations found for different microorganisms showed relatively high variability.

DBP has been shown to be acutely toxic (EC50 or LC50 values) to algae, crustaceans and fish in the range of 0,35-8,0 mg/l and is thus considered very toxic to aquatic organisms. In an estuarine microcosm, the abundance and diversity of crustaceans were affected at low concentrations and NOEC was determined to be 0,04 mg/l. NOEC levels in chronic toxicity tests with crustaceans and fish were both close to 0,1 mg/l (EU RA DBP 2004).

No valid sediment toxicity test results are available (EU RA DBP 2004).

DBP has shown estrogen receptor affinity in *in-vitro* experiments with human breast cancer cells. *In-vitro* experiments with cytosolic extracts from rainbow trout liver also showed estrogenic effects of DBP, but no effects were detected in *in-vivo* tests with rainbow trout. Long-term *in-vivo* studies with rats and mice have shown that DBP is a reproductive/developmental toxicant. Based on the results available, it is concluded that DBP has a potential for endocrine disrupting effects in the marine environment. However, as DBP is readily degradable the risk for endocrine disrupting effects in marine mammals is probably low.

3.3.5 PNEC for the aquatic environment

Toxicity data are available on short-term tests with bacteria, protozoa, algae, crustaceans and fish. Long-term toxicity data are available on algae, crustaceans and fish with NOEC values for crustaceans and fish at 0,1 mg/l as the lowest. The PNEC for the aquatic compartment is derived from the 99-day NOEC of 100 μg/l for rainbow trout *(Oncorhynchus mykiss)*. This key study is supported by the study with scud *(Gammarus pulex)*, in which a similar value was found based on a decrease in locomotor activity. An assessment factor of 10 will be used for deriving a $PNEC_{aquatic}$ for freshwater environments resulting in a $PNEC_{aquatic}$ = 10 μg/l as also proposed by the EU Risk Assessment of DBP (EU RA DBP 2004). As recommended by the revised EC Technical Guidance Document on risk assessment (EU, 2003), an assessment factor of 100 is used on the lowest NOEC data for deriving a PNEC for the marine environment as long-term toxicity data are only available on three trophic levels. The following $PNEC_{aquatic\ (marine)}$ is therefore proposed:
$PNEC_{aquatic(marine)}$ = 1 μg/l.

For the freshwater sediment compartment, a $PNEC_{sediment}$ = 3,1 mg/kg dwt has been calculated from the $PNEC_{aquatic}$ (EU RA DBP 2004). Using an extra assessment factor of 10 results in a $PNEC_{sediment(marine)}$ = 0,3 mg/kg dw. However, using the study by Tagatz et al. (1986) and Ammann et al. (1996), indicatively a $PNEC_{sediment(marine)}$ = 0,64 mg/kg dw can be derived. These two PNEC values agree well with each other.

3.4 PBT assessment and risk assessment

DBP cannot be considered a PBT chemical as neither the OSPAR screening PBT criteria nor the EU P, B and T criteria are fulfilled. However it has the potential to cause endocrine disrupting effects.

The measured concentrations in marine waters are in the range of 0,007-4,8 µg/l, which is in the same range as the proposed $PNEC_{aquatic(marine)}$ at 1 µg/l. As the relevance and representativity of the measured concentrations can not be assessed, it is tentatively concluded that there is a potential risk for ecotoxic effects on aquatic species in the marine environment. However, the freshwater $PEC_{regional}$ of 1 µg/l based on monitoring data (modelled $PEC_{regional}$ = 0,4 µg/l) (EU RA DBP 2004) do indicate that the potential risk towards aquatic species may be on a local scale rather than a general problem for the marine area.

In sediments, concentrations are measured in the range of <0,0001-2,4 mg/kg dwt, which is in the same range as the proposed $PNEC_{sediment(marine)}$ at 0,3 mg/kg dwt (if including an extra assessment factor of 10) or alternatively $PNEC_{sediment(marine)}$ at 3,1 mg/kg dwt. It is thus concluded that there is a low risk of ecotoxic effects on organisms in marine sediments likely to be on a limited scale.

DBP has a potential for endocrine disrupting effects. However, as DBP is expected to degrade relatively rapidly in the environment, and as the bioaccumulative potential is expected to be low in the food chain no risk for marine mammals is foreseen.

3.5 Desired Reduction and Identification of possible measures

There is a potential for that DBP locally may exhibit a risk towards marine aquatic organisms but generally no risk is foreseen. Consequently, no risk reduction is proposed within OSPAR. However, DBP is a potential endocrine disrupter. It is therefore suggested that any potential risk of DBP in this regard that are not covered in the present assessment is evaluated in the context of a general approach towards endocrine disrupting substances.

4. Butylbenzylphthalate

This section has been written on the basis of available information including the draft of EU RAR (2004). It is not expected that the finalisation of the EU RAR will make a revision of this draft necessary for the purpose of the OSPAR Hazardous Substance Strategy.

4.1 Production, use and emission

4.1.1 Production

The Production in the EU was approx. 45 000 t/year in the years 1994-1997 (EU RA BBP 2004).

4.1.2 Use

The consumption within EU is approx. 36 000 t/year. The main uses are (EU RA BBP 2004):

Use	% of EU consumption
Plastisol coating (PVC flooring)	60
Sealants (glass insulation/construction)	25
Leather/cloth coating (PVC)	5
Films, calendering (PVC wall covering, flooring)	3
Coatings, inks (car care, construction, paper, board)	2
Adhesives	1
General PVC	3
Other non-polymer[1]	1

4.1.3 Emissions from diffuse sources

SOURCE INVESTIGATION

A summary of the measured BBP concentrations in waste water from diffuse sources reported by Vikelsøe et al. (1998) is given in Table 4.1. The results of the analyses showed emissions of BBP from car wash and kindergarten while the concentrations of BBP in samples from the laundry and hospital were below the detection limits.

Table 4.1. Concentration intervals of BBP in wastewater samples from diffuse sources in Denmark (Vikelsøe et al. 1998)

Diffuse sources		Car wash 26 samples	Hospital 12 samples	Laundry 2 samples	Kindergarten 1 sample
BBP concentration in waste water	(µg/l)	0,5-150	<2	<2	320

In the EU, the release due to car washing was estimated as described for DBP and using the average release per wash of 2,2 mg from the investigation of Vikelsøe et al. (1998). The estimated value for the EU was 6,3 t/year.

Emissions of 191-2962 µg BBP/kg textile per wash were seen in the two Danish studies of phthalates release from textile wash (Hoffman 1996; Larsen et al. 2000). In the EU, the yearly release of BBP from washing of textiles with PVC printing was estimated as described for DBP (Section 3.1.3). A release of 0,29-45 t/year or approx. 23 t/year was calculated.

[1] According to a recent Swedish survey BBP occurs in very few cosmetic products in Europe and only in trace amounts (RAR 2004). According to the Norwegian Food Control Agency BBP is no longer used in cosmetics in Norway.

Estimations of the BBP release to waste water from the private use of PVC glue in the EU gave an amount of approx. 54 t/year based on DBP emission scenario (Section 3.1.3).

In 28 months laboratory studies under accelerated landfill conditions (Mersiowsky et al. 1999), BBP was detected in the leachate. Concentrations of the same order of magnitude were found for DEHP and BBP. Comparable losses from PVC flooring were observed for DEHP and BBP.

In the EU-RAR, an overall loss of DEHP to waste water from landfills was estimated at 15 t/year. Based on the relative consumption levels of BBP and DEHP, a loss of approx. 1,5 t/year can be estimated for BBP.

In the EU RAR (2004) an estimate is given assuming that waste management in the EU is as in the UK i.e. nearly 100% landfilling and using a leachate concentration of 8 μg/l from a Swedish landfill lysimeter. This estimation gives a yearly emission from landfills to waste water of 2,1 t/year.

Due to the use pattern of BBP waste remaining in the environment is expected to be of very minor importance for this substance (Response to comments from Norway to EU Technical Meeting on risk assessment 2001).

EFFLUENT FROM MUNICIPAL WASTEWATER TREATMENT PLANTS

Table 4.2. BBP concentrations in the effluent from WWTPs

Location	Concentration (μg/l)	Reference
WWTP, Norway	0,06-0,5	EU RA BBP 2004
WWTP, Germany	< 0,04	EU RA BBP 2004
WWTP, Denmark	< 0,5	Boutrup et al. 1998
WWTPs , Denmark	<0,03-0,5	Grüttner et al. 1995
WWTP, Sweden	1,6-5,2	Nordic Council of Ministers 1993
WWTP, The Netherlands 1997	n.d. – 0,07	Belfroid et al. 1999
International data	n.d.-35,9	Nordic Council of Ministers 1996

n.d.: not detected

Assuming a BBP concentration in the WWTP effluents of 0,3 μg/l, the total amount of BBP discharged from the WWTPs to the aquatic environment will be 17,4 t/year in the EU (Section 3.1.3).

STORM WATER

The result of the Danish investigations of storm water in 1995-96 for BBP (Danish EPA 1997) and the estimated amounts for the EU are summarised below:

	Denmark	EU
• Mean BBP concentrations in storm water:	0,41 μg/l	
• Estimated BBP emission to waste water:	0,04 t/year	3,0 t/year
• BBP emitted directly into the aquatic recipient:	0,06 t/year	4,4 t/year

ESTIMATION OF THE BBP DISCHARGE FROM RIVER WATER TO THE MARINE ENVIRONMENT

In the EU, the discharge of BBP from rivers to the marine environment was estimated using the assumptions listed for DBP (Section 3.1.3).

The concentrations of BBP in the water of European rivers (River Rhine) are approx. 0,01-0,09 μg/l (SIME 99/3/21-E 1999). According to the above assumption, this corresponds to a BBP discharge from rivers in the EU of 7,1-64 t/year. Point sources as well as diffuse sources will contribute to this discharge. $PEC_{regional}$ has been estimated to 0,20 μg/l (EU RA BBP 2004) resulting in an estimated volume of 142 t/year discharged to the marine environment. However, estimates based on river concentrations (monitored or modelled) do not take direct discharge from industry or settlements situated in coastal regions into account and have to be added to this amount.

SUMMARY

A summary of the estimated release of BBP from the different diffuse sources above is given in Table 4.3.

Table 4.3. Summary of estimates of release of BBP to aquatic environments

Diffuse source	Emissions to surface water from diffuse sources (t/year)	Emissions to waste water (t/year)	Discharged to the aquatic environment (t/year)	Discharged with river water to the sea (t/year)
Total diffuse emission	?	620[1)]		
Car wash		6,3		
Textile wash		23		
Glue (private use)		211		
Storm water overflow		3,0	4,4	
WWTP emissions			17,4	
Rivers (based on monitoring data)				36 (7,1-64)
Rivers (based on PECregional)				142
Σ	?	245	22	36-142

[1)] EU RA BBP 2004

4.2 Concentrations in the marine environment

4.2.1 Estimated concentrations

In the effluents from WWTPs, the BBP concentrations varied from less than 0,03 to 35,9 µg/l (Table 4.2). Assuming an initial dilution of 10 in the marine water, the BBP concentration in a marine environment close to a WWTP effluent will be <0,003 to 3,6 µg/l.

4.2.2 Measured concentrations

Measured concentrations of BBP in marine environment are given in Tables 4.4 and 4.5.

Table 4.4. BBP concentrations in marine water

Location	Concentration (µg/l)	Reference
Estuaries	0,3	Nordic Council of Ministers (1996)
Mersey Estuary (1985)	0,006-0,029	SIME 99/3/21-E 1999
Mersey Estuary (1985)	0,011-0,021	SIME 99/3/21-E 1999
Mersey Estuary (1986)	n.d. - 0,135	SIME 99/3/21-E 1999
Roskilde Fjord, Denmark, 1998	0,0015 – 0,0071	Vikelsøe et al. 2001
Netherlands, Rhine and Meuse 22 stations	0,01-0,6 µg/l (3 samples each station) median 0,08 and 0,06 µg/l	Vethaak et al. 2002
Netherlands, Western areas, 11 stations	0,01-1,0 µg/l (3 samples each station) median 0,04 µg/l	Vethaak et al. 2002
Netherlands, North Sea, 4 stations	0,01-1,8 µg/l (3 samples each station)	Vethaak et al. 2002

n.d.: not detected (detection limit: 0,017-0,041 µg/l)

Table 4.5. BBP concentrations in marine sediment

Location	Concentration (µg/kg)	Reference
Denmark, 1996-1997	< 10 - 220	Lillebæltsamarbejdet 1998
Roskilde Fjord, Denmark, 1998	2,7 – 7,0	Vikelsøe et al. 2001
Norway, coast (1996)	< 6-112	SIME 99/3/21-E 1999
Netherlands, North Sea, 3 stations	<0,01 – 0,02 µg/kg dwt (3 samples each station)	Vethaak et al. 2002

4.2.3 Conclusions

Investigations of the release in the EU from different diffuse sources showed releases of 211 and 23 t/year from the private use of glue and the washing of textiles with PVC printing, respectively. The estimated release from car wash was 6,3 t/year and the release from storm water to waste water was 2,9 t/year. Furthermore, the total amounts of BBP discharged to fresh and marine water environments from municipal WWTPs and storm water were estimated to be 22 t/year.

In the EU RA BBP (2004) a total release to waste water is 686 t/year, which after treatment gives a release to surface water of 28 t/year. A total release to marine water with rivers was estimated to 36-142 t/year. However, estimates based on river concentrations (monitored or modelled) do not take direct discharge from industry or settlements situated in coastal regions into account and have to be added to this amount.

A BBP concentration of < 0,003-3,6 µg/l was estimated for the marine environment close to the effluent of WWTPs (initial, diluted waste water). This estimate was based on measured BBP concentrations in effluents from different WWTPs.

Measurements in the marine environment showed concentrations of 0,006-1,8 µg/l in water and ~0-220 µg/kg in sediment.

4.3 Environmental properties

4.3.1 Physico-chemical properties

Physico-chemical properties are reported in the EU RA BBP (2004):

- Molecular weight = 312 g/mol
- Melting point < –35°C
- Boiling point = 370°C
- Density = 1,114-1,122 g/ml
- Vapour pressure = 0,00112 Pa at 20°C
- Water solubility = 2,8 mg/l
- log P_{ow} = 4,84

4.3.2 Degradation

The ready biodegradability has been tested in various tests showing ultimate biodegradability in the range from 10% to 81% (CITI 2000, Staples et al. 1997). It is concluded that BBP is readily biodegradable. For risk assessment purposes a degradation half-life in fresh surface water of 8-10 days is used (EU RA BBP 2004). A similar half-life can be expected in marine areas with relative high concentrations of suspended matter e.g. in estuaries. In marine areas where this is not the case a somewhat longer half-life may be expected, a half-life of 50 days is recommended by the EU Technical Guidance Document on risk assessment (EU, 2003). Studies on anaerobic biodegradation in sediments show 78-88% degradation within 22-35 days (Painter & Jones 1990). In 4 different sediments half-lives of 10, 15, 63 and ∞ were determined (Painter & Jones 1990).

In sewage treatment plants, a removal of 92% is estimated of which 50% is degraded (EU RA BBP 2004).

BBP is considered readily biodegradable, therefore neither the OSPAR screening criterion nor the EU P criterion are fulfilled.

4.3.3 Bioaccumulation

No studies on the bioaccumulation in algae and crustaceans were found. Therefore only bioaccumulation studies on fish will be referred.

FISH

Several bioaccumulation studies have been performed on bluegill sunfish (*Lepomis macrochirus*) with total BCFs varying from 188 (17 days of exposure with a flow-through test procedure and an exposure concentration of 2 μg/l) (Heidolph and Gledhill 1979) to 663 (flow-through test procedure and an exposure concentration of 9,7 μg/l; exposure period is not known) (Barrows et al. 1980). In a study by Carr (1992), the total BCF was determined to 449 (3 days of exposure with a flow-through test procedure and an exposure concentration of 34 μg/l). In this test, the corresponding BCF of the parent compound was determined to be 12.

As the toxicity of the metabolites is of concern it should, however, be considered to use a BCF value that includes metabolites. The GLP study by Carr (1992) shows rapid metabolisation and excretion, which indicates that the BCF based on ^{14}C measurements is overestimated. As a first approach the BCF value of 449 can be used for estimating secondary poisoning (EU RA BBP 2004).

Due to the ready biodegradability of BBP, it is not expected that BBP has a potential for food chain transfer in the marine environment. Neither the OSPAR screening criterion for bioaccumulation (BCF>500) nor the EU B criterion (BCF>2000) is therefore fulfilled.

4.3.4 Aquatic toxicity

TOXICITY TO MICROORGANISMS

The toxicity studies with microorganisms are summarised in Table 4.6. The table contains data on both bacteria and protozoa.

Table 4.6. Toxicity of BBP to microorganisms

Test species	(F/S)	N/M	Test duration	Endpoint	EC50 or LC50 (mg/l)	NOEC (LOEC) (mg/l)	Reference
Protozoa *Tetrahymena pyriformis*	F	N	24 h	Growth inhibition		50 (100)	Yoshizawa et al. 1977
Activated sludge inocula	F	N	0,5 h	O_2 consumption inhibition		2,8	Volskay and Grady 1988

F: Fresh water; S: Salt water; N: Nominal concentration M: Measured concentration; NOEC: No Observed Effect Concentration; LOEC: Lowest Observed Effect Concentration.

TOXICITY TO ALGAE

The toxicity studies with BBP for freshwater and marine algae are summarised in Table 4.7.

Table 4.7. Toxicity of BBP to algae

Test species	(F/S)	N/M	Test duration	Endpoint	EC50 or LC50 (mg/l)	NOEC (LOEC) (mg/l)	Reference
Navicula Pelliculosa	F	M	96 h	Cell number	0,60 (0,2-2,0)	0,3	Gledhill et al. 1980
Selenastrum capricornutum	F	N	96 h	Chlorophyll a	0,11 (0,02-0,28)	<0,07	Sugatt and Foote 1981
S. capricornutum	F	N	96 h	Cell number	0,13 (0,02-0,37)		Sugatt and Foote 1981
S. capricornutum	F	M	96 h	Cell number	0,40 (0,2-1,0)	0,1	Gledhill et al. 1980
Dunaliella Tertiolecta	S	M	96 h	Cell number	1,0 (0,2-5,0)	0,3	Gledhill et al. 1980
Skeletonema costatum	S	M	96 h	Cell number	0,6 (0,3-2,0)	0,1	Gledhill et al. 1980
S. costatum	S	N	96 h	Chlorophyll a	0,17 (0,08-0,36)	<0,03	Sugatt and Foote 1981
S. costatum	S	N	96 h	Cell number	0,19 (0,09-0,38)		Sugatt and Foote 1981
Scenedesmus subspicatus	F	M	72 h	Cell number	1,5	0,15	Huntingdon 005/002303. Quoted from EU RA BBP (2004)

F: Fresh water; S: Salt water; N: Nominal concentration M: Measured concentration; NOEC: No Observed Effect Concentration; LOEC: Lowest Observed Effect Concentration.

As appears from Table 4.7, the toxicity data obtained on the different algal species are in close agreement with EC50 values between 0,1 and 1,5 mg/l and NOEC values <0,03 and 0,3 mg/l.

TOXICITY TO INVERTEBRATES

The short-term toxicity data on BBP for freshwater and marine invertebrates are presented in Table 4.8 and the long-term toxicity data on BBP for freshwater and marine invertebrates are presented in Table 4.9.

Table 4.8. Short-term toxicity of BBP to aquatic invertebrates

Test species	(F/S)	N/M	Test duration	Endpoint	EC50 or LC50 (mg/l)	NOEC (LOEC) (mg/l)	Reference
Water flea *Daphnia magna*	F	M	48 h flow through	Mortality	1,8	0,82	Adams and Heidolph, 1985
Water flea *D. magna*	F	M	48 h	Mortality	3,7 (3,0-4,6)	1	Gledhill et al. 1980
Hydra littoralis	F	N	96 h flow through	Mortality	1,1 (0,5-2,0)		ABC Laboratories 1986
Midge *Chironomus Tentatus*	F	N	48 h flow through	Mortality	1,64 (1,22-2,17)		Calvert et al. 1982
Mysid shrimp *Mysidopsis bahia*	S	M	96 h	Mortality	0,9 (0,7-1,2)	0,4	EG&G Binomics BP-79-4-38. Quoted from EU RA BBP (2004)
Mysid shrimp *M. bahia*	S	N	96 h	Mortality	9,63 (7,67-1,26)	3,55	Suggatt and Foote 1981

F: Fresh water; S: Salt water; N: Nominal concentration; M: Measured concentration; NOEC: No Observed Effect Concentration; LOEC: Lowest Observed Effect Concentration.

Table 4.9. Long-term toxicity of BBP to aquatic invertebrates

Test species	(F/S)	N/M	Test duration	Endpoint	EC50 or LC50 (mg/l)	NOEC (LOEC) (mg/l)	Reference
Water flea *Daphnia magna*	F	M	42 d	Survival/ reproduction		0,26 (0,76)	Gledhill et al. 1980
Water flea *D. magna*	F	M	21 d static	Growth/ reproduction		0,35 (0,70)	Adams and Heidolph 1985
Water flea *D. magna*	F	M	21 d flow through	Reproduction		0,26 (0,76)	Adams and Heidolph 1985
Mysid shrimp *Mysidopsis bahia*	S	N	28 d flow through	Reproduction/ growth		0,075 (0,17)	Springborn Bionomics 1986

F: Fresh water; S: Salt water; N: Nominal concentration; M: Measured concentration; NOEC: No Observed Effect Concentration; LOEC: Lowest Observed Effect Concentration. In all tests performed, except for one acute test with *Mysidopsis bahia*, the effect concentrations of BBP were equal to or below the water solubility of the substance. Furthermore, many of the effect concentrations determined are based on measured exposure concentrations. Lowest acute EC50 in a good quality study with *Chironomus tentans* is 0,9 mg/l (EG&G Binomics BP-79-4-38; Quoted from EU RA BBP (2004)). In a valid 28 d reproduction test with *Mysidopsis bahia* a NOEC of 0,075 mg/l (Springborn Bionomics 1986) was determined.

TOXICITY TO FISH

The short-term toxicity data on BBP for freshwater and marine fish are presented in Table 4.10 and the long-term toxicity data on BBP for freshwater and marine fish are presented in Table 4.11.

Table 4.10. Short-term toxicity of BBP to fish

Test species	F/S	N/M	Test duration	Endpoint	EC50 or LC50 (mg/l)	NOEC (LOEC) (mg/l)	Reference
Bluegill sunfish *Lepomis Macrochirus*	F	M	96 h	Mortality	1,7 (1,0-2,8)	0,336	Adams et al. 1995
Rainbow trout *Oncorhynchus mykiss*	F	M	96 h flow through	Mortality	0,82 (0,48-1,4)	0,28	E&G Binomics, 1983 (Quoted fro EU RA BBP, 2004
Rainbow trout *O. mykiss*	F	M	96 h	Mortality	3,3 (2,9-3,9)	<0,36	Gledhill et al. 1980
Fathead minnow *Pimephales promelas*	F	M	96 h flow through	Mortality	2,1 (1,7-2,5)	1	Gledhill et al. 1980
Fathead minnow *P. promelas*	F	M	96 h flow through	Mortality	1,5 (1,0-2,4)	0,44	Adams et al. 1995
Fathead minnow *P. promelas*	F	M	14 d	Mortality	2,3 (1,3-3,8)		Gledhill et al. 1980
Shinner perch *Cymatogaster aggregata*	S	M	96 h flow through	Mortality	0,51 (0,46-0,56)		Ozretich et al. 1983
Shinner perch *C. aggregata*	S	M	165 h flow through	Mortality	0,49 (0,45-0,56)		Ozretich et al. 1983
English sole *Parophrys vetulus*	S	N	96 h flow through	Mortality	0,55 (0,48-0,64)		Randall et al. 1983
Sheepshead minnow *Cyprinodon variegatus*	S	M	96 h	Mortality	3.0 (2,4-3,9)	1	Gledhill et al. 1980

F: Fresh water; S: Salt water; N: Nominal concentration; M: Measured concentration; NOEC: No Observed Effect Concentration; LOEC: Lowest Observed Effect Concentration.

Table 4.11. Long-term toxicity of BBP to fish

Test species	F/S	N/M	Test duration	Endpoint	EC50 or LC50 (mg/l)	NOEC (LOEC) (mg/l)	Reference
Rainbow trout *Oncorhynchus mykiss*	F	N	109 d	Survival/ Growth		0,20	Rhodes et al. 1995
Fathead minnow *Pimephales promelas*	F	M	30 d	Survival/ Growth		0,14 (0,36)	Gledhill et al. 1980

F: Fresh water; S: Salt water; N: Nominal concentration; M: Measured concentration; NOEC: No Observed Effect Concentration; LOEC: Lowest Observed Effect Concentration.

In all tests performed, except for the acute tests with rainbow trout (*O. mykiss*) and sheepshead minnow (*C. variegatus*), the effect concentrations of BBP were equal to or below the water solubility of the substance. Furthermore, most of the effect concentrations determined are based on measured exposure concentrations. The lowest acute LC50 from a good quality study is 0,51 mg/l for *Cymatogaster aggregate* (Ozretich et al., 1983). With respect to chronic tests the lowest NOEC value determined is 0,14 mg/l from the study with *Pimephales promelas* (Gledhill et al., 1980).

TOXICITY TO SEDIMENT-LIVING ORGANISMS

No valid data on the toxicity of BBP to sediment-dwelling organisms are available.

MAMMALIAN TOXICITY

BBP shall be classified for reprotoxicity Cat. 2; R61 and Cat. 3; R62 i.e. for developmental effects and effects on fertility (29th ATP of Annex I to Directive 67/548/EEC).

ENDOCRINE DISRUPTION

Jobling et al. (1995) studied the estrogenic effects of a range of chemicals, including BBP, commonly found in sewage effluents by use of cytosolic extract from liver of rainbow trout, *Oncorhynchus mykiss*. In this study, it was documented that BBP binds to the receptor, inhibiting the binding of natural estradiol. It has also been shown that BBP has mitogenic effect on the *in-vitro* growth of human breast cancer cell at test concentrations of 3,12 mg/l. In transiently transfected breast cancer cells, BBP was reported to affect the transcriptional activity of the estrogen receptor. BBP concentrations in the range of 0,31 to 31,2 mg/l stimulated the activity. In a study by Sharpe et al. (1995), BBP was found to be estrogenically active using a recombinant yeast screening. The relative potency of BBP was approx. $1 \cdot 10^6$ times less than that of 17β-estradiol. Sharpe et al. (1995) assessed whether exposure of male rats to xenoestrogens during gestation and during the first three weeks after birth affects the size of their testes and sperm production in adult life. BBP was added to the drinking water of the pregnant female rat at low concentrations (1 mg/l). In adult life, males thus exposed had testes which were reduced in size by 5-13% and a 10-21% reduction in their sperm production capacity. As regard a potential anti-androgen-like effect of BBP, BBP was shown *in vitro* to be a potentanti-androgen in yeast cells expressing the androgen receptor. Eight *in vivo* studies are available which indicate an anti-androgen-like activity of BBP or its major metabolites in rats, MbuP and MBeP (Piersma et al., 2000; Gray et al., 2000; Parks et al., 1999; Imajima et al., 1997; Shono et al., 2000; Nagao et al., 2000; Tyl et al., 2004; Ema et al., 2003). Effects reported in the Piersma et al., 2000 study included a reduction in testicular weight in offspring, and effects on testicular migration from 270 mg/kg bw/day and 580 mg/kg bw/day, respectively after *in utero* exposure to BBP from gestation day 6 to 20. In the Gray et al., 2000 study malformations in the reproductive organs in 84 % of male offspring (approx. 90 days of age) exposed to 750 mg/kg bw/day BBP from gestation day 14 through postnatal day 2 were reported. Furthermore in the Gray et al., 2000 and Parks et al., 1999 studies a reduced ano-genital distance and testis weight in males at day 2 of age, and males with areolas at day 13 of age were reported. In the Imajima et al., 1997 study and the Shono et al., 2000 study testicular descendent was studied, which is under androgenic control. In this study the testis were located significantly higher in the abdominal cavity on gd 20 compared to control rats exposed *in utero* to MBuP from gd 15-18. Furthermore, in the Imajima et al., 1997 study, on pnd 30-40 cryptorchidism was reported in 84,6 % of the exposed offspring, compared to 0 % in the control group. In the study by Ema et al., 2003 *in utero* exposure to MBeP on gd 15-17 was shown to induce a significant decrease in AGD and a significant increase in the incidence of undescended testis. In the study by Nagao et al., 2000 a decrease in the weights of the testis, epididymis, and seminal vesicle, and tubular atrophy and decreased germinal epithelium were reported in F1 male offspring exposed to 500 mg/kg bw/day BBP during gestation and lactation and evaluated at weaning or after puberty. Furthermore, a decrease in anogenital distance was reported in male offspring in the 500 mg/kg bw/day dose group, which is a sensitive indicator of anti-androgen activity. In the Tyl et al., 2004 study a doserelated decrease in absolute and adjusted AGD was reported in F1 and F2 male pups from 250 mg/kg bw/day. Furthermore, at 750 mg/kg bw/day in F1 and F2 offspring a significant decrease in reproductive organ weights, and a significant increase in the % of males with reproductive tract malformations were reported. A potential anti-androgen-like effect of DBP has been indicated in different studies (Mylchreest et al., 1998; Ema et al., 1998b; Gray et al.,1998; Foster et al., 1998). In some of these studies the authors proposed that the majormetabolite of DBP; MBuP may elicit an anti-androgen-like effect.

The vitellogenin induction in rainbow trout after intraperitoneal injection of BBP was investigated by Christensen et al. (2000). Significant vitellogenin response was seen when BBP was dosed 3 times, on day 0, 6 and 12, during an 18-d exposure scheme both at concentrations of 500 and 1000 mg/kg body weight (wwt).

In a partial life cycle test (exposure from egg until sexual maturity, 60 days post hatch) performed by the Japanese Ministry of Environment on Japanese medaka *(Oryzias latipes)* significantly increased time to hatch and increased length and weight were obtained at concentrations $\geq$ 2,7 μg/l. Although not statistical significant a tendency towards increased vitellogenin (VTG) levels in males and decreased VTG levels in females were obtained.

European Council for Plasticisers & Intermediates (ECPI) has informed that it is not possible to perform a test for the evaluation of endocrine effects due to technical problems with keeping actual exposure concentrations. ECPI has concluded that both BBP and the primary metabolites disappeared very quickly from the test system. However, the industry is still requested to perform the endocrine effect test although this would not meet the ideal test requirements (e.g. constant actual exposure concentrations). It would be an important conclusion if the outcome of a test would be that there were no endocrine effects observed at concentrations comparable to those measured in the environment.

SUMMARY - EFFECT

Variable results were found in the toxicity tests with microorganisms.

BBP has been shown acutely toxic (EC_{50} or LC_{50} values) to algae, crustaceans and fish in the range of 0,1-3,7 mg/l whereas algae seemed slightly more sensitive than crustaceans and fish. BBP is thus considered very toxic to aquatic organisms. NOEC levels in chronic toxicity tests with algae, crustaceans and fish were observed in the range of 0,03-0,35 mg/l.

There are valid long-term tests representing 3 tropic levels. Valid NOEC values available are:

- Fish: 30d early life stage test *(Pimephales promelas):* 0,14 mg/l
- Invertebrates: 21d reproduction test (*Mysidopsis bahia*): 0,075 mg/l
- Algae: 72h growth inhibition test *(Scenedesmus subspicatus)*: 0,15 mg/l

The NOEC values determined are all well below the solubility limit of BBP and are thus valid for use in the estimation of a PNEC value.

BBP is not expected to have a potential for food chain transfer due to rapid metabolisation and excretion at the higher level of the food chain and due to expectedly relatively rapid degradation.

In *in-vitro* and *in-vivo* studies with both mammalian and aquatic organisms, endocrine disrupting effects are determined. Significant effect on the vitellogenin synthesis was observed when BBP was injected into rainbow trout at concentrations of 500 and 1000 mg/kg body weight (wwt). The dose levels seem high compared to what may be expected for the aquatic environment. As no NOEC value was determined in the experiment by Christiansen et al. (2000), BBP is, however, expected to have a potential for causing endocrine disrupting effects on aquatic species.

Due to the mammalian toxicity the OSPAR and EU T criterion are fulfilled.

4.3.5 PNEC for the aquatic environment

Toxicity data are available on short-term tests with bacteria, protozoa, algae, crustaceans and fish. Long-term toxicity data are available on algae, crustaceans and fish. An assessment factor of 10 can be applied to the lowest valid NOEC (0,075 mg/l) deriving a $PNEC_{aquatic}$ of 7,5 μg/l. An assessment factor of 10 should normally be used for deriving a $PNEC_{aquatic}$ for the freshwater environment and an extra assessment factor of 10 is proposed for deriving PNEC for the marine environment as recommended by the revised EU TGD on risk assessment (EU, 2003) resulting in $PNEC_{aquatic(marine)}$ = 0,75 μg/l.

There is only one test available regarding toxicity to micro-organisms showing a NOEC on respiration activity in activated sludge at 2,8 mg/l. Applying an assessment factor of 10, a $PNEC_{microorganisms}$ of 0,28 mg/l is derived.

No effects data are available for the sediment compartment. Using the $PNEC_{aquatic(marine)}$ = 0,75 μg/l the $PNEC_{sediment(marine)}$ can be estimated with the equilibrium partitioning method.

$PNEC_{sediment} = PNEC_{aquatic} \times (Kp_{sed\text{-}water} \times 1000/RHO)$
$PNEC_{sediment} = 0{,}00075 \times 262{,}8 \times 1000/1150 = 0{,}172$ mg/kg

where:
RHO = 1150
$Kp_{sed\text{-}water} = Fwater_{solid} + Fsolid_{sed} \times Kp_{sed} \times RHOsolid/1000 = 262{,}8$
and where
$Fwater_{solid} = 0{,}8$
$Fsolid_{sed} = 0{,}2$
$Kp_{sed} = Foc_{comp} \times Koc = 0{,}05 \times 10500$
RHOsolid = 2500

4.4 PBT assessment and Risk assessment

Neither the OSPAR screening criteria nor the EU criteria for persistency and bioaccumulation are fulfilled. Therefore BBP cannot be considered as a PBT chemical. However there might be a risk of potential endocrine disrupting effects.

The measured concentrations of BBP in marine water are in the range 0,006-1,8 µg/l, which is slightly higher than the proposed $PNEC_{aquatic(marine)}$ at 0,75 µg/l. Therefore, it is concluded that there might be a risk of ecotoxic effects in the marine environment.

For the sediment compartment only a few measurements are available in the range < 0,006-0,220 mg/kg where the maximum concentration found is slightly higher than the provisional $PNEC_{sediment(marine)}$ of 0,172 mg/kg. On this basis it cannot be excluded that a risk to marine sediment dwellers may occur at a local level. However, on the basis of the available (sparse) monitoring data it is unlikely that there is a risk of general concern.

4.5 Desired Reduction and Identification of possible measures

There seems to be only a low risk of ecotoxic effects in the marine environment. There is a potential for that BBP may exhibit a risk towards organisms living in the marine water column at specific location based on few scarce monitoring data in the marine waters. Furthermore, locally, it may also exhibit a risk towards marine sediment organisms based on the estimated $PNEC_{sediment(marine)}$ but generally no risk for the sediment compartment is foreseen. Consequently, no risk reduction is proposed within OSPAR. However, BBP is a potential endocrine disrupter. It is therefore suggested that any potential risk of BBP in this regard that are not covered in the present assessment is evaluated in the context of a general approach towards endocrine disrupting substances.

5. Di(2-ethylhexyl) phthalate

5.1 Production, use and emission

5.1.1 Production

Eighteen companies producing and/or importing di(2-ethylhexyl) phthalate (DEHP) have been listed in the EU RA DEHP (2001). In 1997, the production of DEHP in the EU (Western Europe) was 595 000 t/year and the consumption was 476 000 t/year (EU RA DEHP 2001). The consumption of DEHP can be assumed to be significantly high for the past 30 years. There are no specific consumption data on DEHP for this period. However, data on the amounts of DEHP and DIOP (di-octyl phthalates) together during the period from 1979 to 1998 show yearly consumptions between 350 000 and 500 000 tonnes. An estimation from a manufacture in the 1970s says that the production of DIOP was approx. 10-20% of the DEHP production. In the EU RA DEHP (2001), it was assumed that the same ratio also accounted for the consumption of DIOP and DEHP.

In 1997, 186 000 tonnes of the production in the EU was exported while an import of approx. 67 000 t/year was calculated from the consumption, export and production figures within Europe. There is no information on the DEHP imported into or exported from the EU in products. A Swedish investigation referred in the EU RA DEHP (2001) shows that the EU is the main “countries of origin” for most groups of articles containing DEHP. Exceptions are clothes made of plastics, e.g. PVC, mainly imported from Asia and plastic high boots. It was concluded that the main amount of articles containing DEHP consumed in the EU are produced in the EU but, for some product groups, the imported amount is significant. The export of articles containing DEHP was assumed to be in the same order of magnitude as the import.

In the EU, almost all the consumed phthalates including DEHP are transported via road tankers and approx. 130 kt/year of the plasticizers are transported by ship within the EU. Assuming that DEHP constitutes 47% of all plasticizers, the amount of DEHP transported by ship was calculated at 61 kt/year (EU RA DEHP 2001).

5.1.2 Use

The use of DEHP may be divided into three main product groups (EU RA DEHP 2001):

1. PVC
2. Non-PVC polymers
3. Non-polymers

An overview of the distribution of DEHP within polymers and non-polymers are shown in Table 5.1. The main part of DEHP used in polymers is used for plasticizers in PVC. Non-polymers include use as adhesives and sealant, lacquers and paints, printing inks for paper and plastics, printing inks for textiles, rubber and ceramics for electronic purposes.

Table 5.1. Material types containing DEHP based on industry data. The polymer applications for outdoor use are divided into the different uses (EU RA DEHP 2001)

Material	Distribution	DEHP (t/year)	Indoor uses (t/year) (approx. 78%)	Outdoor uses (t/year) (approx. 22%)	
Polymer – total	97%	462 000	362 000		100 000
				Roofing material	1000
				Roofing (coil coating)	5000
				Cables	20 000
				Coated fabric	21 000
				Hoses & profiles	6000
				Car undercoating	7000
				Shoe soles	40 000
Non-polymers	3%	14 280	*		
Total	100%	476 000			

* No data available.

Estimates of the amounts of DEHP used in different industrial productions are given in the EU RA DEHP (2001). Table 5.2 shows the estimated use of DEHP for the different application or use category in the EU in 1997.

Table 5.2. Estimated quantitative usage distribution of DEHP for different applications in Western Europe 1997 (EU RA DEHP 2001)

Use category	Amount use of DEHP within the EU in 1997 (t/year)
Calendering: Film/sheet and coated products Flooring, roofing, wall covering	 71 400 34 748
Extrusion: Hose and profile Wire and cable Compounding[1]	 57 120 80 920 85 680
Injection moulding: Footwear and miscellaneous (from compounding)	 (83 680)
Spread coating: Flooring General (coated fabric, wall-covering, coil coating etc)	 39 032 76 160
Other plastisols: Car undercoating Slush/rotational moulding, dip coating	 7140 9520
Adhesives/sealant, rubber	11 000
Lacquers and paints	1430
Printing ink (paper and plastics)	1640
Ceramics	210
Total	476 000

1) The semi-product (compound) is assumed to be produced by extrusion and to be further used for extrusion, injection moulding and plastisol applications at an unknown number of downstream users.

The figures in Table 5.2 show that film/sheet and coated products, wire and cable, footwear and miscellaneous, and coated fabric etc. are among the most important applications of DEHP.

There are no details available on the use of the end-products, e.g. the distribution between professional and private use.

5.1.3 Emissions from diffuse sources

Diffuse releases of DEHP mainly occur from the following sources:

- Transportation or distribution of pure DEHP;
- Polymer productions leading the waste water to municipal WWTPs;
- Non-polymer productions leading the waste water to municipal WWTPs;
- End-product use (indoor and outdoor);
- Waste.

Estimation of the environmental release from these sources has been performed by the use of life-cycle stages for different scenarios (EU RA DEHP 2001). The calculated releases directly to the waste water and surface water are given in Table 5.3.

Table 5.3. Estimated emissions of DEHP to fresh water (surface water) and waste water from diffuse sources in the EU

Diffuse source		Total emission of DEHP in the EU	
		Surface water (t/year)	Waste water (t/year)
Transportation		0	50
Polymer production		0	4,3
Non-polymer production		0	0,82
End-product use	Washing clothes with PVC printing	0	99
	Washing and abrasion of polymer floors	0	1212
	Car undercoating	23	46
	Roofing material	24,4	0
	Coils coating	261	0
	Fabric coating	219	0
	Cables & wires – air	62,6	0
	Hoses & profiles	15,6	0
	Shoe soles	36,6	0
	Sealants, adhesives etc.	58,1	16
	Lacquers & paint	99	198
	Printing ink	0	0
Waste	Municipal landfills (leakage water)	0	15,0
	Waste remaining in the environment	2413	0
Total		3212	1641

SOURCE INVESTIGATION

A summary of the measured DEHP concentrations and the estimated release to waste water reported by Vikelsøe et al. (1998) is given in Table 5.4.

Table 5.4. Concentration intervals of DEHP in wastewater samples from diffuse sources in Denmark (Vikelsøe et al. 1998)

Diffuse sources	Car wash 26 samples	Hospital 12 samples	Laundry 2 samples	Kindergarten 1 sample
DEHP concentration in waste water (µg/l)	5,2-760	2,1–35	91–130	<600

The figures in Table 5.4 show that the highest DEHP concentrations were measured in wastewater from laundry and car washing.

An investigation of the release of DEHP from car wash sites showed concentrations in the interval of 17-260 µg DEHP/l (Nielsen et al. 2000). These values are within the intervals found by Vikelsøe et al. (1998). The estimated average release of DEHP per car wash was 17 mg and 16 mg, respectively, in the two investigations. In the EU, the release due to car washing was estimated to 49 t/year as described for DBP and using the average release per wash of 17 mg.

Emissions of 170-6101 µg DEHP/kg textile per wash were seen in two Danish studies of phthalates release from textile wash (Hoffman 1996; Larsen et al. 2000). The yearly release in the EU of DEHP from wash of textiles with PVC printing was estimated as described for DBP (Section 3.1.3). A release of 0,26-93 t DEHP/year or approx. 46 t/year was calculated.

The DEHP concentrations detected in a Swedish investigation of household wastes from six different areas showed values of 3-272 µg/l (Nordic Council of Ministers 1993). In a Danish monitoring program in 1996, DEHP analysis was performed in domestic waste water from four different areas. The mean DEHP concentration was 31 µg/l (Jepsen & Grüttner 1997). Estimation of the DEHP released from households in Denmark gave an amount of 4,6 · 383 · 10^6 · 365) 643 t/year for the whole EU was obtained assuming the same volume of release from the population in the EU (383 mill. people).

EFFLUENT FROM MUNICIPAL WASTEWATER TREATMENT PLANTS

DEHP concentrations measured from monitoring of municipal WWTP effluents in different countries are summarised in Table 5.5.

Table 5.5. DEHP concentrations in the effluent from WWTPs

Location	Concentration (µg/l)	Reference
Three WWTPs, Sweden 1989-1991	<1-28	EU RA DEHP 2001
Four WWTPs, Denmark 1992-1996	0,5-28	EU RA DEHP 2001 Pedersen et al. 1998
Three WWTPs, Norway 1996	0,068-0,127	EU RA DEHP 2001
WWTPs, Germany 1992	0,54-0,90	EU RA DEHP 2001
WWTP, The Netherlands 1997	1 – 2,9	Belfroid et al. 1999
Henriksdal WWTP, Sweden	15-28	Nordic Council of Ministers 1996

Assuming a DEHP concentration in the Danish WWTP effluents of 10 µg/l, the total amount of DEHP discharged from the WWTPs into the aquatic environment will be 584,7 t/year in the EU (Section 3.1.3).

STORM WATER

The results of the Danish investigations of storm water in 1995-1996 for DEHP are summarised below (Danish EPA 1997):

	Denmark	EU
• Mean DEHP concentrations in storm water:	32 µg/l	
• DEHP emission to WWTPs:	3 t/year	231 t/year
• DEHP emitted directly into the aquatic recipient:	5 t/year	347 t/year

ESTIMATION OF THE DEHP DISCHARGE FROM RIVER WATER INTO THE MARINE ENVIRONMENT

In the EU, the discharge of DEHP from rivers into the marine water was estimated according to the assumptions listed for DBP (Chapter 3).

The whole- water concentrations of DEHP in the European rivers are approx. 0,006-21 µg/l (EU RA DEHP and DINP 2001). According to the above assumptions, the DEHP discharge from rivers in the EU will be 4,3-14 923 t/year. However, it should be noted that this estimate is based on the min. and max. concentrations found in surface water. No attempt has been made in this report to establish a representative concentration for surface water.

Based on a calculated PECregional of 2,4 µg/l dissolved DEHP, the discharge to the marine environment is estimated to 1705 tonnes/year. Estimates based on river concentrations (monitored or modelled) do not take direct discharge from industry or settlements situated in coastal regions into account or discharges of DEHP adsorbed to particles if based on the calculated $PEC_{regional}$. The discharge from diffuse sources accounts for approx. 82% of the total discharge of DEHP (EU RA DEHP 2001).

SUMMARY

A summary of the estimated release of DEHP from the different diffuse sources above is given in Table 5.6.

Table 5.6. Total diffuse emission of DEHP in the EU according to the EU RA DEHP and the releases estimated in this report

Diffuse source	Emissions to surface water from diffuse sources (t/year)	Emissions to waste water (t/year)	Discharged to the aquatic environment (t/year)	Discharged with river water into the sea (t/year)
Total diffuse emission (Table 5.3)	3212	1641		
Car wash		49		
Textile wash		46		
Household (assumed to incl. textile wash)		643		
Storm water overflow		231	347	
WWTP emissions			585	
Rivers (based on monitoring data)				7464 (4-14 900)
Rivers (based on PECregional)				1705
Σ	3212	969[1]	932	1705-7464

1) car wash, domestic waste water and storm water

The data in Table 5.6 show that domestic wastewater accounts for approx. 39% of the total DEHP emission to waste water, of which floor cleaning (1212 t/year) is seen to be the most important source. About twice as much is emitted to surface water from polymer waste and debris remianing in the environment, see Table 5.3.

The discharge into the aquatic environment from diffuse sources is estimated to be 4072 (3140 + 932) t/year. The total discharge of DEHP into water was estimated to 5822 t/year by EU RA DEHP (2001). The estimates of the amount of DEHP in river waters are within the same order of magnitude. Based on the calculated PECregional the discharge of DEHP to marine waters via surface water is 1705 t/year. The estimated emission of DEHP to the marine environment is approx. 0,4-1,6% of the total consumption.

5.2 Concentrations in the marine environment

5.2.1 Estimated concentrations

In literature, no estimated concentrations for DEHP in the marine environment are available. Generic estimated values of DEHP calculated by EUSES model are given in the EU RA DEHP (2001) for surface water and WWTP (local). The local values calculated for the diffuse emission from production, end-product

use and waste are given in Table 5.7 together with the regional and continental values for the total release (diffuse and point sources) of DEHP. The diffuse emission due to transportation of DEHP, municipal land fill and waste remaining in the environment were not included in the local scenario but only in the regional and continental scenarios.

Table 5.7. Predicted Environmental Concentration (PEC) of DEHP estimated by the EUSES model (EU RA DEHP 2001)

Step in EUSES	**Source**	**WWTP (µg/l)**	**Surface water (µg/l)**
LOCAL Diffuse emission	End-product use, polymer and non-polymer production	0,03	4,8
	Waste (from air emission): Car shredder Incineration	-	 2,2 2,2
REGIONAL Diffuse & point		-	2,4
CONTINENTAL Diffuse & point		-	0,3

The DEHP concentrations found in effluents from WWTPs were 0,068-28 µg/l (Table 5.5). Assuming an initial dilution factor of 10 in sea and ocean, the DEHP concentration in the marine environment will be 0,0068-2,8 µg/l.

5.2.2 Measured concentrations

Measured concentrations of DEHP in marine environment are given in Tables 5.8 and 5.9 for the water and sediment phase, respectively.

Table 5.8. DEHP concentrations in marine water

Location	Concentration (µg/l)	Reference
Gullaugbukta, Holmen, Fugelvik, Brattöya, Frierflaket, Breviksfjorden, Langesundsbukta, Ormöya, Slemmestad, Gåsöyrenna, Faerder; Norway, 1996	(0)-0,375	EU RA DEHP 2001
Coast Gulf of Mexico before 1978	0,0006-0,316	SIME 99/3/21-E 1999
Open Gulf of Mexico before 1978	0,006-0,097	SIME 99/3/21-E 1999
Atlantic Ocean before 1978	0,0001-0,006	SIME 99/3/21-E 1999
Mersey Estuary (salinity <2%); 1985-1986	0,083-0,693	SIME 99/3/21-E 1999
Crough estuary	0,058-0,078	SIME 99/3/21-E 1999
German estuary	(0)->0,5	SIME 99/3/21-E 1999
Roskilde Fjord, Denmark 1998	0,071– 0,191	Vikelsøe et al. 2001
Estuary, The Netherlands	0,04 – 1,9	Belfroid et al. 1999?

Table 5.9. DEHP concentrations in marine sediment

Location	Concentration (mg/kg dw)	Reference
Århus havn, Århus bugt, Mariager fjord, Stavns fjord, Randers fjord, 68 location in the South Western part of the inner Danish waters (Little Belt); Denmark 1996-1998	<0,05-5,4 0,03 – 16,0	Boutrup et al. 1998 Lillebæltsamarbejdet 1998
Frierflaket, Gullaugbukta, Holmen, Fuglevik, Brattöya, Breviksfjorden, Langesundsbukta, Faerder, Ormöya, Slemmestad, Gåsöyrenna; Norway 1996	0,034-6,551	EU RA DEHP 2001
Terra Nova bay; Antartic 1987-1988	0,007-0,14 (dw?)	EU RA DEHP 2001
Western Baltic Sea	0-0,2	SIME 99/3/21-E 1999
Ems estuary	0,030-0,0605	SIME 99/3/21-E 1999
Helgoland bight	0,0233-0,2223	SIME 99/3/21-E 1999
Crough estuary	0,0112-0,0262	SIME 99/3/21-E 1999
Roskilde Fjord, Denmark, 2001	0,021 – 0,724	Vikelsøe et al. 2001
Netherlands (suspended matter)	<0,1 – 0,51	SIME 05/2/Info. 17 2005
Netherlands (sediment)	0,12 – 0,83	SIME 05/2/Info. 17 2005
North Sea Channel, Ijmuigen, The Netherlands. Suspended matter	26	Belfroid et al. 1999
North Sea Channel, Ijmuigen. Sediment	0,106	Belfroid et al. 1999
Western Scheldt, Terneuzen, The Netherlands, Suspended matter	0,586	Belfroid et al. 1999

A summary of the concentrations show that DEHP is found in:

- estuaries and fjords at 0,058 – 1,9 µg/l in water and 0,0112-16,0 mg/kg dw in sediment
- sea and oceans at an interval of between 0,0001-0,375 µg/l in water and 0,007- 0,12 (5,4[1]) mg/kg dw in sediment. Suspended matter concentrations as high as 26 mg/kg dw has been reported from the North Sea though much lower concentrations seems more representative.

5.2.3 Conclusions

The main part of DEHP is used in semi-products used for extrusion, injection moulding and plastisol application, footwear and miscellaneous, and in general applications as coated fabric, wall covering, coil coating etc. The review of the diffuse release from different sources showed that washing and abrasion of polymer floors were the most important source for the emission of DEHP to waste water. The estimated release was 1212 t/year. The releases from the use of lacquers and paints, sealants and adhesives etc. were 198 and 16 t/year, respectively. The estimated release from car wash and washing of textiles with PVC printing was 49 and 46 t/year, respectively. Furthermore, it was estimated that an amount of 932 t/year was discharged into the fresh and marine water environments from storm water and WWTPs. The estimated emission of DEHP into the marine environment is 4 000-7 500 t/year corresponding to approx. 0,9-1,6% of the total consumption.

Measurements in the marine environment (estuary and ocean water) show concentrations of 0,0001-1,9 µg/l in water and 0,007- 16,0 mg/kg dw in sediments.

[1] Harbour sediment

5.3 Environmental properties

5.3.1 Physico-chemical properties

Physico-chemical properties are reported in the EU RA DEHP (2001):

- Molecular weight = 391 g/mol
- Melting point = –55°C
- Boiling point ~ 230°C
- Density = 0,980-0,985 g/ml
- Vapour pressure = $3,4 \cdot 10^{-7}$ hPa at 20°C
- Water solubility = 0,003 mg/l
- log P_{ow} = 7,5

5.3.2 Degradation

Abiotic degradation in aquatic environments is reported to be very slow with a hydrolysis half-life of approx. 2000 years (Howard 1989) and a very slow photooxidation as well (TSD 1991).

In most reported tests for ready biodegradability, the mineralization is lower than the pass level for judging DEHP as readily biodegradable. In one test, however a degradation of 82% was determined (Hüels 1994). In tests, in which adapted inocula are used, and in inherent biodegradability tests, considerably higher degradation is determined. It is concluded that, with regard to WWTPs, DEHP should be considered readily biodegradable (EU RA DEHP 2001). Based on simulation test results, a half-life of 50 days has been proposed for the freshwater environment (EU RA DEHP 2001) EUSES calculations. A similar half-life can be expected in marine areas with relative high concentrations of suspended matter e.g. in estuaries. In marine areas where this is not the case, a somewhat longer half-life may be expected. A half-life of 150 days is recommended by the EU Technical Guidance Document on risk assessment (EU, 2003).

In tests for anaerobic biodegradation, almost no degradation was found even at favourable temperatures and after relatively long incubation periods (EU RA DEHP 2001).

In sewage treatment plants, a removal of approx. 93% is estimated of which 15% is degraded. Thus 7% is expected to be released to the environment (EU RA DEHP 2001).

In conclusion, DEHP is inherently biodegradable and passes as readily biodegradable according to at least one of the available studies in the RAR. On balance, the P-criterion is therefore not fulfilled (EAF, 2004).

5.3.3 Bioaccumulation

The bioconcentration factors determined on di(2-ethylhexyl) phthalate for a number of organisms are presented in Table 5.10. The results of the studies show that the bioaccumulation of DEHP varies between different aquatic species. The studies referred are all performed with radiolabelled DEHP. The measured radioactivity thus refers to total ^{14}C-recidues, and the concentration of DEHP may be overestimated. As, however, the main metabolisation product of DEHP is the reprotoxic MEHP, these data are considered suitable for the purpose of risk assessment. In general, the bioaccumulation of DEHP decreases at concentrations higher than approx. 5 μg/l. A possible explanation is that, at a test concentration above the non-colloidal water solubility (approx. 3 μg/l), a significant amount of DEHP is in the colloidal form, which might make it less bioavailable.

The bulk of measured concentrations of DEHP in fish are in the range of 0,1 – 10 mg/kg ww EU RA DEHP 2001. Flounder from the North Sea Channel, Ijmuigen, contained 2,15 mg/kg dw in the liver. In the most extensive study (Pfannhauser et al. 1997), 8 out of 170 fish samples contained concentrations of DEHP above 1 mg/kg with a maximum value of 2,6 mg/kg ww. These measured concentrations are in the same order of magnitude as the predicted concentrations based on the water-fish transfer only. If the measured concentrations are assumed to be caused by the water-fish exposure route only the BCF for the high-end measured data would be in the order of 300-1000 if the true water solubility is 3 μg/l. This corresponds with the recent findings from a multigeneration study with fathead minnow (Caunter et al., 2004). In this study it was concluded that the main uptake route was via the water phase and with an exposure concentration of approx. 3 μg/l a BCF value of 553 was determined.

A study by Mackintosh et al. (2004) report the distribution of DEHP and 7 other individual phthalate esters in a marine aquatic food web (18 different species, representing approx. 4 different trophic levels). Based on lipid equivalent concentration the conclusion from the study was that the concentration of DEHP did not increase at higher trophic levels in the marine food web. More or less the same concentrations were found in

lower trophic levels (e.g. zooplankton) as in the higher trophic levels (e.g. fish). This indicated that DEHP did not biomagnify in the studied aquatic food web. No measurements of the metabolite MEHP were performed.

Table 5.10. Bioconcentration factors (BCF) of DEHP for various aquatic organisms

Species	Exposure conc. (µg/l)	Duration (days)	BCF (including potential metabolites)	Reference
Algae				
Oedogonium sp.	340	33 d	53 890	Metcalf et al. 1973
Chara chara	1430	27 d	18 263	Södergren 1982, cited from Lundberg and Nilsson 1994
Invertebrates				
Water flea *Daphnia magna*	3,2 10 32 100	21 d	241 190 330 312	Brown and Thomson 1982
Midge *Chironumus plumosus*	0,3	7 d	350	Mayer and Sanders 1973. Quoted from EU RA DEHP 2001
Acartia sp.	1 10 100	30 d	5376 4485 1995	Perez et al. 1983. Quoted from EU RA DEHP 2001
Scud *Gammarus pulex*	1430	27 d	24 456	Södergren 1982, cited from Lundberg and Nilsson 1994
Blue mussel *Mytilus edulis*	4,1 42,1	28 d	2366 2627	Brown and Thomsen 1982b
Fish				
Bluegill sunfish *Lapomis macrochirus*	5,8	28 d	114	Barrows et al. 1980
Fathead minnow *Pimephales promelas*	1,9 2,5 4,6 8,1 14 30 62	56 d	737 880 891 444 357 287 155	Mehrle and Mayer 1976
Rainbow trout *Oncorhynchus mykiss*	5 14 54	28 d	78 113 42	Mehrle and Mayer 1976
Carp *Cyprinus carpio*	3 48 144	42 d	221 32 20	Scholtz et al. 1998. Quoted from EU RA DEHP 2001
Fathead minnow	3	200 d	553	Caunter et al., 2004

In the EU RA DEHP (2001) the bioconcentration potential is summarised as:

Type of prey	BCF*	Reference
Fish	840 wwt	(Mayer et. al. 1976)
Invertebrate, mussels	2500 wwt	(Brown & Thomson 1982)
Invertebrates, amphipods	2700	(Mayer & Sanders, Walsh 1973)

* based on C-14 technique

SUMMARY - BIOACCUMULATION

The two mentioned BCF values obtained in algae are both from mesocosmos studies. Both studies have been performed with C^{14}-labelled DEHP and the BCFs given thus include potential metabolites. In the study with *Chara chara*, the exposure concentration of 1,4 mg/l is much higher than the solubility limit and, consequently, the BCF value may either be underestimated or being a result of adsorption to the algae.

As DEHP is readily adsorbed onto organic surfaces and particles in the water phase and to sediment, the highest bioaccumulation factors are obtained for zooplankton (*Acartia* sp.) with a high surface/volume ratio, for *Gammarus* sp. and for filtrating molluscs. For these kinds of organisms, DEHP in the colloidal form and DEHP adsorbed to particles can be assumed to be more easily available due to their surface/volume ratio and /or feeding strategy.

The bioaccumulation study with rainbow trout was performed with eggs and larvae. Exposure started 14 days before hatching and the BCF values were determined on larvae 24 days after hatching. The study with carp was a flow-through with deuterium-labelled DEHP. The fish were exposed for 42 days followed by 14 days of depuration. Steady-state conditions were only reached in the lowest test concentrations. The elimination half-life of DEHP, based on total radioactivity, is between 3 and 14 days in the different fish species tested. The major degradation pathway seems to be through hydrolysis of the ester group resulting in the monoester MEHP and subsequent conjugation (EU RA DEHP 2001). As the monoester is reprotoxic, a BCF value of 840 for fish is considered the most likely value for risk assessment purposes in the EU RA DEHP (2001).

Based on the above BCF values, DEHP has the potential to bioaccumulate in the aquatic environment. As the BCF values seem to be lower for fish than for crustaceans and molluscs other food chain effects than through fish seem to be as relevant as the fish food chain. For example whales ingest huge amounts of krill and are long-lived thus the recognised reprotoxic effects on mammals[1] may be of importance. Also a number of birds and other mammal species are known to feed on molluscs and crustaceans.

DEHP is therefore a borderline case regarding bioaccumulation exceeding the OSPAR screening criterion of BCF > 500. However, DEHP does not fulfil the EU B-criterion for fish, but for invertebrates BCF values are observed above 2000 for some species (EAF, 2004). Apparently, DEHP does not biomagnify in foodchain.

Based on the assumption that the DEHP concentration in the prey is solely dependent on water-prey transfer The PEC_{oral} is established in the EU RA DEHP 2001 based on the $PEC_{regional}$ as

Predator eating	Prey	PEC_{oral} (mg/kg)
	Fish	1,6
	Mussel	2
	Zooplankton	2,2

5.3.4 Aquatic toxicity

MICROORGANISMS

No effect on soil respiration or nitrification has been observed at a concentration of 250 mg/kg in soil (Lundberg and Nilsson 1994). In a study by Kirchmann et al. (1991), soil was incubated for 3 months with DEHP at concentrations of 5 and 259 mg/kg. No effects, compared to control, were observed on soil microbial processes (respiration, nitrogen mineralization and nitrification) at any of the test concentrations. A measured NOEC of 250 mg/kg was obtained. Whether this value is related to wet or dry weight is unclear. As a worst case, it should be assumed that it is reported per dry weight.

TOXICITY TO ALGAE

The toxicity studies with DEHP for freshwater and marine algae are summarised in Table 5.11.

[1] In the proposal for the 28. amendment of the Annex 1 of Directive 67/548/EEC DEHP is proposed to be classified Repr. Cat. 2; R60-61.

Table 5.11. Toxicity of DEHP to algae

Test species	(F/S)	N/M	Test duration	Endpoint	EC50 or LC50 (mg/l)	NOEC (LOEC) (mg/l)	Reference
Selenastrum capricornutum	F	M	96 h	Growth inhibition	>0,1	0,1	Adams et al. 1995
Scenedesmus subspicatus	F	M	72 h	Growth inhibition	-	130 (>130)	Hüels 1995
Scenedesmus quadricauda	F	N	7 d	Growth inhibition	-	10	Bringmann and Kühn 1980. Quoted from EU RA DEHP 2001
Gymnodium breve	S	N	96 h	Growth inhibition	30 000	-	Wilson et al. 1995. Quoted from EU RA DEHP 2001

F: Fresh water; S: Salt water; N: Nominal concentration; M: Measured concentration; NOEC: No Observed Effect Concentration; LOEC: Lowest Observed Effect Concentration.

No measured LOEC values are available for algae. The LOEC value of 10 mg/l, reported for *Scenedesmus quadricauda*, and the EC50 value of 30 g/l, reported for the marine algae *Gymnodium breve*, are far above the reported solubility for DEHP. Therefore, the actual effect concentrations are probably lower than the reported nominal values and/or observed effects might be due to physical interference. In the study with *Scenedesmus subspicatus* (Hüels, 1995), MARLOWET R 40 was used as solubilizer. As the solubilizer might have affected the availability of DEHP, the NOEC obtained cannot be used as a basis for PNEC. In the study by Adams et al. (1995), the effect concentration given is based on measured concentration. Unfortunately, no LOEC was obtained.

TOXICITY TO INVERTEBRATES

The short-term toxicity data on DEHP for freshwater and marine invertebrates are presented in Table 5.12 and the long-term toxicity data on DEHP for freshwater and marine invertebrates are presented in Table 5.13. Only studies, in which the effect concentrations are based on actual measured concentrations, are included.

Table 5.13. Short-term toxicity of DEHP to aquatic invertebrates

Test species	F/S	Test duration	Endpoint	EC50 or LC50 (mg/l)	NOEC (LOEC) (mg/l)	Reference (All cited in EU RA DEHP 2001)
Water flea *Daphnia magna*	F	48 h	Survival	>0,113	0,113	Buchen and Vogel 1995
Water flea *D. magna*	F	48 h	Survival	>0,16	0,16	Adams et al. 1995
Water flea *D. magna*	F	48 h	Immobilisation	>0,304	0,304	Brown and Thompson 1982
Mysid shrimp *Mysidopsis bahia*	S	96 h	Survival	>0,37	0,37	Adams et al. 1995
Midge *Paratanytarsus parthenogenetica*	M	48 h	Immobilisation	>0,18	0,18	Adams et al. 1995

F: Fresh water; S: Salt water; N: Nominal concentration; M: Measured concentration; NOEC: No Observed Effect Concentration; LOEC: Lowest Observed Effect Concentration.

No EC50 and/or LOEC values were obtained in any of the reported acute effect studies. In other tests on invertebrates, in which exposure concentrations higher than the solubility in the actual test media have been applied, problems with the formation of micro-droplets or surface films have occurred. This may contribute to effects by direct physical interference that might lead to an overestimation of the toxicity. One example that has attained special attention in this context is entrapment of daphnids at the surface, so-called floaters. Studies, in which such effects have occurred, are not taken into consideration in the determination of PNEC.

Table 5.14. Long-term toxicity of DEHP to aquatic invertebrates

Test species	F/S	N/M	Test Duration	Endpoint	NOEC (LOEC) (mg/l)	Reference
Water flea *Daphnia magna*	F	M	21 d	DNA content, RNA/DNA ratio at day 7 Survival and reproduction	0,072 (0,158) 0,158 (0,811)	Knowles et al. 1987. Quoted from EU RA DEHP 2001
Water flea *D. magna*	F	M	21 d	Survival Reproduction	0,077 (0,16) 0,29 (>0,29)	Rhodes et al. 1995
Water flea *D. magna*	F	M	21 d	Survival and reproduction Growth (7 d)	0,640 (1,300) 1,30 (>1,30)	Adams and Heidolph 1985
Water flea *D. magna*	F	M	21 d	Survival and reproduction	14 (>14)	Scholz 1994 and 1995. Quoted from EU RA DEHP 2001
Mussel *Mytilus edulis*	S	N	28 d	Deposition of faecal material, byssal thread attachment, general appearance, activity and survival	0,05 (>0,05)	Brown and Thompson 1982

F: Fresh water; S: Salt water; N: Nominal concentration; M: Measured concentration; NOEC: No Observed Effect Concentration; LOEC: Lowest Observed Effect Concentration.

In the study by Knowles et al. (1987), daphnids were trapped at the surface (dose and time dependent). As they grew, the number at the surface decreased. By day 21 surfacing behaviour was observed only in daphnids exposed to 811 μg/l. The study with *Mytilus edulis* comprises a bioconcentration study with ^{14}C-labelled DEHP. No adverse effect at the highest tested concentration (0,05 mg/l) was observed.

In the chronic toxicity tests, the lowest NOEC determined for *Daphnia magna* is 0,077 mg/l (LOEC 0,16 mg/l) based on survival and 0,072 mg/l (LOEC 0,158 mg/l) based on RNA/DNA ratio (dispersant not added). There are several indications that the effects observed in the toxicity tests with *Daphnia* could be caused by physical effects, which probably do not have any relevance in the environment. It appears highly uncertain whether floaters were actually present in all test concentrations showing effects. If present, it is also unclear if the floating (or other physical interference) is the actual cause of the effects reported. Based on the present data, it is considered not feasible to determine a level of toxicity for DEHP to aquatic invertebrates exposed via water. Hence, it is not possible to state a $NOEC_{water}$ for aquatic invertebrates.

TOXICITY TO FISH

DEHP showed no toxicity at the apparent solubility limit in numerous well-performed acute toxicity tests with different fish species (e.g., bluegill sunfish, fathead minnow, carp, rainbow trout, zebra fish and guppy). The LC50 values determined after 96 h of exposure were in the range of >0,16 - >10 mg/l (EU RA DEHP 2001). No toxicity was seen and a determination of LOEC values was not possible. Effect concentrations between 6 and >100 mg/l have also been reported. In these tests different vehicles were used. As all the effect concentrations reported in these studies are far above the solubility limit of DEHP, they are not referred below.

Table 5.15. Short-term toxicity data on DEHP for fish

Test species	(F/S)	N/M	Test Duration	Endpoint	EC50 or LC50 (mg/l)	Reference
Bluegill sunfish *Lepomis macrochirus*	F	M	96 h	Mortality	>0,20	Adams et al. 1995
Fathead minnow *Pimephales promelas*	F	M	96 h	Mortality	>0,16	Adams et al. 1995
Sheepshead minnow *Cyprinodon variegatus*	F	M	96 h	Mortality	>0,17	Adams et al. 1995
Rainbow trout *Salmo gairdneri*	F	?	96 h	Mortality	>10	Mayer and Sanders 1973
Zebra fish *Brachydanio rerio*	F	?	96 h	Mortality	>0,32	Canton et al. 1984. Quoted from EU RA DEHP 2001
Guppy *Poecilia reticulata*	F	?	96 h	Mortality	>0,32	Adema et al. 1981. Quoted from EU RA DEHP 2001

F: Fresh water; S: Salt water; N: Nominal concentration; M: Measured concentration; NOEC: No Observed Effect Concentration; LOEC: Lowest Observed Effect Concentration.

From the acute effect studies available, it can be concluded that DEHP has no acute effect at exposure levels far exceeding its apparent water solubility.

The results from long-term studies performed on different fish species are given in Table 5.16. A few long-term studies, in which fish have been exposed to DEHP via the diet, have been performed. These studies are also included in the table below.

Table 5.16. Long-term toxicity data on DEHP for fish

Test species	(F/S)	N/M	Test Duration	Endpoint	NOEC (LOEC) (mg/l)	Reference
Brook trout *Salvelinus Fontinalis* (adult 1,5 year)	F	N	150 d	Reduced vertebral collagen levels, increased hydroxyproline levels in collagen	0,0037 (<0,0037)	Mayer et al. 1977
Rainbow trout *Salmo Gairdneri* (embryo, eyed egg)	F	N	90 d	Reduced vertebral collagen levels	0,014 (0,005)	Mayer et al. 1977
Fathead minnow *Pimephales promelas* (juvenile 10 d)	F	N	127 d	Reduced vertebral collagen levels increased hydroxyproline levels in collagen	0,011 (<0,011)	Mayer et al. 1977
Rainbow trout *S. gairdneri* (Embryo-larval)	F	M	102 d (12+90)	Hatchability Mortality 5 d post hatch	>0,05 0,005 (0,014)	Merhle and Mayer 1976
Rainbow trout *Oncorhynchus mykiss* (Embryo-larval)	F	Flow	90 d	Hatchability, survival and growth	>0,502	DeFoe et al. 1990
Rainbow trout *O. mykiss* (Embryo-larval)	F	Flow M	70 d	Hatchability, survival and growth	>0,0073	Cohle and Stratton 1992 (quoted from EU RA DEHP 2001)
Stickle Back *Gasterosteus aculeatus*	F	N	35 d	Mortality, egg development and growth	>0,32	Van den Dikkenberg 1989 (quoted from EU RA DEHP 2001)
Zebra fish *Brachydanio rerio*	F	Food	90 d	Reproduction rate and fry survival	>50 (50)	Mayer and Sanders 1973
Cod *Gadus morhua*	S	Food	121 d	Steroid metabolism	10 (100)	Freeman et al. 1981 (quoted from EU RA DEHP 2001)
Atlantic salmon Salmo salar	F	Food	5 month	Sex ratio and liver somatic index	300 (1500)	Norrgren et al. 1990
Fathead minnow Pimephales promelas	F	M + Food	472 d	F0; F1 and F2 generation:Egg production, egg hatch, survival, growth (length and weight) VTG	0,003 <0,003 (0,003)	Caunter et al. 2004
Japanese medaka *Oryzias latipes*	F	N	14 d followed by reproduction in clean water	Egg number and hatching rate	0,391	Shioda and Wakabayashi, 2000

Test species	(F/S)	N/M	Test Duration	Endpoint	NOEC (LOEC) (mg/l)	Reference
Japanese medaka *Oryzias latipes*	F	N	3 month	Females: VTG (decrease) GSI maturation	<0,001 (0,001) 0,001 (0,01) <0,001 (0,001)	Kim et al., 2002
Japanese medaka *Oryzias latipes*	F	N	5 month	Hatching time Mortality Body weight (males) Sex ratio (no dose/response) GSI (females)	0,00001 (0,0001) < 0,00001 (0,00001) 0,0001 (0,00001) < 0,01 (0,01) 10	Chikae et al., 2004

F: Fresh water; S: Salt water; N: Nominal concentration; M: Measured concentration; NOEC: No Observed Effect Concentration; LOEC: Lowest Observed Effect Concentration.

No significant mortality was seen in the long-term toxicity studies with juvenile and adult fish. There are, however, indications that DEHP may have effects on growth at relatively high exposure concentrations. The slightly impaired growth in these studies may be an effect of physical influence from the test substance as the test concentrations were well above the true water solubility. Mayer et al. (1977) observed effects on collagen synthesis at exposure levels as low as 0,004 mg/l. However, no effects on growth were seen in this study and the biological and ecological significance of the effects on collagen synthesis is unknown. Mehrle and Mayer (1976) used rainbow trout eggs for studying the effects of DEHP. In this test, acetone was used as a carrier solvent at concentrations not exceeding 0,28 ml/l. There was no increase in egg mortality or any effects on hatchability in the DEHP exposed groups compared to control. There was, however, an increase in the mortality of sac fry within 5 days after hatching in the two highest concentrations. An increased mortality compared to the control group was also observed at 24 days after hatch. The dose response relationship in this study was weak and the acetone concentration was higher than accepted by OECD-guideline 210 but within the range accepted by OSPAR. Furthermore, the NOEC obtained in this study was far below that in other studies with rainbow trout (Defoe *et. al* (1990), Mayer *et. al* (1977), Birge *et. al* ((1979) In: EU RAR 2001). The results from the test are thus not considered reliable..

There are studies showing effects of DEHP when fish are exposed via the food. In the test performed by Mayer and Sanders (1991), zebra fish were via the diet exposed to DEHP at the concentrations of 50 and 100 mg/kg. The test is considered invalid due to 49% mortality in the control group. In the test performed on cod (*Gadus morhua)* (Freeman et al. 1981. Quoted from EU RA DEHP 2001), no significant differences in steroid metabolic profiles in male fish at highest dose (1000 mg DEHP/kg food) compared to control were obtained. In female fish, there was a significant alteration of steroid biosynthetic pathways in the head kidneys and ovaries of the DEHP fed fish. The ratios of 11-deoxycortisol from 100 and 1000 mg/kg groups were greater than twice the observed ratios obtained from the control and 10 mg/kg.

The studies performed by Norrgren et al., 1990; Caunter et al. 2004; Shioda & Wakabayashi, 2000; Kim et al., 2002 and Chikae et al., 2004 are discussed under the issue “Endocrine disruption” as the endpoints evaluated in these studies are specific related to endocrine effects.

TOXICITY TO SEDIMENT LIVING ORGANISMS

The available studies with sediment-dwelling organisms exposed to DEHP show largely varying results. Short-term and long-term tests with *Chironomus spp*. larvae did not result in any effect at the highest concentration tested, 3000 and 11000 mg/kg (dwt,) respectively (EU RA DEHP 2001). For amphibians a

$NOEC_{sediment}$ > 1000 mg/kg (dwt) was obtained. In the Risk Assessment Report DEHP the NOEC of > 1000 mg/lg derived from frog studies is chosen for the derivation of a $PNEC_{sediment.}$ Effect studies exist with organisms from three trophic levels. Therefore, an assessment factor of 10 is used, resulting in a PNEC of >100 mg/kg (dwt) for the freshwater compartment. For the marine environment, as three long term tests with species representing different living and feeding are available but not on marine species, we propose to apply an assessment factor of 50 as recommended by the TGD on the NOEC amphibians of > 1000 mg/kg (dwt). This would result in a $PNEC_{marine\ sediment}$ of > 20 mg/kg (dwt). Any such PNECsed derived should only be used indicatively.

In conclusion, no adverse effects have been observed for DEHP on sediment organisms.

ENDOCRINE DISRUPTION

The specific endocrine activity (estrogenic, anti-androgenic, anti-estrogenic) of DEHP is not clear. It has been reported that DEHP had anti-androgenic activity in male rats (Gray et al., 2000), anti-estrogenic activity in female medaka after 3 months of exposure (decreased VTG, decreased Gonado Somatic Index (GSI) and decrease in oocyte maturation rate). The observed effects were most pronounced at the lowest exposure concentrations (Kim et al., 2002). No effect on reproduction was seen in male medaka exposed for 2 weeks to 39-390 μg/l and then allowed to breed with unexposed females (Shioda & Wakabayashi, 2000). Chikae *et al*. (2004a) studied the effects of DEHP on embryos of Japanese medaka (*Oryzias Latipes*). Newly fertilised eggs were incubated in a semistatic system and exposed to the nominal concentrations 0; 0,01; 0,1; 1,0 and 10,0 µg DEHP/l (40 eggs/group) until hatching. Ethanol was used as solvent (< 100 µg/l). Eyeing, hatching time and hatching success were studied. After hatch, the fry (n = 25 – 43) were transferred to a post-hatch solution for growth for 5-6 months. Mortality and body weight were recorded as well as sex ratio and gonadosomatic index for the surviving fish. No effects were seen on eyeing or hatching success. Significantly delayed hatching were observed at 0,1 and 1,0 µg DEHP/l, but not at 10,0 µg DEHP/l. Mortality was significantly higher in the 0,01; 0,1 and 1,0 µg DEHP/l groups, although not in the 10,0 µg DEHP/l group. Sex ratio was significantly changed towards females at 0,01 µg DEHP/l. Whether this was caused by feminisation or by male specific lethality could not be determined. Body weights of males decreased in a dose dependent manner and were significantly lower (≤ 25 %) at the three highest concentrations, 0,1; 1 and 10 µg DEHP/l respectively. Bodyweights of exposed females were not significantly different from the control. No significant effects on gonadosomatic index neither for male nor female medakas could be seen. The only dose-related effects that could be seen in this study were the decreased body weight in males. However, dose-related effects on body weights have not been observed in any other studies on DEHP. The interpretation of the study is hampered by the high and variable mortality. Overall, this study is not considered sufficiently robust for use in the risk characterisation.

In another study by Chikae *et al*. (2004b), the effects of DEHP on the fry stage of medaka were studied. The fry were exposed in a semi-static system to the nominal concentrations 0; 0,01; 0,1; 1 and 10 µg DEHP/l (n = 20/group) for three weeks after hatching, starting on day one post-hatch. Ethanol was used as solvent. After three weeks of exposure, fish were transferred to a balanced salt solution for growth. After five months, mortality, body weight, sex ratio and gonadosomatic index (GSI) were measured in the adult fish. The result showed significantly lower body weights in males at 0,01 and 10 µg DEHP/l and in females at 0,1; 1 and 10 µg DEHP/l, although not dose-dependent. A significantly decreased GSI, however not dose-dependent, was observed in males only, and at the concentrations 0,01; 1 and 10 µg DEHP/l. An increase in mortality was observed at 0,1 µg/l and higher, although not statistically significant. No effects on sex ratios could be seen. The results of this study do not demonstrate any dose-related effects. The effects still indicated, i.e. significantly lower GSI, are in conflict with the results in the previous study conducted by the same authors using identical concentrations and species, although at different developmental stages.

Norrgren et al. (1999) studied the effects on sexual differentiation in Atlantic salmon *(Salmo salar)* exposed to DEHP via the food. The exposure concentrations were 300 and 1500 mg/kg food (dry weight). At the highest exposure concentration (1500 mg/kg), significant effects on the sex ratio (increased number of females) were obtained. The food conversion rate from the diet used in the study is 1 g wet weight increase/g food. The conversion rate of natural food is only 0,2 g wet weight increase/g food or even lower (L. Norrgren, pers. comm. 2005). In order to obtain the same weight increase fish fed on natural diets are thus expected to eat 5 times the amount of food compared to fish fed on commercial diets. Thus, NOEC for natural diets then becomes 60 mg DEHP/kg food (wwt) instead of 300 mg/kg. In a study by Norman *et al* (manuscript), which is a follow up study to the study by Norrgren (1999), Atlantic salmon were fed DEHP contaminated food with nominal concentrations of 0, 400, 800 and 1500 mg DEHP/kg food (dwt). The mean measured concentrations, based on measurements at the start and the end of the exposure period, were 358, 827 and 1648 mg/kg for the three exposure levels, respectively. Feeding with DEHP contaminated food was initiated at the end of the yolk sac stage and continued for 4 weeks, as in the first study. Thereafter the fish were fed uncontaminated food. Each exposure group consisted of approximately 1000 individuals.

Sampling was performed after 4 and 9 month. In this study no effects on the sex ratio were observed. However, a 6% incidence of ovotestis in males, which was statistically significant compared to the control were no ovotestis was observed, occurred in the highest dose group (1500 mg/kg dwt) after 4 month. After 9 month an incidence of ovotestis of 1% (not statistically significant) was observed. Also at 800 mg/kg ovotestis was observed both after 4 month and 9 months. However, the incidence was not statistically different from the control group. The findings indicate that the effects obtained are reversible as statistically significant effects were seen after 4 months but not after 9 months. The effects seen in the presents study was weaker (ovotestis) compared to the effects seen in the earlier study by Norrgren *et al* (shift in sex ratio). No analytical confirmation of the exposure concentrations was made in the earlier study and the authors speculates that the actual exposure in that study may have been higher thus explaining the difference in response between the two studies. Based on the results from both studies it is concluded that the NOEC for effects on sexual differentiation of Atlantic salmon is 800 mg/kg food (160 mg/kg food for natural diet).

A multi-generation study with fathead minnow *(Pimephales promelas)* has recently been finalised. The study was performed in 2001-2003 by Brixham Environmental Laboratory, AstraZeneca, UK, and sponsored by the European Council for Plasticisers and Intermediates (ECPI). The test guideline used was adapted from US EPA, Fish Life-Cycle Toxicity Test, incorporating biological endpoints (vitellogenin and gonad histopathology).

DEHP was dosed simultaneously in the diet (125 and 500 mg/kg dry weight) and in the water (5 μg/l). Two replicates of each concentration were applied. Tri-ethylene glycol (2,5 μl/l) was used as solvent. The study encompassed three generations and included measurements of survival, weight, length, reproduction, vitellogenin (VTG) and gonad histopathology. Adult F0 and F1 fish were analysed for their content of DEHP and its metabolite MEHP.

The evaluated endpoints were processed using different statistical methods with the following general principles: If no significant difference between the dilution water control and the solvent control was found, these groups were pooled for subsequent analysis. If differences were found, the control group without solvent was excluded from the subsequent analysis.

Although a number of statistically significant decreases in the weight, length and survival of fish were observed in both High Dose Food (HDF) exposures and Low Dose Food (LDF) exposures, these decreases do not seem to be related to DEHP exposure. The majority of the decreases were observed in the LDF group.

A significant increase in VTG in the F2 generation (females) was observed in the HDF group when compared to the pooled control groups (solvent control + dilution water control). However, when compared to the solvent control, no significant increase could be observed. In the conclusions of the report, it is argued that for this specific endpoint (VTG), it is more relevant to compare with the solvent control. In a recently performed international ring test (OECD 2004) on a draft guideline for detection of endocrine active substances, all data (including VTG measurements) were evaluated using the approach: If no significant difference between the dilution water control and the solvent control was found, these groups were pooled for subsequent analysis. The statistical evaluation of the control groups in the present study showed that there was no significant difference between the dilution water control and the solvent control, i.e. the control groups should be pooled and compared with the exposure group as for all other endpoints. Thus, based on the reported results, it must be concluded that the multigeneration study showed a significant increase in VTG in the females from the F2 generation.

The exposure of F2 fish to the HDF treatment showed an increase in the number of males with moderate spermatogenesis when the numbers of individuals were combined*. This may indicate an anti-estrogenic effect*. No other apparent effect on gonadal development or histopathological endpoints was observed. The histopathological endpoints and sex ratios are only reported for the F2 generation but not for the F0 and F1 generation. According to the Appendices of the report it is clear that approx. 25-30 fish were sampled on day 100 post hatch from 3 generations (F0; F1 and F2). All fish were sampled for potential vitellogenin and histopathology analysis. It is stated that inconsistencies between replicates meant that statistical tests could not be meaningfully applied to the ratio of males to females. On day 100 post hatch, Fathead minnow have reached sexual maturity and it therefore seems striking that there were inconsistencies between replicates for each generation, which meant that statistical test could not be applied to determine the ratio of males to females. Furthermore, secondary sex characteristics are pronounced in fathead minnows and therefore it should have been possible to determine and report the sex ratios of the different groups and replicates for all generations (F0; F1 and F2). Taking the duration and effort of the study into consideration it also seems strange that only the F2 generation was thoroughly evaluated, e.g. no VTG measurements and histopathology were performed on either the F0 or the F1 generations.

Based on measurements of the body burdens of DEHP and its main metabolite MEHP and the corresponding BCF value (553), it is concluded that the main route of uptake of DEHP is via the water phase. The BMF values are very low. The MEHP:DEHP ratio was low (1:17) indicating a rapid loss of the metabolite from the fish.

As stated in the above discussion the overall conclusion given in the report that the effects observed were not related to exposure to DEHP when dosed in combination in food and water, is questionable, as a significant effect on VTG in the high dose group was obviously observed. All together the study or at least the report seems to have several shortcomings:

- In the summary of the report the effect on hatchability on all 3 generations (F0; F1 and F2) is shown in a graph. Such graphs are missing for all other endpoints;
- The effect of DEHP on survival of the F0 generation is not shown either in tables or figures;
- Results from the VTG measurements are only given for F2 generation although it according to the Appendix 2 of the report is clear that approx. 25-30 fish were sampled on day 100 post hatch from all 3 generations (F0; F1 and F2). All fish were sampled for potential vitellogenin and histopathology;
- Results from the histopathology is only given for the F2 generation;
- In the conclusions of the report the results from the VTG measurements (HDF) are compared with the solvent control and not the pooled control (solvent control and dilution water control) although no significant difference between the dilution water control and the solvent control was obtained. This conflicts with the statistical evaluation used for all other endpoints in the study and the statistical approach used in a recently performed international ring test for the detection of endocrine disrupting effects (OECD 2004);
- In the report p. 28 it is stated that "Within studies that use VTG concentration as an endpoint there is often evidence of an effect within the solvent control" (with reference to Panter et al. (2001) and Harris et al. (2001)). In none of these articles this conclusion is drawn and no increase in VTG levels in the solvent control compared to the dilution water control is shown (Panter et al., 2002). In Harris et al (2001) no dilution water control is included, furthermore, this article is not cited correctly in the reference list.

In a study by Metcalfe et al (2001), the estrogenic properties of DEHP were assessed *in vivo*, in Japanese medaka, and *in vitro* in a yeast estrogen screening (YES) assay. In the in vivo study, fry of medaka were exposed from one day post-hatch (dph) until approximately 90 dph in a semistatic system to the nominal concentrations 0, 500, 1000 and 5000 µg DEHP/l (n = 60 – 90/group). Acetone was used as solvent. After sampling, sex ratio, presence of intersex as well as morphometric parameters was studied. In the YES assay, concentrations of DEHP ranging between 50 µg/l – 100 mg/l were tested. No effects on sex ratios, incidence of intersex or morphometric parameters could be observed in the medaka and no estrogenic activity could be detected in the YES assay. The study is considered reliable. However, concentrations above the water solubility of DEHP were used for all groups, indicating that they could have been exposed to the same concentration, i.e. the water solubility limit of DEHP.

Shioda and Wakabashi (2000) studied the reproductive effects of DEHP in a semistatic test on male medakas. Adult males were exposed to the nominal concentrations 39, 120 and 390 µg DEHP/l (n = 3/group) for two weeks. Acetone was used as solvent (< 100 µg/l). Each male were then transferred to dechlorinated tap water and allowed to spawn with two females for two weeks. The number of eggs as well as the hatching rate was examined. No effects of DEHP on the number of eggs or hatchings could be observed. This study is considered to be of poor quality and unreliable.

From the studies performed for indication of endocrine effects it seems that the effects seen from DEHP exposure are not always dose-response related. Especially the studies performed by Kim et al. (2002) and Chikae et al. (2004) indicate that DEHP might have a low dose effect.

TOXICITY FOR BIRDS AND MAMMALS (SECONDARY POISONING)

Please refer to EU RA DEHP 2001 for details. A NOEC for birds has been established to 1700 mg/kg food on the basis of a 28 d repeated dose study. For mammals a LOAEL has been established to (<) 5 ppm. At this dosage irreversible testicular damages were seen on rat male pups. This value was supported by another study showing a NOAEL of 50 ppm also with rat testis development as endpoint.

SUMMARY - EFFECT

The very low water solubility of DEHP causes problems when testing toxicity to aquatic organisms and when interpreting the results. Most aquatic studies with DEHP have been at test levels exceeding the 'molecular' solubility of approx. 3 µg/l. Formation of micro-droplets or surface films may also contribute to effects by direct physical interference, e.g. entrapment at the surface (floating) or obstruction of the gas flow over the gills (Pedersen & Larsen 1996). DEHP shows no acute toxicity to algae, crustaceans or fish. From the recent

studies performed (Kim et al., 2002 and Chikae et al., 2004) it seems that DEHP might have a low-dose effect as effects (increased mortality and skewed sex-ratio) were determined at concentrations (0,01 μg/l) well below the water solubility limit. In a multi-generation study with fathead minnow an increased VTG concentration was observed in females (F2 generation) exposed to 3 μg/l and 474 mg/kg wwt (measured concentrations) simultaneously. In this study the main uptake route was found to be via water. In a long-term study, in which Atlantic salmon were exposed via the food, a NOEC value of 800 mg DEHP/kg artificial food wwt was determined corresponding to 160 mg DEHP kg/ natural food.
Only few tests have been conducted with sediment-dwelling species and only with freshwater species (amphibians and insects). In a frog study a NOEC of > 1000 mg/kg was derived.

In different *in-vitro* and *in-vivo* studies with mammals and fish, DEHP has been shown to have endocrine disrupting effects. The specific endocrine activity (estrogenic, anti-estrogenic or anti-androgenic) is not yet clear. It has also been shown that DEHP might have effects on the population level by altering the sex ratio at concentrations of 0,01 μg/l. Increased VTG levels in females have been determined in fathead minnow exposed to 3 μg/l DEHP. This indicates that DEHP might have a potential endocrine disrupting effect in aquatic species at realistic exposure concentrations.

For secondary poisoning a mammalian LOAEL of 50 ppm showing irreversible testicular damages on rats can be used.

In conclusion, the RAR concludes from the available reliable studies that DEHP has no acute or chronic effects on aquatic species at or below the water solubility of the substance. However DEHP might have a potential of causing endocrine disrupting effects and is classified as reprotoxic in many mammalian species with testes being the target organ. The classification for human health is: toxic to reproduction (category 2, R60-61), thus DEHP fulfils the T-criterion (EAF, 2004).

5.3.5 PNEC for the aquatic environment

From the acute effect studies available, it can be concluded that DEHP has no acute effect at exposure levels far exceeding its apparent water solubility. Indications on long-term effects (increased mortality and skewed sex ratio) have been detected at low concentrations (0,01 μg/l). This study, however, need to be further validated before used for risk assessment. In a multigeneration study with fathead minnow a LOEC value of 3 μg/l was determined for VTG in females. Applying an assessment factor for example of 10^2 leads to a $PNEC_{aquatic}$ of 0,3 μg/l. However, as the relation between observed effect and effects in the environment are questionable, such PNEC should only be used indicatively as supporting evidence. There are studies available showing effects of DEHP when fish are exposed via the food (NOEC 160 mg/kg wwt natural food). This NOEC value will be used for deriving a $PNEC_{food.}$ Applying an assessment factor of 10 leads to a $PNEC_{food}$ of 16 mg/kg wwt. This value is in agreement with the $PNEC_{food}$ value proposed by the EU Risk Assessment on DEHP (2001). If we consider that the food of fish is primarily composed of zooplankton the PNECfood of 16 mg/kg wwt might be recalculated to a concentration in water using the BCF crustacean of 2700 wwt. This leads to a tentative $PNEC_{aquatic}$ of 6 µg/L.

No adverse effects have been observed on sediment organisms. An indicative $PNEC_{sediment(marine)}$ > 20 mg/kg dw can be derived based on the lowest NOEC at >1000 mg/kg dw observed in sediment tests and applying an assessment factor of 50.

For secondary poisoning of birds and mammals the $PNEC_{bird}$ is established to 17 ppm on the basis of a 28 d repeated dose NOEC of 1700 ppm and with the use of an assessment factor of 100. The $PNEC_{oral,\ mammal}$ is established to (<) 5 ppm on the basis of LOAEL showing irreversible testicular damages on rat male pups and using an assessment factor of 10. This value was supported by another study showing a NOAEL of 50 ppm.

5.4 PBT assessment and Risk assessment

In conclusion, DEHP is assessed as not fulfilling the EU or OSPAR P-criterion. The OSPAR screening B-criterion is fulfilled. The EU B-criterion is not fulfilled for fish, whereas, for invertebrates DEHP is a borderline case. No biomagnification is expected. Due to the reprotoxicity of DEHP for mammalian species, the T-criterion is fulfilled. Overall DEHP is considered not to meet the PBT criteria in the marine risk assessment.

However, due to the large quantities used annually ant the use pattern in many articles with long service life, large amounts of DEHP are diffusely spread in the environment. DEHP is therefore found in all

[2] No guidance is available on which assessment factor to apply for such effects.

environmental compartments, also in remote areas. Consequently all organisms including man are exposed to DEHP during their whole life-time (EAF, 2004).

DEHP has been measured in concentrations in water in the range of 0,058 – 1,9 µg/l (estuaries and fjords) and 0,0001-0,375 µg/l (open sea and ocean). Compared to the indicative PNEC of 6 µg/l based on the Norrgren *et al.*- studies no general risk is expected for the marine environment. This conclusion is in general supported by comparison of most measured concentrations with an indicative PNEC based on VTG levels in fathead minnows.

Concentrations of DEHP in sediments have been measured in the range of 0,0112-16,0 mg/kg dw in sediment (estuaries and fjords) and 0,007-15,21 mg/kg dw (open sea and ocean). Higher concentrations have been measured in suspended matter. No adverse effects have been observed on sediment organisms. Using an indicative $PNEC_{sediment(marine)}$ of > 20 mg/kg dwt no risk is expected. In conclusion, risks to sediment organisms are not expected at environmental concentrations.

Concentrations in biota are available for planktonic algae (63 mg/kg), invertebrates (0,1-14 mg/kg ww) and fish (<0,1-19 mg/kg). The highest values measured are above the $PNEC_{food}$ indicating a potential for ecotoxic food chain effects. However, the highest measured concentrations in biota are fairly old and should be used with some reservation.

Potential concentrations in biota can be estimated from the measured concentrations of DEHP in marine waters and the BCF values. Using a realistic concentration of 0,1 µg/l and the maximum BCF value for algae (53 890) and the recommended value of 2700 for zooplankton (EU RA DEHP 2001) result in estimated concentrations of up to 5,4 mg/kg and 3 mg/kg, respectively. These values are in the same order of magnitude as the measured values and support the above conclusion.

Regarding the risk for secondary poisoning for birds and mammals, based on the $PEC_{regional}$ and water-prey transfer only, risk quotients (PEC/PNEC ratio) are almost all below 1. DEHP do not seem to biomagnify in the foodchain. Furthermore, it should also be noted that extrapolation from rat to whales or seal are dubious and that the assessment factor used (AF=10) are small compared to what is acceptable if extrapolation was done from rat to humans with a similar exposure route.

Furthermore, there is a potential risk of endocrine disrupting effects in aquatic organisms. Finally, the bioconcentration potential may result in relatively high exposure of certain marine birds and mammals that combined with the reprotoxic effects and potential for endocrine disrupting effects may lead to the general conclusion that DEHP could exhibit a risk for the marine environment.

5.5 Desired Reduction and Identification of possible measures

Overall DEHP is considered not meeting the PBT criteria in the marine risk assessment.

However, due to the large quantities used annually ant the use pattern in many articles with long service life, large amounts of DEHP are diffusely spread in the environment. DEHP is therefore found in all environmental compartments, also in remote areas.

No risk is apparently foreseen for organisms living in the marine environment (water column and sediment) at environmental concentrations, particularly in open marine waters. However, there might be potential endocrine disrupting effects. It is therefore suggested that any potential risk of DEHP in this regards that are not covered in the present document should be assessed in the context of a general approach towards endocrine disrupting substances.

6. Di(isononyl)phthalate

6.1 Production, use and emission

6.1.1 Production

Five main producers or importers of di(isononyl)phthalate (DINP) are listed in the EU RA DINP (2003). One of the producers stopped production in 1995. There are three different DINPs (CAS 68515-48-0, CAS 28553-12-0, CAS 28552-12-0), which are manufactured by different processes and may have different physico-chemical and toxicological properties. However, based on available data, no distinction between the different types was possible.

[1] Based on the assumption that 90% of total phthalates is DEHP.

The total DINP production in the EU was 185 200 t/year in 1994. The import volume was estimated as 5 400 t/year and approx. 83 400 t/year were exported outside the EU. Estimations performed by the producers show an increase in the DINP consumption in Western Europe during the last decade from 70 000 t/year in 1980 to 107 000 t/year in 1994 (EU RA DINP 2003).

Almost all the DINP consumed in the EU is transported by road tankers or by ship. Approx. 15% corresponding to 16 050 t/year are transported by ship. The remaining 85% of the substance (90 950 t/year) are transported by road.

6.1.2 Use

The use of the consumed DINP is distributed over the following applications:

- 95% in PVC;
- Approx. 2,5% in non-PVC involving polymer-related use (e.g. rubbers);
- Approx. 2,5% in non-PVC and non-polymers including inks, pigments, adhesives, sealants, paints, lacquers and lubricants.

No precise quantitative assignment was available for the non-PVC use categories. Therefore, an even distribution among the three main categories of non-PVC non-polymer related use was assumed (EU RA DINP 2003). The estimated amounts of DINP used in the various applications are given in Table 6.1.

Table 6.1. Estimated quantitative usage distribution of DINP for different applications (EU RA DINP 2003)

Use category		**Amount use of DINP within the EU in 1994 (t/year)**
PVC end-uses	*Calendering:* Film, sheet and coated products Flooring, roofing, wall covering	 15 936 3 552
	Extrusion: Hose and profile Wire and cable Clear, medical, film	 5 379 29 020 7 125
	Injection moulding: Footwear and miscellaneous	 8 313
	Platisol spread coating: Flooring General (coated fabric, wall covering, etc.)	 10 658 11 571
	Other plastisol applications: Car undercoating and sealants Slush/rotational moulding etc.	 7 714 1 929
Non-PVC end-uses		
• Polymer related		2 750
• Non-polymer related	Adhesives, glues and sealing compounds Inks Paints	915 915 915
Total		107 000

No information is available on the quantitative use of non-PVC end-products containing DINP. In the EU RA DINP (2003), the PVC end-products were split up in in-door and out-door use and the volumes of DINP in applications were estimated for the different applications (Table 6.2).

Table 6.2. PVC products containing DINP divided into in-door and out-door applications based on data from the industries (EU RA DINP 2003)

Application		DINP (t/year)
In-door	Coated product	[1]
	Film & sheet	[1]
	Wires & cables	14 510
	Hoses &profiles	[1]
	Floor	10 658
Our-door	Roofing material	230[2]
	Roofing (coil coating)	1 150[2]
	Wires & cables	14 510
	Coated fabric	4 850[2]
	Hoses & profiles	1 380[2]
	Car under-coating	7 714
	Shoe soles	8 313

1) No data
2) Estimated from DEHP, based on marked shares

6.1.3 Emissions from diffuse sources

DINP is emitted from following diffuse sources (EU RA DINP 2003):

1. Distribution of DINP within the EU by road transport and shipping of the substance;
2. Exterior and interior use of DINP-containing PVC products;
3. Use of non-PVC polymers containing DINP;
4. Applied adhesives, glues and sealing containing DINP;
5. Paper recycling;
6. Applied paints containing DINP;
7. Disposal of end-products (waste).

The calculation of the total emission from distribution of DINP was based on estimated losses from transportation by road and ship, respectively. A loss of 1 kg DINP per 20 tonnes was estimated for transportation by road and an estimated loss close to 0,3% was found for the transportation by ship. The losses come from cleaning of tanks, lines and pumps etc.

The emission from the above listed steps 1-8 of the life cycles was primarily estimated from experimental studies and assumed technical lives of the products (EU RA DINP 2003). A summary of the total releases from the different diffuse sources is given in Table 6.3.

Table 6.3. Total releases to surface water and wastewater

Life cycle step	Total emission of DINP in the EU	
	Surface water (t/year)	Waste water (t/year)
Distribution of DINP within the EU by road transport and shipping of the substance		79,2
Exterior and interior use of DINP-containing PVC products	179,6	212,7
Use of non-PVC polymers containing DINP	4,87	5,8
Applied adhesives, glues and sealing containing DINP	4,57	
Paper recycling		5
Applied paints containing DINP	16,1	
Disposal of end-products (waste)	710	
Total	915	302

SOURCE INVESTIGATION

A summary of the measured DINP concentrations and the estimated release to waste water reported by Vikelsøe et al. (1998) is given Table 6.4.

Table 6.4. Concentration intervals of DINP in wastewater samples from diffuse sources in Denmark (Vikelsøe et al. 1998)

Diffuse sources		Car wash 7 samples	Hospital 12 samples	Laundry 2 samples	Kindergarten 1 sample
DINP concentration in waste water	(µg/l)	88-510	<50	<50	<20 000

The release in the EU due to car washing was estimated as described for DBP and using the average release per wash of 38 mg found in the investigation of Vikelsøe et al. (1998). An amount of 109 t/year is released to the surface water according to EU RA DINP (2003). However, the calculation of this estimate was based on the release to waste water found by Vikelsøe et al. (1998). This value should thus be used as an estimate for the release to waste water. Calculation of the DINP release from car undercoating to surface water made 55 t/year using the method described in EU RA DEHP (2001).

Analysis of DINP was not included in the Danish studies of phthalates release from textile wash (Hoffman 1996; Larsen et al. 2000). It was assumed that, within the EU, the DINP release due to textiles wash corresponds proportionately to the release of DEHP. The yearly release of DINP was thus estimated on the basis of the consumption of ink [(915 t/year of DINP/1 640 t/year of DEHP) · 46 t/year of DEHP released] = 26 t/year (see Section 6.2.2).

EFFLUENT FROM MUNICIPAL WASTEWATER TREATMENT PLANTS

Monitoring data only exist from three municipal Danish WWTPs. The DINP concentrations in the effluent were <0,05-0,08 µg/l (Grüttner et al. 1995).

Assuming an average DINP concentration in the WWTP effluents of 0,06 µg/l, the total amount of DINP discharged from the WWTPs into the aquatic environment will be 3,5 t/year in all of the EU (Section 3.1.3).

STORM WATER

The Danish investigations of phthalates in storm water 1995-1996 did not include analysis of DINP (Danish EPA 1997). The amount of DINP discharged from storm water was assumed to correspond proportionately to the use of DINP and DEHP [(107 000/476 000) · 32 µg/l DEHP/l] = 7,2 µg/l.

	Denmark	EU
• Mean DINP concentrations in storm water:	7,2 µg/l	
• DINP emission to WWTPs:	0,72 t/year	52 t/year
• DINP emitted directly into the aquatic recipient:	1,08 t/year	78 t/year

ESTIMATION OF THE DINP DISCHARGE FROM RIVER WATER INTO THE MARINE ENVIRONMENT

In the EU, the discharge of DINP from rivers into the marine environment was estimated by use of the assumptions listed for DBP (Section 3.1.3).

Only two measured results for DINP at approx. 1 µg/l are available. According to the assumptions given for DBP, the DINP discharge from rivers in the EU will be approx. 710 t/year. Based on an estimated PECregional at 0,70[1] µg/l (EU RA DINP 2003), a discharge of 497 t/year is estimated. Estimates based on river concentrations (monitored or modelled) do not take direct discharge from industry or settlements situated in coastal regions into account or discharges of DINP adsorbed to particles if based on the calculated $PEC_{regional}$. The discharge from diffuse sources accounts for approx. 64% of the total discharge of DINP (EU RA DINP 2003).

SUMMARY

A summary of the estimated release of DINP from the different diffuse sources above is given in Table 6.5.

Table 6.5. Total diffuse emission of DINP within the EU according to the EU RA DINP (2003) and the estimated releases in this report

Diffuse source	Emissions to surface water from diffuse sources (t/year)	Emissions to waste water (t/year)	Discharged to the aquatic environment (t/year)	Discharged with river water into the sea (t/year)
Total diffuse emission (Table 6.3)	915	302		
Car wash	55	109		
Textile wash		26		
Storm water overflow		52	78	
WWTP emissions			3,5	
Rivers (based on monitoring data)				710
Rivers (based on PECregional)				554
Σ	915	187	82	554-710

The total emission to the aquatic environment from diffuse sources is estimated to 915+82 t/year. The estimated amount in river waters is based on only one measured data but it corresponds relatively well with the estimated discharge. It is concluded that 500~1000 t/year is emitted to the marine environment corresponding to 0,5-1,0% of the consumption.

6.2 Concentrations in the marine environment

6.2.1 Estimated concentrations

Local estimates (PEC_{local}) for the freshwater compartment are given in the EU RA DINP (2003) for the diffuse emission from production, processing of PVC and non-PVC, use in adhesives, glue and sealing, use in paint and ink. The estimated values range between 0,6 - 9 µg/l and 10 000 – 34 400 µg/kg dw for surface water and sediment, respectively. The estimated values for effluents from WWTPs were <0,05-0,06 µg/l. Corresponding the regional estimates (PEC regional) are estimate 0,7µg/l and 17 800 µg/kg dw.

The DINP concentrations found in effluents from WWTPs were <0,05-0,06 µg/l. Assuming an initial dilution factor of 10 in sea and ocean, the DINP concentration in the marine environment will be <0,005-0,006 µg/l.

6.2.2 Measured concentrations

The number of measured concentrations of DINP in the environment is limited and only two measured concentrations ~1 µg/l in an estuary were found in literature. However, comparison of DEHP and DINP measured at the same locations and in the same studies has shown that it may be suggested that the environmental concentrations of DINP are of the same order of magnitude or lower than those of DEHP (EU RA DINP 2003), cf. Section 5.2.

[1] Model prediction of dissolved PECregional above apparent solubility of 0,6 µg/l.

6.2.3 Conclusions

Based on the estimated amounts of DINP used in various applications, the most important usage was found to be wire and cables, film, sheet and coated products, coated fabric, wall covering etc. and flooring. The review of the diffuse release from different sources showed that floor cleaning and car washing resulting in emissions to waste water of 236 and 109 t/year, respectively, were the most important specific sources of emission of DINP to waste water. Furthermore, it was estimated that, within the EU, an amount of 82 t/year was discharged into the fresh and marine water environments from storm water and WWTPs. It is concluded that 500~1000 t/year is emitted to the marine environment corresponding to 0,5-1,0% of the consumption. Estimates based on river concentrations (monitored or modelled) do not take direct discharge from industry or settlements situated in coastal regions into account and have to be added to this amount.

A DINP concentration of <0,005-0,006 µg/l was estimated in the marine environment after initial dilution of wastewater effluents. The estimate was based on measured DINP concentrations in effluents from WWTPs in Denmark.

There were no monitoring data on the DINP concentrations in the marine environment. However, as the consumption and estimated emission of DINP is approx. 5 times lower than for DEHP, 5 times lower concentrations may be assumed for the marine environment (estuary and ocean water) corresponding to <0,0001-0,075 µg/l in water and <0,007-3 mg/kg dw in sediments.

6.3 Environmental properties

6.3.1 Physico-chemical properties

Physico-chemical properties are reported in the EU RA DINP (2003):

- Molecular weight = 421 g/mol
- Melting point ~ –50°C
- Boiling point ~ 420°C
- Density = 0,97 g/ml
- Vapour pressure = $6 \cdot 10^{-7}$ hPa at 20°C
- Water solubility = 0,0006 mg/l
- log P_{ow} = 8,8

6.3.2 Degradation

Only insignificant abiotic degradation is expected in aquatic environments (2003 EU RA DINP 2003, Staples et al. 1997).

Results of ready biodegradability tests differ largely with degradations from <1 to 81% (Staples et al. 1997, Scholz et al. 1997, EU RA DINP 2003). It is concluded in the EU RA DINP (2003) that DINP is readily biodegradable, but that a half-life of 50 days in surface waters should be used for risk assessment purposes. A similar half-life can be expected in marine areas with relative high concentrations of suspended matter e.g. in estuaries. In marine areas where this is not the case a somewhat longer half-life may be expected. In the revised EU TGD on risk assessment a half life of 150 days is recommended (EU, 2003). In sediment, a very low degradation (≤1%) was determined under both aerobic and anaerobic conditions (Johnson et al. 1984). A half-life in sediments of 3 000 days is proposed in the EU RA DINP (2003).

For WWTP a removal of 93% has been estimated of which only 10% is a result of biodegradation (EU RA DINP 2003).

As the substance is considered readily biodegradable neither the OSPAR screening criterion not the EU P-criterion are fulfilled.

6.3.3 Bioaccumulation

Only one bioaccumulation study performed with molluscs was found. A total BCF of 1844 was reported by Solbakken et al. (1985) (the exposure concentration was 61 µg/l and the test procedure was static). A BCF based on the parent compound was not determined.

As for DEHP not only the parent compound but also the main metabolite the mono-ester MINP may be of interest in the context of risk assessment as the mono-ester is expected to exhibit toxicity.

In a recent study (not yet included in the EU RA) regarding concentration in fish in the Netherlands (23 locations), indicates concentrations in fish < 0,01 mg/kg ww, whereas concentrations of < 0,025 to 6,2 mg/kg dw had been measured in sediment at the same locations (Bob Diderich pers. comm. 2001). These results indicate a low potential for bioaccumulation.

For DIDP a BCF value of 4000 based on ^{14}C on mussels has been established and can indicatively be used for DINP (EU RA DINP 2003). For fish the value obtained for DEHP of 840 may be chosen for a first approach (EU RA DINP 2003).

As the BCF values seem to be lower for fish than for crustaceans (based on DEHP) and molluscs (based on DIDP) other food chain effects than through fish seem to be as relevant as the fish food chain.

Considering the similarity between DINP and DEHP, and that DINP is regarded as not rapidly biodegradable in the marine environment and has a potential for bioaccumulation, DINP may have a high potential for food chain transfer.

In conclusion, the OSPAR screening B-criterion is fulfilled whereas the EU B-criterion (BCF>2000) is not fulfilled for fish. However, as for DEHP it is fulfilled for some invertebrates such as crustacean.

6.3.4 Aquatic toxicity

MICROORGANISMS

Studies concerning toxicity to microorganisms are reported in Table 6.7.

Table 6.7. Toxicity of DINP to microorganisms

Test species	F/S	N/M	Test duration	EC0 (mg/l)	Reference
Activated sludge of predominantly domestic sewage	F	M	3 h	≥83	EXXON 1997. Quoted from EU RA DINP 2003
Bacteria *Photobacterium phosphoreum*	M	M	15 min	>100	EXXON 1997. Quoted from EU RA DINP 2003
Protozoa *Tetrahymnia pyriformis*	F	M	24 h	>200	Yoshizawa et al. 1977. Quoted from EU RA DINP 2003

F: Fresh water; S: Salt water; N: Nominal concentration; M: Measured concentration

No toxic effect of DINP on microorganisms was observed in any of the tests performed, which is the reason why it is not possible to derive a NOEC.

TOXICITY TO ALGAE

The toxicity studies with DINP for freshwater and marine algae are summarised in Table 6.8.

Table 6.8. Toxicity of DINP to algae

Test species	(F/S)	N/M	Test duration	Endpoint	EC50 or LC50 (mg/l)	NOEC (LOEC) (mg/l)	Reference
Selenastrum capricornutum	F	M	5 d static	Cell number	>1,8	1,8	Adams et al. 1995
S. capricornutum	F	M	120 h	Growth rate	>2,8	≥2,8	CMA 1984. Quoted from EU RA DINP 2003
Scenedesmus subspicatus	F	N	72 h	Growth rate	>500	-	BASF 1988. Quoted from EU RA DINP 2003
S. subspicatus	F	?	72 h	Growth rate	>100	≥100	Hüels 1995. Quoted from EU RA DINP 2003

F: Fresh water; S: Salt water; N: Nominal concentration; M: Measured concentration; NOEC: No Observed Effect Concentration; LOEC: Lowest Observed Effect Concentration.

From the above results, DINP seems to have no acute or chronic toxicity to algae. It is thus not possible to derive a NOEC. The effect concentrations given are all far above the water solubility of the substance.

TOXICITY TO INVERTEBRATES

The short-term toxicity data on DINP for freshwater and marine invertebrates are presented in Table 6.9 and the long-term toxicity data on DINP for freshwater and marine invertebrates are presented in Table 6.10.

Table 6.9. Short-term toxicity of DINP to aquatic invertebrates

Test species	(F/S)	N/M	Test duration	Endpoint	EC50 or LC50 (mg/l)	NOEC (LOEC) (mg/l)	Reference
Water flea *Daphnia magna*	F	M	48 h static	Mortality	>0,06	0,06	Adams et al. 1995
Water flea *D. magna*	F	N	48 h	Mortality	>1,0	1,0	Brown and Williams 1995. Quoted from EU RA DINP 2003
Midge *Paratanytarsus parthenogenica*	F	M	48 h static	Mortality	>0,08	0,08	Adams et al. 1995
Mysid shrimp *Mysidopsis bahia*	S	M	96 h static	Mortality	>0,39	0,39	Adams et al. 1995

F: Fresh water; S: Salt water; N: Nominal concentration; M: Measured concentration; NOEC: No Observed Effect Concentration; LOEC: Lowest Observed Effect Concentration.

Table 6.10. Long-term toxicity of DINP to aquatic invertebrates

Test species	(F/S)	N/M	Test duration	Endpoint	NOEC (LOEC) (mg/l)	Reference
Water flea *D. magna*	F	M	21 d	Survival	0,034 (0,089)	Rhodes et al. 1995
Water flea *D. magna*	F	N	21 d	Survival/growth reproduction	>1,0	Brown and Williams 1994

F: Freshwater; S: Saltwater; N: Nominal concentration; M: Measured concentration; NOEC: No Observed Effect Concentration; LOEC: Lowest Observed Effect Concentration.

From the result given above, DINP seems to have no acute toxicity to crustaceans. In a chronic toxicity study with *D. magna*, a NOEC of 0,034 mg/l (Rhodes et al. 1995) was obtained. It is, however, assumed that, due to the low solubility of the substance, the effect observed may in part be ascribed to an indirect effect such as floating (entrapment) of the test animals or microdroplets which may adhere to the surface of the animals. As no toxic effects of DINP towards invertebrates have been observed, it is not possible to derive a NOEC.

TOXICITY TO FISH

The short-term toxicity data on DINP for freshwater and marine fish are presented in Table 6.11 and the long-term toxicity data on DINP for freshwater and marine fish are presented in Table 6.12.

Table 6.11. Short-term toxicity data on DINP for fish

Test species	(F/S)	N/M	Test duration	Endpoint	EC50 or LC50 (mg/l)	NOEC (LOEC) (mg/l)	Reference
Bluegill sunfish *Lepomis macrochirus*	F	M	96 h static	Mortality	>0,17	0,14	Adams et al. 1995
Rainbow trout *Oncorhynchus mykiss*	F	M	96 h flow through	Mortality	>0,16	0,16	Adams et al. 1995
Fathead minnow *Pimephales promelas*	F	M	96 h static	Mortality	>0,14		Adams et al. 1995
Fathead minnow *P. promelas*	F	M	96 h flow through	Mortality	>0,19	0,19	Adams et al. 1995
Sheepshead minnow *Cyprinodon variegatus*	S	M	96 h flow through	Mortality	>0,52	0,52	Adams et al. 1995
Zebra fish *Brachydanio rerio*	F	M	96 h semi static	Mortality	>100	100	Hüels 1995. Quoted from EU RA DINP 2003

F: Fresh water; S: Salt water; N: Nominal concentration; M: Measured concentration; NOEC: No Observed Effect Concentration; LOEC: Lowest Observed Effect Concentration.

In summary, no acute effects have been reported in fish exposed to concentrations of DINP at or above the solubility limit.

A two-generation feeding study with a dose of 20 mg DINP/kg dry flake food has been carried out with Japanese medaka *(Oryzias latipes)* by Patyna et al. (1999) (quoted from EU RA DINP 2003). No toxic effects were observed on the F0, the F1 or the F2 generation.

As no toxic effect of DINP has been obtained in chronic toxicity studies with fish, it is not possible to derive a NOEC value for fish.

TOXICITY TO SEDIMENT LIVING ORGANISMS

A 10-day sediment toxicity test on DINP has been performed with the chironomid *Chironomus tentans* and the amphipod *Hyalella azteca*. No effects were observed at the maximum concentration tested. The NOECs based on measured sediment concentrations were 2900 and 2679 mg/kg (dwt) for chironomids and amphipods, respectively (EU RA DINP 2003).

In addition to the short term tests, a 28-day study with *Chironomus riparius* revealed no effects upon adult emergence, time to emergence and sex ratio with either DIDP or DEHP up to a concentration of 10 000 mg/kg dw. A read-across to DINP is possible (Brown et al. 1995 in EU RA DINP 2003).

Recently, a test with frog eggs (Rana arvalis) was performed in parallel with coarse and fine sediment. 7000 fertilised frog eggs from at least 10 females were collected. None of the examined eggs had reached the gastrula stage. DINP was dissolved in acetone and mixed with air-dried sediment. After evaporation, the sediment was mixed with fresh test sediment slowly agitated until equilibrium (up to 20 days). The test was performed at 10 degrees C. The nominal test concentrations were 100, 300 and 1000 mg/kg dw. A control as well as an acetone control was also performed. The average measured test concentrations were 113, 245 and 1010 mg/kg dw for fine sediment and 120, 295 and 710 mg/kg dw for coarse sediment. The porewater concentrations and overlying water concentrations were not determined (Solyom et al., 2000). No significant effects were observed on hatching success as well as mortality and deformation of hatched (EU RA DINP 2003).

On the basis that no effects have been seen in sediment effect studies with DINP and by reading across to DIDP and DEHP the tentative conclusion is that there are no effects on sediment dwellers of DINP at environmentally realistic concentrations.

ENDOCRINE DISRUPTION

In an *in-vivo* test with DINP, no effects on the uterine wet weight and vaginal cornification were observed in rats after oral exposure for 4 days at doses corresponding to 20, 200 and 2000 mg/kg (Zacharewski et al. 1998). In an investigation by Harris et al. (1997), DINP was shown to be weakly estrogenically active in *in-vitro* recombinant yeast screening test, with a relative potency of approx. $5 \cdot 10^7$ times less than 17β-

estradiol. Regarding the effects on ecosystems, the most relevant test results are from the multigeneration study with Japanese medaka *(Oryzias latipes)* by Patyna et al. (1999) as already described above. The male to female ratio (3:1) was the same in all groups. Phenotypic gender classification of male and female fish was histopathologically confirmed to be 100% correct. Somatic gonadal index and live somatic index were not significantly different in any group. Based on these results, chronic exposure to DINP does not seem to impact any populational parameter.

TOXICITY FOR BIRDS AND MAMMALS (SECONDARY POISONING)

Please refer to EU RA DINP (2003) for details. DINP has low toxicity towards mammals and is not classified. The lowest NOAEL of 88 mg/kg bw/d has been determined in a two-year repeated dose toxicity study with rats. This corresponds to a food concentration of 1500 mg/kg. Using an assessment factor of 10, a $PNEC_{oral}$ of 150 mg/kg can be estimated (EU RA DINP 2003). DINP is not classified in Annex 1 of Directive 67/548/EEC and no classification has been proposed in the EU RA DINP (2003).

SUMMARY - EFFECT

The reason for the variability in the toxicity test should most probably be sought in experimental difficulties arising from the low water solubility of DINP. The formation of micro-droplets, surface films and adsorption to surfaces of the test organisms lead to difficulties in maintaining steady exposure concentrations and/or cause direct physical effects.

DINP shows no acute toxicity to algae, crustaceans or fish. Toxicity was observed in a long-term test with *Daphnia magna* (LOEC = 0,089 mg/l, NOEC = 0,034 mg/l) performed at concentrations higher than the water solubility level (0,6 µg/l). Thus, the toxicity observed is expected mainly to be ascribed to an indirect effect such as floating (entrapment) or micro-droplets, which may adhere to the surface of the animals. As DINP readily adsorbs to organic particles and also to various surfaces, the toxicity of DINP should be determined by oral exposure. In a two-generation test with Japanese medaka, no effect was observed when the fish were exposed to DINP via the food.

Acute toxicity tests with sediment-dwelling organisms have been performed with a chironomid and an amphipod, and tests with prolonged exposure have been performed with a chironomid and tadpoles of a frog. No toxicity (NOEC) was observed at the highest concentrations tested at 10 000 and 1000 mg/kg dw, for chironomids and tadpoles respectively.

Only weak estrogenic activity of DINP has been shown in an *in-vitro* test with a recombinant yeast screening test (Harris et al., 1997). No endocrine disruption effect at the population level has been observed. Endocrine disrupting effects in the aquatic environment are thus not expected.

DINP has low toxicity towards mammals and is not classified.

In conclusion, DINP does not fulfil the OSPAR or EU T-criterion.

6.3.5 PNEC for the aquatic environment

As there are no short- or long-term studies showing effect at or below the water solubility level, it is not possible to specify any acute or chronic NOEC values for organisms exposed via water. Hence, a $PNEC_{aquatic}$ cannot be established. Also when DINP was exposed via the food, no effects were obtained. It is thus not possible to derive a PNEC for aquatic species. No $PNEC_{aquatic}$ is proposed in the EU RA DINP 2003 or in the review by Petersen & Pedersen (1998).

For the freshwater sediment compartment no effects have been seen and consequently a $PNEC_{sediment}$ cannot be established.

For secondary poisoning the lowest NOAEL of 88 mg/kg bw/d has been determined in a two-year repeated dose study with rats. This corresponds to a food concentration of 1500 mg/kg. Using an assessment factor of 10, a $PNEC_{oral}$ of 150 mg/kg can be estimated for top predators (EU RA DINP 2003).

6.4 PBT assessment and Risk assessment

DINP cannot be considered a PBT chemical according to the OSPAR or EU criteria.
No acute or chronic toxic effects are determined at the water solubility level of 0,6 µg/l. The two measured concentrations for an estuary are ~1 µg/l while estimated concentrations in the marine environment are in the range of <0,0001-0,075 µg/l. Thus, no direct ecotoxic effects are foreseen. No information on potential food chain effects is available.

For the sediment compartment, no measured concentrations are available but, when comparing with DEHP, a realistic concentration range of <0,007-3 mg/kg dwt has been proposed. No effects have been seen in

testing with freshwater sediment organisms. Thus, no direct effects of DINP on sediment organisms are foreseen.

DINP has a potential for food chain transfer in marine mammals. However, with the present consumption of DINP and the resulting concentrations in the environment, taking the apparently low toxicity of DINP into account no long-term effects and no endocrine disrupting effects in the aquatic environment is expected. Thus, risks to marine mammals are not expected at present.

6.5 Desired Reduction and Identification of possible measures

Risk reduction seems not to be needed at present.

7. Di(isodecyl)phthalate

7.1 Production, use and emission

7.1.1 Production

Five producers or importers of di(isodecyl)phthalate (DIDP) are listed in the EU RA DIDP (2003). The total DIDP production within the EU was estimated to 279 450 t/year in 1994. There was no import of DIDP in 1994 and the export data for three companies amounted to approx. 38 000 t/year. There has been an increase in the DIDP consumption in Western Europe during the last decade. The consumption has increased from 120 000 t/year in 1985 to approx. 200 000 t/year in 1994 (EU RA DIDP 2003).

Almost all the DIDP consumed in the EU is transported by road tankers or by ship. Approx. 15% corresponding to 30 000 t/year are transported by ship. The remaining 85% of the substance (170 000 t/year) are transported by road.

7.1.2 Use

The use of the consumed DIDP is distributed over following applications:

- 95,5% in PVC;
- Approx. 3% in non-PVC involving polymer-related use (e.g. rubbers);
- Approx. 1% in non-polymers including inks, paints, sealants and ceramics.

No precise quantitative assignment was available for the non-polymer use categories. Therefore, an even distribution among the main categories of non-polymer-related use was assumed (EU RA DIDP 2003). The estimated amount of DIDP used in the various applications is given in Table 7.1.

Table 7.1. Estimated quantitative usage distribution of DIDP for different applications (EU RA DIDP 2003)

Use category		**Amount use of DIDP within the EU in 1994 (t/year)**
PVC end-uses	*Calendering:* Film, sheet and coated products Flooring, roofing, wall covering	 29 987 6 685
	Extrusion: Hose and profile Wire and cable Clear, medical, film	 10 123 54 807 13 580
	Injection moulding: Footwear and miscellaneous	 15 843
	Platisol spread coating: Flooring General (coated fabric, wall covering, etc.)	 20 055 21 774
	Other plastisol applications: Car undercoating and sealants Slush/rotational moulding etc.	 14 516 3 629
Non-PVC end-uses		
• Polymer-related		6 390
• Non-polymer-related	Anti-corrosion paint Antifouling paint Sealing compounds Textile inks	520 520 520 520
Total		199 480

No information on the quantitative use of the non-PVC end-product containing DIDP was found. In the EU RA DIDP (2003), the PVC end-products were split into in-door and out-door use and the volumes of DIDP in applications estimated (Table 7.2).

Table 7.2. PVC products containing DIDP based on industry data divided into in-door and out-door applications (EU RA DIDP 2003)

Application		**DIDP (t/year)**
In-door	Coated product	[1]
	Film & sheet	[1]
	Wires & cables	27 400
	Hoses & profiles	[1]
	Floor	20 055
Our-door	Roofing material	430[2]
	Roofing (coil coating)	2 150[2]
	Wires & cables	27 400
	Coated fabric	9 060[2]
	Hoses & profiles	2 590[2]
	Car undercoating	14 516
	Shoe soles	15 843

1) No data
2) Estimated from DEHP, based on marked shares

7.1.3 Emissions from diffuse sources

Emission of DIDP is expected from the following diffuse sources (EU RA DIDP 2003):

1. Distribution of DIDP within the EU by road transport and shipping of the substance;
2. Exterior and interior use of DIDP-containing PVC products;
3. Use of non-PVC polymers containing DIDP;
4. Applied anti-corrosion paints containing DIDP;
5. Applied anti-fouling paints containing DIDP;
6. Applied sealings containing DIDP;
7. Use of DIDP-treated textiles;
8. Disposal of end-products (waste).

The calculation of the total emission from distribution of DIDP was based on estimated losses from transportation by road and ship, respectively. A loss of 1 kg DIDP per 20 tonnes was estimated for transportation by road and an estimated loss close to 0,3% was found for the transportation by ship. The losses are achieved from cleaning of tanks, lines and pumps etc.

The emission from the above listed steps 2-8 of the life cycles was primarily estimated from experimental studies and assumed technical lives of the products (EU RA DIDP 2003). A summary of the total releases from the different diffuse sources is given in Table 7.3.

Table 7.3. Estimated emissions of DIDP to fresh water (surface water) and waste water from diffuse sources in the EU

Diffuse source	**Total emission of DIDP in the EU**		
		Surface water (t/year)	Waste water (t/year)
Transportation/ distribution		0	17,5
End-product use	Floor cleaning (PVC products for interior use)	0	444
	Car undercoating (PVC products for outdoor use)	109	
	Roofing material (PVC products for outdoor use)	2,4	0
	Coils coating (PVC; outdoor)	60	0
	Fabric coating (PVC; outdoor)	50,6	0
	Cables & wires (PVC; outdoor)	45,9	0
	Hoses & profiles (PVC; outdoor)	3,6	0
	Shoe soles (PVC; outdoor)	8,2	0
	DINP containing non-PVC polymers (in- and outdoor)	9,4	15
	Applied anti-corrosion paints	10,2	0
	Applied antifouling paints[1]	0	0
	Applied sealings	2,9	0
	Use of DIDP-treated textiles	0	99
Waste (disposal of end-products)	Landfills (leakage water)	≈0[3]	≈0
	Waste remaining in the environment	1460	0
Total		1762	576

1) 520 t/year will be released into the marine environment

Table 7.3 shows that floor cleaning was found to be the predominant source of diffuse releases to waste water. Waste remaining in the environment is seen to be the principal source of emission into surface water. Furthermore, 520 t DIDP/year are expected to be released directly into the marine environment from the use of antifouling paints.

[3] Based on 28 months laboratory studies under accelerated landfill conditions (Mersiowsky et al. 1999).

SOURCE INVESTIGATION

No analysis of DIDP was included in the Danish studies of phthalates release from car wash etc. (Vikelsøe et al 1998) or textile wash (Hoffman 1996; Larsen et al. 2000). It was assumed that, within the EU, the DIDP released due to car wash and textiles corresponds proportionately to the release from DEHP. The releases are estimated by use of the ratio of the consumption of DIDP to DEHP for car undercoating and textile inks, respectively, together with the estimated releases of DEHP. The estimates are:

- car wash to waste water: $\left[\frac{14500\text{ t DIDP/year}}{7140\text{ t DEHP/year}} \cdot 109\text{ t/year}\right]$ = 221 t/year
- car wash to surface water: $\left[\frac{14500\text{ t DIDP/year}}{7140\text{ t DEHP/year}} \cdot 55\text{ t/year}\right]$ = 112 t/year
- textile wash to waste water: $\left[\frac{570\text{ t DIDP/year}}{1600\text{ t DEHP/year}} \cdot 46\text{ t/year}\right]$ = 16,4 t/year

EFFLUENT FROM MUNICIPAL WASTEWATER TREATMENT PLANTS

There are no available monitoring data from municipal WWTPs. The total amount of DIDP discharged into fresh water and marine environments from municipal WWTPs in the EU is assumed to equal the values of DIDP of 3,5 t/year.

STORM WATER

The Danish investigations of phthalates in storm water 1995-1996 did not include analysis of DIDP (Danish EPA 1997). It is assumed that, within the EU, the amount of DIDP discharged from storm water is proportionate to the DEHP discharge. The ratio of the total consumption of DIDP (200 000 t/year) to DEHP (476 000 t/year) was used in the estimation:

- Mean DIDP emission to storm water: $\left[\frac{2000}{476000} \cdot 32\ \mu\text{g/l}\right]$ = 13,4 µg/l
- DIDP emission to WWTPs: 97 t/year
- DIDP emitted directly into the aquatic recipient: 146 t/year

ESTIMATION OF THE DIDP DISCHARGE FROM RIVER WATER INTO THE MARINE ENVIRONMENT

In the EU, the discharge of DIDP from rivers into the marine water was estimated by use of the assumptions listed for DBP (Section 3.1.3).

The only measured value of DIDP is approx. 1 µg/l (EU RA DIDP 2003). According to the assumptions given for DBP, the DIDP discharge from rivers in the EU will be approx. 710 t/year. Based on PECregional = 1,8 µg/l (total concentration) a discharge via rivers to the marine environment of 1280 t/year is estimated. Estimates based on river concentrations (monitored or modelled) do not take direct discharge of DIDP from industry or settlements situated in coastal regions into account. The discharge from diffuse sources accounts for approx. 59% of the total discharge of DIDP (EU RA DIDP 2003).

SUMMARY

A summary of the estimated release of DIDP from the different diffuse sources above is given in Table 7.4.

Table 7.4. Total diffuse emission of DIDP in the EU according to the EU RA DIDP (2003) and the releases estimated in this report

Diffuse source	Emissions to surface water from diffuse sources (t/year)	Emissions to waste water (t/year)	Discharged to the aquatic environment (t/year)	Discharged with river water into the sea (t/year)
Total diffuse emission (Table 7.3)	1762	576		
Car undercoating/wash	112[1]	221		
Textile wash		16		
Storm water overflow		97	146	
WWTP emissions			3,5	
Rivers (based on monitoring data)				710
Rivers (based on PECregional)				1280
Σ	1762	334	150	710-1280

1) Assumed to be included in the total diffuse emission figure.

The data in Table 7.4 show that the summation of the emissions to waste water from car wash, textile and storm water corresponds to 42% of the estimated total diffuse emission to waste water in the EU RA DIDP (2003). Emission from floor cleaning (444 t/year) is still seen to be the largest source to the total diffuse emission of DIDP (Table 7.3). Furthermore, the estimated release from textile wash of 16 t/year is much lower than the estimated value given in the EU RA DIDP (2003) of 99 t/year.

The total emission to the aquatic environment from diffuse sources is estimated to be 1917 (1762 + 150) t/year. The content in river waters is estimated to be 710-1280 t/year (based, however, on only one measured concentration and the estimated PECregional), which corresponds to 0,4-0,6% of the consumption. Additionally, 520 t/year are emitted directly into the marine environment from use in antifouling paints. Thus, the total emission to the marine environment is in the range of 1200-4500 t/year. Estimates based on river concentrations (monitored or modelled) do not take direct discharge from industry or settlements situated in coastal regions into account and have to be added to this amount.

7.2 Concentrations in the marine environment

7.2.1 Estimated concentrations

No estimated DIDP concentrations were found in literature for the marine environment. Regional and continental estimates (PEC_{local}) for the fresh water compartment are given in the EU RA DIDP (2003) for diffuse and point emission. A summary of the estimated PECs for the fresh water environment achieved by the use of the SIMPLEBOX model in EUSES is given in Table 7.5.

Table 7.5. Predicted Environmental Concentration (PEC) of DIDP estimated by the EUSES model (EU RA DIDP (2003)

Step in EUSES	WWTP (µg/l)	Surface water (µg/l)	Sediment (mg/kg dw)
REGIONAL Diffuse & point	-	1,8	32

Based on measured DIDP concentrations in sediment samples from freshwater environment, the calculated $PEC_{regional\text{-}sed}$ was overridden by a value of 1 mg/kg dw in the EU RA DIDP (2003).

7.2.2 Measured concentrations

The amount of measured concentrations in the environment for DIDP is limited and no data were found in literature on the marine environment. As mentioned above, comparison of DEHP and DIDP measured at the same location and the same studies has, however, shown that it may be suggested that the environmental

concentrations of DIDP are of the same order of magnitude or lower than those of DEHP (EU RA DIDP 2003), cf. section 5.2.

7.2.3 Conclusions

Based on the estimated amount of DIDP used in various application the most important usage was: wire and cable, film, sheet and coated products, coated fabric, wall covering etc. and flooring. The review and estimations of the diffuse release from different sources showed, that floor cleaning and car washing resulting in emissions of 444 and 333 t/year, respectively, were among the most important sources of emission of DIDP into waste water. The estimated release due to the use of DIDP-treated textiles was 16 t/year. A release of 150 t/year has been estimated for the total amount of DIDP discharged into freshwater and marine environments from municipal WWTPs and storm water. The discharges with river water into the sea are estimated to be 710-1280 t/year corresponding to 0,4-0,8 % of the consumption. Furthermore, it was estimated that 520 t/year will be released into the marine environment from antifouling paints. Thus, the total emission to the marine environment is in the range of 1200-1830 t/year. Estimates based on river concentrations (monitored or modelled) do not take direct discharge from industry or settlements situated in coastal regions into account and have to be added to this amount.

No monitoring data on the DIDP concentrations in effluent from WWTPs and the marine environment were found. As the consumption and estimated emission of DIDP is, however, approx. 2,5-5 times lower than for DEHP, 2,5-5 times lower concentrations may be assumed for the marine environment (estuary and ocean water) corresponding to <0,0001-0,150 µg/l in water and <0,007-3 mg/kg dw in sediments.

7.3 Environmental properties

7.3.1 Physico-chemical properties

Physico-chemical properties are reported in the EU RA DIDP (2003):

- Molecular weight = 447 g/mol
- Melting point ~ –45°C
- Boiling point > 400°C
- Density = 0,97 g/ml
- Vapour pressure = $2{,}8 \cdot 10^{-7}$ hPa at 20°C
- Water solubility = 0,0002 mg/l
- log P_{ow} = 8,8

7.3.2 Degradation

Only insignificant abiotic degradation is expected in aquatic environments (EU RA DIDP 2003, Staples et al. 1997).

In tests of ready biodegradability, degradations of 42% and 67% have been reached after 14 and 28 days of incubation, respectively (CITI 1992, Staples et al. 1997). In the EU RA DIDP (2000), it is concluded that DIDP is readily biodegradable (but failing the 10-day window). For risk assessment purposes a half-life of 50 days for surface water is proposed. A similar half-life can be expected in marine areas with relative high concentrations of suspended matter e.g. in estuaries. In marine areas where this is not the case a somewhat longer half-life may be expected. The revised EU TGD on risk assessment recommends the use of a half life of 150 days (EU, 2003). In a sediment-water test system, a mineralization of 1% was determined after incubation in 28 days at aerobic conditions (Johnson et al. 1984).

For WWTP a removal of 92% has been estimated of which only 4% is a result of biodegradation (EU RA DIDP 2003).

In conclusion DIDP is considered as readily biodegradable and therefore the OSPAR screening or the EU P-criterion is not fulfilled.

7.3.3 Bioaccumulation

In the review given by Stables et al. (1996), several bioaccumulation studies with molluscs, crustaceans and fish are referred. A number of studies on bioaccumulation of DIDP referred in Stables et al. (1996) are given below.

Brown and Thompson (1982a) determined the total BCF for *Mytilus edulis,* BCF values of 3977 and 2998, respectively, were found based on total ^{14}C measurements (exposure concentrations 4,4 and 41,7 µg/l i.e. above water solubility, test procedure: flow through, exposure period: 28d). For crustaceans, only data on *Daphnia magna* exist. Brown and Thompson (1982b) determined the total BCF in static renewal tests and

found the following BCF values based on total ^{14}C measurements: 90 (exposure concentration: 100,4 μg/l); 128 (exposure concentration: 32,6 μg/l); and 147 (exposure concentration: 9,6 μg/l). For fish, a total BCF for carp (*Cyprinus carpio)* of <3,6 to <14,4 for the parent compound, (test procedure: flow through; exposure concentration: 0,1-1,0 μg/l; exposure period not known) (CITI, 1992). Due to the similarity between DEHP and DIDP the BCF for fish of 840 retained for the risk assessment of DEHP should also be used for DIDP (EU RA DIDP 2003).

As for DEHP not only the parent compound but also the main metabolite the mono-ester MIDP may be of interest in the context of risk assessment as the mono-ester is expected to exhibit toxicity. BCF values based on ^{14}C measurements have been determined for blue mussels (3000-4000), *Daphnia magna* (90-147) and for fish based on the parent compound (<3,6 to <14,4). Thus, it is concluded that DIDP has a potential for being bioaccumulated in aquatic organisms.

Considering the similarity between DIDP and DEHP, and that DIDP is regarded as not rapidly biodegradable in the marine environment and has a potential for bioaccumulation, DIDP is expected to have a potential for food chain transfer.

In conclusion, DIDP does not fulfil the EU B-criterion (BCF>2000) for fish. However, the B criterion is fulfilled for molluscs where BCF around 3000 to 4000 are reported.

7.3.4 Aquatic toxicity

MICROORGANISMS

Studies concerning toxicity to microorganisms are reported in Table 7.6.

Table 7.6. Toxicity of DIDP to microorganisms

Test species	N/M	Test duration	EC 0 (mg/l)	Reference
Activated sludge of predominantly domestic sewage	M	3 h	≥85	EXXON 1997. Quoted from EU RA DIDP 2003
Bacteria *Photobacterium phosphoreum*	M	15 min	≥85	EXXON 1997. Quoted from EU RA DIDP 2003
Protozoa *Tetrahymnia pyriformis*	M	24 h	>200	Yoshizawa et al. 1977. Quoted from EU RA DIDP 2003

F: Fresh water; S: Salt water; N: Nominal concentration; M: Measured concentration.

As no toxic effect of DIDP on microorganisms was observed in any of the tests performed, it is not possible to derive a NOEC.

TOXICITY TO ALGAE

The toxicity studies with DIDP for freshwater and marine algae are summarized in Table 7.7.

Table 7.7. Toxicity of DIDP to algae

Test species	(F/S)	N/M	Test duration	Endpoint	EC50 or LC50 (mg/l)	NOEC (LOEC) (mg/l)	Reference
Selenastrum capricornutum	F	M	8 d static	Cell number	>0,80	0,80	Adams et al. 1995
S. capricornutum	F	M	196 h	Growth rate	>1,3	≥1,3	CMA 1984. Quoted from EU RA DIDP 2003
Scenedesmus subspicatus	F	N	72 h	Growth rate	>500	-	BASF 1988. Quoted from EU RA DIDP 2003

F: Fresh water; S: Salt water; N: Nominal concentration; M: Measured concentration; NOEC: No Observed Effect Concentration; LOEC: Lowest Observed Effect Concentration.

From the above results, DIDP seems to have no acute or chronic toxicity to algae. It is thus not possible to derive a NOEC. The effect concentrations given are all far above the water solubility of the substance.

TOXICITY TO INVERTEBRATES

The short-term toxicity data on DIDP for freshwater and marine invertebrates are presented in Table 7.8 and the long-term toxicity data on DIDP for freshwater and marine invertebrates are presented in Table 7.9.

Table 7.8. Short-term toxicity of DIDP to aquatic invertebrates

Test species	(F/S)	N/M	Test Duration	Endpoint	EC50 or LC50 (mg/l)	NOEC (LOEC) (mg/l)	Reference
Water flea *Daphnia magna*	F	M	48 h static	Immobilisation	>0,32		Brown and Thompson 1982b
Water flea *D. magna*	F	M	48 h static	Mortality	>0,02	0,07	Adams et al. 1995
Water flea *D. magna*	F	M	48 h	Mortality	>1,0	1,0	Brown and Williams 1994
Midge *Paratanytarsus parthenogenica*	F	M	48 h static	Mortality	>0,64	0,64	Adams et al. 1995
Mysid schrimp *Mysidopsis bahia*	S	M	96 h static	Mortality	>0,08	0,08	Adams et al. 1995

F: Fresh water; S: Salt water; N: Nominal concentration; M: Measured concentration; NOEC: No Observed Effect Concentration; LOEC: Lowest Observed Effect Concentration.

Table 7.9. Long-term toxicity of DIDP to aquatic invertebrates

Test species	(F/S)	N/M	Test duration	Endpoint	NOEC (LOEC) (mg/l)	Reference
Water flea *D. magna*	F	M	21 d	Survival	0,03 (0,06)	Rhodes et al. 1995
Water flea *D. magna*	F	M	21 d	Survival/ Reproduction	0,10	Brown and Thompson 1982b
Water flea *D. magna*	F	M	21 d	Survival/ Reproduction	1,0	Croudace et al. 1995
Mussel *Mytilus edulis*	S	N	28 d	Mortality, byssal thread attachment	0,042	Brown and Thompson 1982a

F: Fresh water; S: Salt water; N: Nominal; M: Measured concentration; NOEC: No Observed Effect Concentration; LOEC: Lowest Observed Effect Concentration.

From the result given above, DIDP seems to have no acute toxicity to crustaceans. In a chronic toxicity study, a NOEC of 0,03 mg/l (Rhodes et al. 1995) was obtained. It is, however, assumed that, due to the low solubility of the substance, the effect observed may in part be ascribed to an indirect effect such as floating (entrapment) of the test animals or micro-droplets which may adhere to the surface of the animals. As no toxic effects of DIDP on invertebrates have been observed, it is not possible to derive a NOEC.

TOXICITY TO FISH

The short-term toxicity data on DIDP for freshwater and marine fish are presented in Table 7.10 and the long-term toxicity data on DIDP for freshwater and marine fish are presented in Table 7.11.

Table 7.10. Short-term toxicity data on DIDP for fish

Test species	F/S	N/M	Test Duration	Endpoint	EC50 or LC50 (mg/l)	NOEC (LOEC) (mg/l)	Reference
Bluegill sunfish *Lepomis macrochirus*	F	M	96 h	Mortality	>0,37	0,37	Adams et al. 1995
Rainbow trout *Oncorhynchus mykiss*	F	M	96 h flow through	Mortality	>0,62	0,14	Adams et al. 1995
Fathead minnow *Pimephales promelas*	F	M	96 h	Mortality	>0,47		Adams et al. 1995
Fathead minnow *P. promelas*	F	M	96 h flow through	Mortality	>1,0	1,0	Adams et al. 1995
Red killifish *Oryzias latipes*	F	M	48 h static	Mortality	>3000		CITI 1992
Sheepshead minnow *Cyprinodon variegatus*	S	M	96 h flow through	Mortality	>0,47	0,47	Adams et al. 1995

F: Fresh water; S: Salt water; N: Nominal concentration; M: Measured concentration; NOEC: No Observed Effect Concentration; LOEC: Lowest Observed Effect Concentration.

No studies on chronic effects on fish exposed to DIDP via the water phase have been carried out.

A two-generation feeding study with a dose of 20 mg DIDP/kg dry flake food has been carried out with red killifish *(Oryzias latipes)* by Cooper et al. (1998) (quoted from EU RA DIDP 2003). The F0 adults were terminated at day 123. No statistically significant effects (mortality, fecundity and egg production) were observed in the F0, the F1 or the F2 generation.

As no toxic effect of DIDP has been obtained in chronic toxicity studies with fish, it is not possible to derive a NOEC value for fish.

TOXICITY TO SEDIMENT-LIVING ORGANISMS

Several recent studies have been carried out for different sediment-living organisms.

Table 7.11. Effects of DIDP to sediment-living organisms

Test organism	Duration (Days)	Endpoints	NOEC (mg/kg dwt)	Reference
Midge *Chironomus riparius*	28	Adult emergence, time to emergence, sex ratio	≥10 000*	Brown et al. 1995 (Quoted from EU RA DIDP 2003)
Midge *Chironomus tentans*	10	Survival, growth	≥3 000*	Call et al. 1997 (Quoted from EU RA DIDP 2003)
Amphipod *Hyalella azteca*	10	Survival, growth	≥3 000*	Call et al. 1997 (Quoted from EU RA DIDP 2003)
Moorfrog *Rana arvalis*	28	Egg hatching, tadpole survival	≥600*	Wennberg et al. 1996 (Quoted from EU RA DIDP 2003)

* Highest concentration tested

No NOEC can be derived from the tests performed as no effect was observed at the highest tested concentrations.

Recently, a test with frog eggs (Rana arvalis) was performed in parallel with coarse and fine sediment. 7000 fertilised frog eggs from at least 10 females were collected. None of the examined eggs had reached the gastrula stage. DINP was dissolved in acetone and mixed with air-dried sediment. After evaporation, the sediment was mixed with fresh test sediment slowly agitated until equilibrium (up to 20 days). The test was performed at 10 degrees C. The nominal test concentrations were 100, 300 and 1000 mg/kg dw. A control as well as an acetone control was also performed. The average measured test concentrations were 113, 245

and 1010 mg/kg dw for fine sediment and 120, 295 and 710 mg/kg dw for coarse sediment. The porewater concentrations and overlying water concentrations were not determined (Solyom et al., 2000). No significant effects were observed on hatching success as well as mortality and deformation of hatched tadpoles (Bob Diderich pers. comm. 2001).

The results from this test with DINP support the tentative conclusion that DIDP has no adverse effects towards sediment-living organisms (EU RA DIDP 2003).

TOXICITY FOR BIRDS AND MAMMALS (SECONDARY POISONING)

Please refer to EU RA DIDP (2003) for details. DIDP is a developmental toxicant based on consistently decrease in survival indices in the two two-generation studies available. A NOAEL of 33 mg/kg bw/d for the rat can be used (decreased offspring survival day 1 and 4 in F2 generation).

Hepatic effects have been observed for the dog, and a NOAEL of 15 mg/kg bw/d can be used despite the large limitations in the study (EU RA DIDP 2003).

DIDP is not classified in Annex 1 of Directive 67/548/EEC and no classification has been proposed in the EU RA DIDP (2003).

ENDOCRINE DISRUPTION

In an *in-vivo* test with DIDP no effects on the uterine wet weight and vaginal cornification were observed in rats after oral exposure for 4 days at doses corresponding to 20, 200 and 2000 mg/kg (Zacharewski et al. 1998). No estrogenic activity was observed in an *in-vitro* test with a recombinant yeast screening test (Harris et al. 1997). Regarding the effects on ecosystems, the most relevant test results are from the multigeneration study with Japanese medaka *(Oryzias latipes)* by Cooper et al. (1998) as already described above. The male to female ratio (3:1) was the same in all groups. Phenotypic gender classification of male and female fish was histopathologically confirmed to be 100% correct. Somatic gonadal index and live somatic index were not significantly different in any group. Based on these results, chronic exposure to DIDP does not seem to affect any populational parameter.

SUMMARY

DIDP shows no acute toxicity to algae, crustaceans or fish. Toxicity was observed in a long-term test with *Daphnia magna* (NOEC = 0,03 mg/l), which, however, was performed at concentrations above the water solubility limit (0,2 µg/l). Thus, it is expected that the toxicity observed can mainly be ascribed to an indirect effect such as floating (entrapment) or micro-droplets, which may adhere to the surface of the animals.

DIDP is a developmental toxicant, however, at exposure levels one order of magnitude higher than for DEHP. DIDP is not classified.

In the investigations performed, DIDP did not have any endocrine disrupting effects.

In conclusion, DIDP doesn't fulfil the EU T-criterion.

7.3.5 PNEC for the aquatic environment

As there are no short- or long-term studies showing effect at or below the water solubility limit, it is not possible to specify any acute or chronic NOEC values for organisms exposed via water. Hence, a $PNEC_{water}$ cannot be specified. Also when DIDP was administered via the food, no effects were obtained. It is thus not possible to derive a PNEC for aquatic species. No $PNEC_{aquatic}$ has been proposed in the EU Risk Assessment on DIDP (EU RA DIDP 2003) or in the review by Petersen & Pedersen (1998).

No toxic effects were found at the highest concentrations tested with sediment organisms and consequently, no $PNEC_{sediment}$ can be derived (EU RA DIDP 2003).

For secondary poisoning the NOAEL based on hepatic effects in the dog of 15 mg/kg bw/d can be used. This corresponds to a food concentration of 500 mg/kg. Using an assessment factor of 10, a $PNEC_{oral}$ of 50 mg/kg can be estimated for top predators (EU RA DIDP 2003).

7.4 PBT assessment and Risk assessment

The overall PBT assessment of DIDP indicates that DIDP cannot be considered a PBT chemical as defined in the OSPAR or EU-TGD approaches.

No acute or chronic toxic effects are determined at the water solubility level of 0,2 µg/l. The estimated concentrations in the marine environment are in the range of <0,0001-0,15 µg/l. Thus, no direct ecotoxic effects are foreseen.

The highest estimated concentration in sediments is 3 mg/kg dw. No effects have been observed towards sediment organisms. Thus, no direct effects towards sediment organisms are foreseen.

DIDP has a potential for food chain transfer in marine mammals. However, with the present consumption of DIDP and the resulting concentrations in the environment, taking the toxicity of DIDP into account no long-term effects and no endocrine disrupting effects in the aquatic environment are expected. Thus, risks to marine mammals are not expected at present.

7.5 Desired Reduction and Identification of possible measures

Risk reduction seems not to be needed at present.

8. Desired reduction and identification of possible measures

8.1 Achieving the desired reduction

8.1.1 OSPAR targets

In 1998, phthalates were included in the OSPAR List of Chemicals for Priority Action through the "safety net" because of suspicions of endocrine-disrupting properties and indications that the substances are widespread in the environment. The OSPAR objective with regard to hazardous substances is to continuously reduce discharges, emissions and losses, with the ultimate aim of achieving concentrations in the marine environment near background values for naturally occurring substances and close to zero for man-made synthetic substances. Every endeavour will be made to move towards the target of cessation of discharges, emissions and losses of hazardous substances by the year 2020 (OSPAR, 1998).

OSPAR 2002 adopted guidance on the role of marine risk assessment, which gives, in particular, advice on the urgency of taking measures based on the PEC/PNEC ratios and the PBT properties of the chemicals (reference number: 2002-19). The following conclusions apply this guidance and follow the on-going risk assessments in the framework of the Existing Substances Regulation.

DI-N-BUTYL PHTHALATE (DBP)

DBP is not a PBT chemical according to either the EC or OSPAR PBT criteria. There is a potential risk of ecotoxicological effects on aquatic species in the marine environment (including organisms living in the sediment) on a local scale. At the regional scale, risks are expected to be negligible to low. Furthermore, as DBP is expected to degrade relatively rapidly in the environment, and the bioaccumulative potential is expected to be low in the food chain, no risk for marine mammals is foreseen. Nevertheless, DBP has a potential for endocrine-disrupting effects that need to be taken into account.

BUTYLBENZYL PHTHALATE (BBP)

Discussions on the EC Risk Assessment Report in the frame of the Existing Substances Regulation are still on-going. BBP is not a PBT chemical according to either the EC or OSPAR PBT criteria. The risk of ecotoxicological effects in the marine environment is low. No risk for the marine sediment compartment is foreseen, although, locally, there might be a risk for sediment-dwelling organisms. BBP might, nevertheless, have a potential for endocrine-disrupting effects and that needs to be taken into account.

DI(2-ETHYLHEXYL)PHTHALATE (DEHP)

Discussions on the EC Risk Assessment Report in the frame of the Existing Substances Regulation are still on-going. Regarding the PBT status of DEHP, it was considered by the PBT TM meeting that, when assessed separately, the PBT properties of DEHP should not be considered as fulfilling the EC PBT criteria, although for some of the criteria it may be a borderline case. OSPAR PBT criteria are also not fulfilled. However its widespread diffusion into the environment and measurements in remote areas suggest potential for life-time exposure that needs to be taken into account. Discussions are still on-going in the context of the Water Framework Directive in order to decide whether DEHP should be listed as a PHS (Priority Hazardous Substance) or PS (Priority Substance). The preliminary risk assessment indicates that most of the monitoring data from open marine waters are below the proposed indicative PNECs (water column and sediment). DEHP might also have a potential for endocrine-disrupting effects however the studies available are not clear and uncertainties should be taken into account.

DI(ISONONYL)PHTHALATE (DINP) AND DI(ISODECYL)PHTHALATE (DIDP)

DINP and DIDP are not PBT according to either the EU or OSPAR PBT criteria. The results of the risk assessment indicate that no direct ecotoxicological effects of DINP and DIDP are foreseen either on marine organisms living in the water column or on sediment-dwelling organisms. DINP and DIDP have a potential for food-chain transfer in marine mammals. However, with the present consumption of DINP/DIDP and the resulting concentrations in the environment, taking into account the apparently low toxicity of these two compounds, no long-term effects and no endocrine-disrupting effects in the aquatic environment are expected. Thus, at present, no risks to top-predators are expected.

CONCLUSIONS

For DBP, BBP and DEHP, the main potential concern comes from the potential endocrine-disrupting effects of these chemicals. At present, uncertainties are still prominent and special attention in the future should be given to the on-going development and the progress of work in the framework of endocrine disrupters, particularly within the EC and OECD programs. In particular, for DEHP the suspicion of endocrine disruption, in combination with the high production volume and use and the evidence of a widespread occurrence in the environment, has raised reasonable concern in society and emphasises the desirability of the implementation of reduction measures. For DINP and DIPP, there is at present no risk foreseen for the marine environment. As these compounds are not considered PBT, they should be deleted from the List of Chemicals for Priority Action.

8.1.2 OSPAR's role in achieving the desired targets

The results of the risk assessment of the different phthalates (DBP, BBP, DEHP, DINP and DIDP) indicate that, at present, on the basis of the PEC/PNEC ratio in the risk assessment and of present consumption data, the risks for organisms living in the marine environment are negligible to low. The main risk comes from the potential for endocrine disrupting effects of some of these chemicals (DBP, BBP and DEHP).

In order to meet the targets specified in the objectives and timeframe set out by the OSPAR Hazardous Substances Strategy, it will therefore be necessary:

- to assess the need for further reductions from the various sources and the practicability of such reductions;
- to review existing regulations and controls in the light of the need for further reductions;
- to deal with the question of endocrine-disrupting compounds as part of a general approach. Developments in this field are on-going at the EU and OECD level. OSPAR already followed the progress of work. This should be continued;
- to follow the development of consumption of those phthalates in the future.

8.2 Identification of possible measures

8.2.1 Review of existing OSPAR, EU and national measures

8.2.1.1 Measures in OSPAR

No measures have been taken to date.

8.2.1.2 On-going activities within the European Union

Di-n-butyl phthalate (DBP), Di(isononyl) phthalate (DINP) and Di(isodecyl) phthalate (DIDP) were assessed for the risk they might cause to the environment and the human health under the EC Existing Substances Regulation. Final reports are available on the website of the European Chemicals Bureau (ECB - http://ecb.jrc.it). The conclusions on the different risk assessments for the environment were that there is at present no need for further information or testing or risk reduction measures beyond those that are being applied already (conclusion (ii)). However, for DBP, conclusion (iii) was reached for local DBP processing scenarios (PVC production, adhesive production, printing ink usage and glass fibre production). But this has no evident implications for the marine environment.

Butylbenzyl phthalate (BBP) and Di(2-ethylhexyl) phthalate (DEHP) are still undergoing environmental risk assessment under the EU Existing Substances Regulation. For both compounds, conclusions on the question of endocrine-disrupting effects and reprotoxicity still needs to be agreed. At the last EU TCNES (Technical Committee on New and Existing Substances) in March 2005, there was no clear evidence from the available multigeneration fish study that DEHP has endocrine-disrupting potential. For BBP, the results of the long-term fish study on reproductive and endocrine-disrupting effects were not presented. No final decision on the environmental risk assessment of both BBP and DEHP was therefore achieved.

Measures are under development in the EC (Risk Reduction Strategy meetings within the framework of Council Regulation 793/93/EEC) on the distribution and use of phthalates. These, however, are not based upon assessments of the risk to the non-human environment.

WATER FRAMEWORK DIRECTIVE 2000/60/EC

DEHP is on the list of priority substances in the EC WFD. Discussions on the status of DEHP as a priority substance or priority hazardous substance are still on-going. Whichever of these statuses it is given, however, DEHP is of particular concern for the aquatic environment, since EC WFD priority hazardous substances are to be subject to cessation and/or phase-out of discharges, emissions and losses into surface, transitional and coastal waters within 20 years of being placed on the list of such substances, and EC WFD priority substances are to be the subject of measures to reduces discharges, emissions and losses.

COUNCIL DIRECTIVE 76/769/EEC

On the 4th April 2005 the 22nd amendment of directive 76/769/EEC relating to restrictions on the marketing and use of certain dangerous substances and preparations (phthalates in toys and childcare articles) was adopted by the Council. The scope of the ban covers DEHP, DBP and BBP to all toys and childcare articles; DINP, DIDP and DNOP for toys and childcare articles intended for children under three years of age and which can be placed in the mouth by them. In both cases, it has been clarified that the concentration limit of 0,1% of the mass applies to the plasticised material mass, so that in case of items which include both plasticised material and other components, it remains applicable in full to the plasticised part only.

8.2.2 Choice for actions

8.2.2.1 General considerations

When considered in the light of the guidance on the role of marine risk assessment, the results from the risk assessments indicate that there should be concern over some of the phthalates studied: DBP, BBP and DEHP. For these compounds, the main potential concern comes from the potential endocrine-disrupting effects of these chemicals.

At present, uncertainties are still prominent. It is suggested that any potential risk of these chemicals with regard to endocrine disruption that are not covered in the present document should be evaluated in the context of a general approach to endocrine-disrupting substances. Special attention should therefore be given to the on-going developments and progress of work in the framework of endocrine disrupters, particularly within the EC and OECD programmes.

8.2.2.2 Action in the EC

Contracting Parties which are also EU Member States should support the on-going development of the risk assessment report, and provide further information, if available, particularly on endocrine-disrupting effects, in order to refine the assessment.

To support this process and to ensure that the information in this Background Document and the conclusions reached by OSPAR regarding the marine environment are generally taken into account in the approach of the European Community, OSPAR should communicate this Background Document to the European Commission.

8.2.2.3 Action in OSPAR

The main concern regarding phthalates is the potential for endocrine disruption of DBP, BBP and DEHP. Although there are still uncertainties about the potential of endocrine disruption of these compounds, and that monitored levels of phthalates in the environment are relatively low or at least below effect values, this issue should be dealt with in a general approach for endocrine-disrupting compounds. This might be the OECD and/or the EC where work is on-going. Nevertheless, OSPAR should urge the relevant forums - in particular the EC - to start the preparation of an overview of possible reduction measures, including the identification of alternatives to the use of DEHP.

Particularly in respect of DEHP, consumption rates should be observed since it seems that it is currently decreasing.

OSPAR should re-evaluate the risks posed by phthalates when further information becomes available. Any associated measures, which might be justified in the light of new findings, should be addressed through the Background Document review process.

8.2.3 Action in other forums

To ensure that the information in this Background Document can be considered in the context of other international agreements which deal with hazardous substances, and with which Contracting Parties are

associated, OSPAR should send copies of this Background Document to the appropriate bodies dealing with those agreements and invite Contracting Parties who are parties both to OSPAR and those other agreements to promote action to take account of this Background Document by those other international bodies in a consistent manner. In particular, Contracting Parties which are Member States of European Community should pay attention to this in negotiating the regulation of DEHP as a priority substance (or priority hazardous substance) under the EC Water Framework Directive.

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Annex 1: Monitoring strategy for certain phthalates

As part of the Joint Assessment and Monitoring Programme *(reference number 2003-22),* OSPAR 2005 adopted a revised Agreement on Monitoring Strategies for OSPAR Chemicals for Priority Action (*reference number 2004-14)* to implement the following monitoring for tracking progress towards the objectives of the OSPAR Hazardous Substances Strategy *(reference number 2003-21*) with regard to certain phthalates. The monitoring strategy for certain phthalates will be updated as and when necessary, and redirected in the light of subsequent experience.

The Background Document on Phthalates agreed in 2005 concluded that the five phthalates examined DBP, BBP, DEHP, DINP and DIDP did not in general present a risk to the marine environment with the present level of exposure. Furthermore the Background Document concluded that none of the five phthalates met the criteria for being considered as meeting the PBT criteria.

However, the Background Document also concluded that some of the phthalates, namely DBP, BBP and DEHP are potential endocrine disrupting substances but that any risk in this regard should be considered in the context of a general approach to endocrine disrupting substances.

The concern for keeping remote areas free of man-made hazardous substances has not been addressed due to the lack of guidance on how to address this so-called “remote area concern”.

Phthalates have been used for decades in very large volumes e.g. in 1990-1995 around 900,000 tonnes per year (DEHP SIAR 2005). DEHP constitutes a major fraction of this total consumption of phthalates. Resulting from this large use phthalates are ubiquitously present in the environment as evident from a broad range of analytical surveys and monitoring programmes.

A large proportion of the consumption of phthalates has been incorporated into PVC articles, many of them having long service life. Use of such phthalates-containing articles significantly contributes to the total release of these substances to the environment and releases will continue as long as the articles remain in the technosphere or the environment.

Thus, in the light of the historical use of phthalates further build up or decrease of environmental concentration of phthalates cannot be expected unless consumption changes markedly. Consequently, the conclusions of the Background Document that no general risk is expected for the marine environment due to the presence of phthalates will remain valid unless the consumption and releases of phthalates increases.

DEHP is a priority hazardous substance under review within the Water Framework Directive 2000/60/EC. Therefore, monitoring programmes for DEHP, including monitoring in coastal waters, should be operational by end of year 2006. Currently, the Chemical Monitoring Activity under the Common Implementation Strategy of the WFD is developing guidance for such monitoring.

On this background the monitoring strategy is targeted against the consumption of phthalates and not suggesting the need for further analytical environmental monitoring within OSPAR unless there are reasons to believe that environmental concentrations increase. There should be a regular review of the European consumption of phthalates of concern e.g. every three years.

Denmark and France have addressed the European Council for Plasticisers and Intermediates (ECPI) requesting industry to provide information on the consumption of the phthalates of concern in Europe. ECPI has confirmed its willingness to provide information in this regard on a regular basis. Denmark and France will provide HSC with information on the consumption of phthalates as soon as the ECPI information has been reviewed. If no marked increase in consumption of phthalates of concern becomes evident, Denmark and France will suggest that no further action is needed for the time being and furthermore, that the conclusion drawn in the Background Document will remain valid.

CERTAIN PHTHALATES MONITORING STRATEGY	
Implementation of actions and measures	Examination of progress in the implementation of regulations on marketing and/or use or emission and/or discharge which have been agreed, or are endorsed, by the Background Document.
Concentration in air	No monitoring
Discharges and losses to water	No monitoring

CERTAIN PHTHALATES MONITORING STRATEGY	
Production/use/ sales/figures	Collect, with the assistance from industry (European Council for Plasticisers and Intermediates, ECPI) data on consumption of phthalates of concern and assess these data e.g. every three years.
Atmospheric inputs	No monitoring
Riverine inputs	No monitoring
Maritime area:*	
Concentrations in sediments	No monitoring
Concentrations in water	No monitoring
Concentrations in biota	No monitoring

* Where available, data will be periodically compiled from EC WFD monitoring. The choice of matrix will depend on the outcome of the Chemical Monitoring Activity under the Common Implementation Strategy of the WFD.