# *Common Implementation Strategy for the Water Framework Directive*

**Environmental Quality Standards (EQS)**

**Substance Data Sheet**

**Priority Substance No. 12**

# Diethylhexylphthalate (DEHP)

**CAS-No. 117-81-7**

***Final version***
***Brussels, 31 July 2005***

***Disclaimer***

*This data sheet provides background information on the setting of the Environmental Quality Standard in accordance with Article 16 of the Water Framework Directive (2000/60/EC). The information was compiled, evaluated and used as outlined in the Manual [4] and has been discussed in a consultative process with the Expert Advisory Forum on Priority Substances and the Expert Group on Quality Standards. Furthermore, it has been peer-reviewed by the SCTEE[17]. The substance data sheet may, however, not necessarily represent the views of the European Commission.*

*New upcoming information was considered and included up to the date of finalisation of this data sheet. Information becoming available after finalisation of this document will be evaluated in the review process of priority substances according to Art. 16(4) of the Water Framework Directive. If necessary, the Environmental Quality Standard substance data sheets will then be revised in the light of technical and scientific progress.*

# 1 Identity of Substance

| | |
|---|---|
| Priority Substance No: 12 | bis(2-ethylhexyl) phthalate, Diethylhexylphthalate (DEHP) |
| CAS-Number: | 117-81-7 |
| Classification WFD Priority List *: | PSR |

* PS: priority substance; PHS: priority hazardous substance; PSR: priority substance under review according to Decision 2455/2001.

# 2 Proposed Quality Standards

## 2.1 Overall Quality Standards

| **Ecosystem** | **Quality Standard** | **Comment:** |
|---|---|---|
| AA-QS<br>all types of surface waters covered by the WFD | 1.3 µg/l | Prevention of secondary poisoning of predators; see section 8.3 & 8.6. |
| MAC-QS (ECO)* | derivation not possible | No effects up to water solubility of substance; see section 8.1 |

* The proposal by the Commission may include a MAC-QS value which is based on the calculation of 12 * AA-EQS. This derivation is based on the minimum annual frequency of monitoring of priority substances in accordance with the Water Framework Directive. The derivation of such a MAC-QS is based on monitoring, compliance and reporting considerations rather than derived from effect data as presented in this EQS datasheet.

## 2.2 Specific Quality Standards

| **Protection Objective** # | **Quality Standard** | **Comment:** |
|---|---|---|
| Pelagic community (freshwater and saltwater) | derivation of QS not suitable | see section 8.1 & 6.1 |
| Benthic community (freshwater sediment) | 100 mg/kg sediment dry wt | see section 8.2 |
| Benthic community (marine sediment) | no data available | Freshwater sediment QS may be used provisionally. |
| Predators (secondary poisoning) | 3.2 mg/kg (tissue of prey, wet wt)<br>corresponding conc. in water: 1.3 µg/l<br>corresponding conc. in freshwater SPM: 17.2 mg/kg dry wt<br>saltwater SPM: 20.4 mg/kg dry wt | see section 8.3 |
| Food uptake by man | 2920 µg/kg (fishery products, wet wt);<br>corresponding conc. in water:<br>mussel: 1.7 µg/l;<br>fish: 3.5 µg/l | see sections 8.4 & 8.6 |
| Abstraction of water intended for human consumption (AWIHC) | not required | no “guide value” or drinking water standard set in the context of Council Directives 75/440/EEC and 98/83/EC, respectively; setting of such standards is not required; see section 8.5 |

# If justified by substance properties or data available, QS for the different protection objectives are given independently for freshwater environments, transitional waters or coastal and territorial waters

## 3 Classification

| R-Phrases and Labelling | Reference |
|---|---|
| T; R: 60-61(Repr. Cat. 2) | ECB 2001 |

## 4 Physical and chemical properties

| Property | Value: | Ref: | Comments: |
|---|---|---|---|
| Mol. Weight: | 390.6 g/mol | [1] | |
| Water Solubility | 3 µg/l at 20ºC | [1] | |
| Vapour Pressure: | 0.000034 kPa at 20ºC | [1] | |

## 5 Environmental Fate and Partitioning

| Property | Value | Ref: | Comments |
|---|---|---|---|
| Abiotic degradation<br>Hydrolysis<br><br>Photodegradation in the atmosphere<br><br><br><br>Photooxidation in aquatic systems | <br>DT50 = 2000 y<br><br>DT50 = 1 d<br><br><br><br>"very slow" | [1] | <br>estimated<br><br>[1]: reaction with OH-radicals, 25 °C, summary of experimental results and estimations with / without aerosols; used in the assessment<br>used as stabiliser in plastics |
| Biodegradation | Tests on "readily biodegradable": from 4-5% to 60 – 86% mineralisation after 28 days<br><br><br><br><br>Tests on "inherently biodegradable": >95% mineralisation in 5 days in industrial sludge to 86% degradation in 28 days<br><br>Anaerobic conditions: screening data indicate that DEHP is persistent.<br><br>DEHP is assumed to be readily biodegradable | [1] | [1]: Due to the widespread use of DEHP, a major part of the domestic STP sludge can be expected to be adapted to the substance, and the strict requirement of unadapted inoculate for ready biodegradability testing may be difficult to fulfil.<br><br><br><br><br><br><br><br>[1]: Used in the assessment |
| Partition coefficients<br>Octanol – Water<br><br>Koc (organic carbon-water)<br><br><br><br><br>$K_{susp-water}$ (suspended matter-water) | <br>log Kow about 7.5<br><br>63,100 - 888,000 l/kg (with freshwater)<br>589,000 l/kg<br>165,000 l/kg<br><br><br>4,130 $m^3/m^3$ | [1] | <br><br>Experimentally derived<br><br>assessed by EUSES<br>PCKOC model, more in agreement with experimental data and used for EUSES calculation<br><br>TGD default |

| Property | Value | Ref: | Comments |
|---|---|---|---|
| Bioaccumulation<br>Bioconcentration Factor (BCF)<br>Fish<br><br><br>Invertebrate, mussel<br>Invertebrates, amphipods | <br><br>842±105<br><br><br>2,500 wwt<br>2,700 wwt | [1] | <br><br>[1]: based on total $^{14}C$; mean from many experiments<br><br>[1]: besides the fish scenario two invertebrate scenarios are introduced. BCF values are chosen to represent realistic worst case conditions |

## 6 Effect Data (aquatic environment) [1]

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 made at test levels, which exceed the ”molecular” solubility of approx. 3 µg/l. However, stable dispersions are formed up to levels of around 300 µg/l. DEHP readily adsorbs to organic particles and also to various surfaces. In test solutions with concentrations higher than the water solubility, emulsions of micro-droplets of DEHP and surface films may be formed. This may cause unstable test solutions where the bioavailable fraction is lower than the nominal concentration, and the exposure of the organism cannot be correctly quantified. Formation of micro-droplets or surface films may also contribute to effects by direct physical interference, e.g. entrapment at the surface (flotation) or obstruction of the gas flow over the gills. Judging from toxicity tests with Daphnids where problems with solubility have been reported, the ”apparent water solubility” in the tests seems to be roughly in the order of 0.1 mg/l. Above this approximate level test solutions seem not to be stable and solubility-related problems start to arise, e.g. floaters.

In the environment, DEHP is likely to be sorbed to any suspended particles in natural waters and the presence of dissolved organic material may furthermore increase the apparent water solubility. This may lead to a higher apparent water solubility of DEHP than predicted from the physico-chemical properties. The adsorption of lipophilic substances onto particles and colloids may either decrease or increase the bioavailability and thus the toxicity. For most substances, particles and colloids probably decrease the bioavailability, but for certain types of organisms (especially suspension feeders or detritivores) the reverse fact might be true. As DEHP tends to accumulate in sediments, the evaluation of toxicity to organisms living in or on sediment is essential.

### Toxicity to fish [1]

From the effect studies on fish it can be concluded that DEHP have no acute effects at exposure levels far exceeding its apparent water solubility.

No significant mortality was seen in the long-term toxicity studies with juvenile and adult fish. However, there are indications that DEHP may have effects on growth at relatively high exposure concentrations. Defoe *et al* (1990) noted a statistically significant weight reduction of 13% when juvenile Japanese medakas were exposed to a mean measured concentration of 0.554 mg DEHP/l for 168 days. When fertilised rainbow trout eggs and resulting fry were exposed to DEHP for a total of 90 days a weight reduction of ca 10% was observed at 0.259 and 0.502 mg/l which were the two highest exposure levels. These weight reductions were not statistically significant (Defoe *et al*, 1990) but may be an indication of effects on growth. The slightly impaired growth in these studies may be an effect of physical influence of the test substance as the test concentrations were well above the “true” water solubility. On the other hand the effects on growth may be a result of DEHP

affecting the collagen synthesis in fish. Mayer *et al,* (1977) observed effects on collagen synthesis at exposure levels as low as 0.004 mg/l when three different fish species were exposed to DEHP at concentrations up to 0.1mg/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. Therefore, these results are not considered relevant to use in the risk assessment.

In the embryo larval studies effects were indicated at lower exposure levels and the most sensitive life stage seems to be the period between hatch and swim up (yolk adsorption). The lowest NOEC is 0.005 mg/l for rainbow trout (Mehrle & Mayer, 1976). However, the results from this study are not considered valid for the purpose of risk assessment as discussed earlier (cf. section 3.2.1.1.1 of [1]). In a semi-static study with channel catfish (Birge *et al*, 1978) the mortality was 10% at a nominal concentration of 0.1 mg/l. However, it is uncertain if the effects seen are due to the intrinsic toxicity of DEHP as effects were seen only at nominal concentrations well above the true water solubility for fresh water. Another reservation for this study is that the carrier solvent concentration differed between the different DEHP exposure levels while the solvent concentration of the control was not stated.

No firm conclusions can be drawn from the few studies where the effects of DEHP on biochemical parameters have been studied. Effects on collagen and hydroxyproline synthesis have been demonstrated. The dose-response relationship was lacking or weak and no effects on growth were seen. Furthermore, it seems like DEHP has slight effects on lipid metabolism and steroid synthesis when administered via the feed at relatively high concentrations. The biological significance of these findings is uncertain.

In conclusion, there is no reliable long-term study indicating effects below the “apparent” water solubility of DEHP. Therefore, it is not considered suitable to specify a chronic NOEC for fish exposed via water.

### Toxicity to aquatic invertebrates [1]

An overall conclusion regarding the toxicity to aquatic invertebrates exposed via water in terms of specifying a NOEC-value for use in the risk assessment is bound up with problems. There are several indications that the effects observed in the toxicity tests with Daphnia could be caused by physical effects, which probably have no relevance in the environment. There are also indications that DEHP has no shown genuine toxic effect in concentrations up to and markedly exceeding the water solubility (neither the ”true” solubility predicted from the physico-chemical properties nor the ”apparent” solubility found in some toxicity tests).

Based on the present data it is considered not feasible to determine a level of toxicity for DEHP to aquatic invertebrates exposed via water. Accordingly, for the purpose of this risk assessment the last option is considered legitimate to use. Hence it is not possible, for the time being, to state a $NOEC_{water}$ for aquatic invertebrates.

### Toxicity to algae [1]

From studies of toxicity on algae and higher plants it can only be concluded that it is impossible from the current data to determine whether any effects observed in the toxicity tests may be relevant to use for derivation of a PNEC for water.

Hence, from the available data, it is not possible to state a $NOEC_{water}$ for algae and higher plants.

### Toxicity to amphibians [1]

The studies indicate that amphibians might be sensitive to DEHP at high concentrations. But since the reported effect concentrations (of which only one is measured) are above both the 'true' as well as the 'apparent' water solubility, they cannot be used in order to derive a $PNEC_{water}$ for this risk assessment.

### Toxicity to sediment organisms [1]

Although many questions remain regarding the different sediment studies, the results taken together indicates that effects of DEHP in the sediment compartment may arise at concentrations around 1mg/kg (dw).

### Summary on endocrine disrupting potential

| | Reference |
|---|---|
| Substance with evidence of ED or evidence of potential ED, already regulated or being addressed under existing legislation | [2] |
| Possible effects of DEHP on the normal functioning of the endocrine system have been investigated in a number of studies summarised in section 4.1.2.9.3 (of [1]). Both in vivo and in vitro study results indicate that DEHP can interfere with the endocrine function and also influence the sexual differentiation. Due to the effects on the Leydig cells as measured by a decreased testosterone output, it cannot be excluded that DEHP may exert an antiandrogen effect. The results of recently performed in vivo studies in rats exposed to DEHP or DBP support the hypothesis that exposure to phthalates may provoke an antiandrogen mechanism (Gray et al., 1999, Mylchrest and Foster, 1998; full ref. in [1]).<br>Norrgren *et al,* 1999 (full ref. in [1]) studied the effects on sexual differentiation in Atlantic salmon (S*almo salar*) exposed to DEHP. The control group consisted of 49% females. The number of females in the groups fed DEHP contaminated food was similar to the control in the low dose (47% females, 300 mg DEHP/ kg food dwt) while in the high dose it was significantly higher (64% females, 1,500 mg/kg food dwt).<br>In addition to the feeding trial juvenile salmon weighing approx. 7.5 g were injected intraperitoneally with different test compounds in order to study induction of vitellogenin synthesis. The group injected with DEHP received a total dose of 160 mg $kg^{-1}$ during 17 days. At termination of the exposure period blood was sampled from the fish and analysed for vitellogenin. No vitellogenin was detected in the blood of the DEHP injected fish. | [1] |

## 6.1 Predicted No Effect Concentrations (aquatic environment)

### 6.1.1 Calculation of PNEC surface water [1]

Short term and/or long-term effect studies, where the test organisms are exposed to DEHP via water, are available for fish, amphibians, aquatic invertebrates, algae, higher plants, and micro-organisms. However, there are no reliable long-term studies below the apparent water solubility of DEHP indicating effects on organisms exposed to DEHP in water. Therefore, it is not considered suitable to specify a chronic NOEC for organisms exposed via water.

Hence a $PNEC_{water}$ cannot be specified.

### 6.1.2 Calculation of PNEC sediment [1]

The NOEC of >1000 mg/kg derived from the 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).

Since no $PNEC_{water}$ could be determined, the equilibrium partitioning method, given in TGD, cannot be used to estimate $PNEC_{sediment}$.

$PNEC_{sediment}$ = > 100 mg/kg (dwt)

### 6.1.3 Calculation of the PNEC for non compartment specific effects relevant for the food chain (secondary poisoning)

#### Toxicity to fish

At the Technical Meeting 2 in the year 2000 (TM'2 2000) it was decided that a multi-generation fish study with exposure both via food and water was needed in order to properly assess the aquatic toxicity of DEHP. The study was completed in august 2004 (Caunter et al., 2004 [12]) and evaluated along with a number of other recently performed studies on the effects of DEHP on fish in an addendum to the RAR [13]. One of these latter studies [15] is a follow-up study to the study from which the so far used $PNEC_{oral,fish}$ of 6 mg DEHP/kg (wwt) for exposure of fish via the food was derived [16].

With respect to the new studies, it was concluded by the rapporteur (SE) and agreed by TEC NES I 2005 [14] that it is hard to draw firm conclusions regarding possible effects of DEHP from the study by Caunter et al. [12] and, therefore, it is not possible to derive a NOEC from the study. However, in another study with Atlantic salmon [15] a small but statistically significant increase on ovotestis was observed at an (analytically confirmed) exposure level of 1500 mg DEHP / kg food. The NOEC was 800 mg DEHP/kg. The effects observed in this study was weaker than in the previous study by Norrgren et al. on Atlantic salmon [16] where an effect on the sex ratio was observed. No analytical confirmation of the exposure concentrations was made in the earlier study, which may have been higher thus explaining the difference in response between the two studies. Based on the results of the two studies it was proposed by the rapporteur and agreed by TC NES I 2005 [14] to substitute the so far used $NOEC_{fish}$ of 300 mg DEHP/kg food with the NOEC of 800 mg DEHP / kg food for the derivation of a **$PNEC_{fish,oral}$ of 16 mg/kg food (wwt)** [1].

#### Toxicity to birds [1]

In a study on hens exposed to DEHP for 28 days decreased egg production (14 %) and effects on lipid metabolism were found at the lowest test concentration, LOEC = 10,000 ppm (Wood & Bitman 1980) An effect level of 14 % fulfils the prerequisite for dividing the LOEC by two, resulting in a NOEC of 5,000 ppm. The feed in this study was a standard laying mash (consisting mainly of different meals). In TGD (appendix VII) it is stated that the energy content of grain is higher than fish. This means that in order to obtain the same amount of energy more wet weight of fish must be consumed compared to grain. Therefore a correction factor of 3 may be applied for the difference

[1] The commercial food used in these studies was a very high quality food. The approximate food conversion from this diet is 1: ingestion of 1 g of food results in 1 g of increase in body weight. The corresponding food conversion from natural diets is 0.2: 1 g food gives 0.2 g increase in body-weight. This difference is in part due to the difference in water content between dry pelleted food and natural food. To take account of this, the NOEC is recalculated to wet weight basis using a factor of 5. The NOEC for natural diets then becomes 160 mg DEHP/kg food (wwt). Applying an assessment factor of 10 leads to a $PNEC_{oral,fish}$ of 16 mg/kg (fresh food).

in caloric content of the diet of laboratory animals and the diet of fish-eating birds or mammals". Hence, the NOEC can be lowered with a factor 3 resulting in a NOEC of about 1 700 mg/kg food.

Strictly applying the TGD would lead to the use of an assessment factor of 10 on the NOEC in order to derive a PNEC . However, there is also a long-term study (230 days exposure) in which egg laying was totally impaired at a dose of 5000 ppm. From this study no NOEC can be derived but the results imply that a larger assessment factor than 10 is needed for the derivation of PNEC from the 28 day study. Therefore, an assessment factor of 100 is chosen resulting in a **$PNEC_{bird,oral}$ of 17 mg/kg food**. This PNEC will be used in the risk characterisation for secondary poisoning of birds feeding on mussels.

### Toxicity to mammals

In the current draft (September 2001) of the environmental risk assessment of DEHP the PNEC for secondary poisoning is set to 5 mg/kg food [1]. The PNEC was derived from a 13 weeks dietary study on rats (Poon et al., 1997). In this study the NOAEL for testicular effects (Sertoli cell vacuolisation) was 50 mg/kg food. Furthermore, irreversible testicular damage was shown in male pups *in utero* and during suckling, LOAEL = 3.5 mg/kg b.w. (Arcadi et al., 1998). This LOAEL was recalculated to dose per kg body weight using a conversion factor of 15, resulting in a LOAEL for food at about 50 mg/kg b.w. Using an assessment factor of 10 according to the "old" TGD results in a PNEC of 5 mg/kg. This PNEC was agreed by the TM.

Since then the Human health section of the risk assessment has been revised and the NOAEL for reproductive and developmental effects have been changed (and agreed by the TC NES) [8]. The current NOAEL for testicular effects is 4.8 mg/kg b.w. based on a three generation study in rats (Wolfe et al., 2003; for motivation of choice of this NOAEL see section 7).

The rationale for using this NOAEL in the assessment of secondary poisoning is that effects on the male reproductive organs may give effects also on the population level. Furthermore, as TM agreed the formerly used NOAEL from the Poon study for deriving a $PNEC_{oral}$ the rapporteur sees no reason why this new NOAEL for the same effect should not be accepted. [8]

A recalculation of the NOAEL using the conversion factor of 20 suggested by TGD leads to a $NOEC_{food}$ of 96 mg/kg. Applying the assessment factor of 30 as suggested in the current TGD gives a **$PNEC_{mammal,oral}$ of 3.2 mg/kg**. [8]

### Biomagnification

Regarding the possible biomagnification of DEHP, this is not dealt with in the September 2001 draft of environmental risk assessment. The next revision of the document will include such an assessment. The rapporteur does not think that biomagnification is an issue for DEHP [8].

First of all monitoring data from biota do not indicate higher DEHP levels in predators compared to animals from the lower region of the food web. Secondly, from toxicokinetic studies on mammals it is clear that DEHP is relatively rapidly metabolised and does not accumulate in mammals [8]. Furthermore recent data by Mackintosh et al. (2004 [10]) indicate that DEHP is not biomagnifying. Regression analysis of the measured log lipid equivalent concentration versus trophic level data for DEHP yields a Food-Web Migration Factor of 0.34. This means that the concentration at a given trophic level is 34% of that at previous trophic level [9]. Studies by Norman et al. [11], where Atlantic Salmon were exposed to DEHP via the food for 4 weeks and by Caunter et al. [12], where Fathead minnows were exposed in two generations both via water and via food, revealed as well lipid normalised BMF-factors far below 1, which indicates that the potential for biomagnification of

DEHP is limited[2]. All in all, this indicates that biomagnification should not be considered when $QS_{sec.\ pois.}$ is calculated (i.e. a BMF of 1 should be used) [8].

**PNECs**

| Compartment | Value | Reference |
|---|---|---|
| Surface water | cannot be specified | [1] |
| Sediment | > 100 mg/kg dry weight | [1] |
| $PNEC_{oral}$ (secondary poisoning)<br>fish<br>birds<br>mammals | <br>16 mg/kg food<br>17 mg/ kg food<br>3.2 mg/kg food | <br>[13, 14]<br>[1]<br>[8, 9] |

## 7 Effect data (human health)

The critical NOAEL in the consolidated final report of the human health part of the DEHP-RAR [7] is 4.8 mg/kg bw for testicular and developmental toxicity based on a three generation study in rats (Wolfe et al. 2003 [full ref. in the RAR]). This NOAEL is agreed by TC NES and its choice motivated in the following way in the human health risk assessment [8]:

"However, as there remain some doubts as to the toxicological significance of the sertoli cell vacuolisation observed in the Poon study, a **NOAEL of 4.8 mg/kg/day** (100 ppm) is chosen from the Wolfe study (2003) for the risk characterisation, based on occurrence of small male reproductive organs (testis/epididymes/seminal vesicles) and minimal testis atrophy (exceeding those of the current controls as well as historical control groups) at 300 ppm and above."

| Effect | Quantitative data |
|---|---|
| Chronic toxicity | NOAEL: 28.9 mg/kg/d<br>Species: rat<br>Source: Moore et al, 1996 |
| Fertility<br>(Testicular effects)<br><br><br>Fertility | NOAEL: 4.8 mg/kg bw/d (100 ppm in food)<br>Species: rat<br>Source: Wolfe et al., 2003<br><br>NOAEL: 20 mg/kg bw/d (100 ppm in food)<br>Species: mouse<br>Source: Lamb et al., 1987 |
| Developmental<br>Toxicity | NOAEL: 4.8 mg/kg bw/d (100 ppm in food)<br>Species: rat<br>Source: Wolfe et al., 2003 |

[2] A third study by Brown et al., corroborating the findings of the cited papers on the BMF of DEHP, is currently being drafted [9].

Further non-quantitative information [1]:

| **Effect** | **Summarised information** |
|---|---|
| Acute oral toxicity | Various studies with mammals; no effect value below 980 mg/kg; highest value > 40000 mg/kg.. |
| Conclusion: acute toxicity: | The acute toxicity of DEHP seems to be very low. |
| Conclusion: mutagenity: | Most test results are negative. Positive results are obtained using test-systems which also react on non genotoxic agents.<br>DEHP and metabolites are classified as not being genotoxic. |
| Conclusion cancerogenity : | "The relevance for humans of the liver tumours in rodents induced by DEHP is regarded to be negligible. Also the relevance of the DEHP-induced MCL in F344 rats is questionable. On the other hand, the induction of LC tumours in rats exposed for DEHP should be regarded as relevant to humans and, therefore, a careful evaluation of the orignal data of Berger (1995) is necessary before concluding the possible carcinogenic risk of DEHP."<br>Based on the overall evaluation of the available data, no classification for carcinogenicity is proposed. |
| Estrogenic activity | An anti-androgen effect as basis for other effects is assumed. |
| Conclusion: reproductive toxicity: | "The results of recently performed *in vivo* studies in rats exposed to DEHP or DBP support the hypothesis that exposure to phthalates may provoke an antiandrogen mechanism. The present data in experimental animals are of concern for humans."<br>Classification according to R 60 and R61 |

## 8. Calculation of Quality Standards

### 8.1 Quality Standards for Water

In the RAR [1] it is stated that there are no reliable studies below the apparent water solubility of DEHP indicating effects on organisms exposed to DEHP in water. For this reason it is not considered suitable to specify a PNEC for exposure via water.

Consequently, this conclusion entails that the derivation of a quality standard for exposure via water is also not suitable. Therefore, no quality standard for the protection of the pelagic communities in inland waters or in transitional, coastal and territorial waters is derived.

### 8.2 Quality Standard for Sediment

A PNEC > 100 mg/kg sediment (dry weight) is derived in the risk assessment [1] (see also section 6.2 of this data sheet). This value is adopted and the $QS_{sediment}$ set to:

**$QS_{sediment}$ = 100 mg DEHP / kg sediment (dry wt)**

The equilibrium partitioning method can be used to calculate a tentative corresponding water concentration:

$$QS_{water}\ [mg.l^{-1}] = \frac{QS_{sediment.wet}\ [21.7\ mg/kg]}{1000} * \frac{bulk\ density_{SPM.wet}\ [1{,}150\ kg/m^3]}{Kp_{susp\text{-}water}\ [4{,}130\ m^3/m^3]}$$

with:

$QS_{sediment.wet}$ 100 mg/kg / 4.6 (wet:dry ratio) = 21.7 mg/kg (The TGD defines wet SPM as 90% vol/vol water (density 1 kg/l) and 10% vol/vol solids (density 2.5 kg/l), thus giving a wet density of (0.9 × 1) + (0.1 × 2.5) = 1.15 kg/l. The dry weight of solids is therefore 0.25 kg (per litre wet SPM) and thus the wet:dry ratio is 1.15/0.25 = 4.6)

$Kp_{susp\text{-}water}$ see secion 5 of this data sheet

bulk $density_{SPM.wet}$ = 1,150 kg/$m^3$ (default value TGD)

1,000 = conversion factor $m^3$/kg to l/kg

Tentative $QS_{water}$ = 6 µg DEHP /l

The tentative value derived by the EP-method is above the water solubility of DEHP.

### 8.3 Secondary Poisoning of Top Predators

$PNECs_{oral}$ for fish (16 mg/kg food) and birds (17 mg/kg food) are identified in the "Addendum on Fish toxicity" [13] and the environmental part of the DEHP risk assessment [1], respectively. The mammalian $PNEC_{oral}$ was revised on the basis of the 2004 update of the human health part of the risk assessment [7, 8]. As a result of this update, the critical NOAEL for mammalian toxicity has been changed to 4.8 mg/kg bw for testicular and developmental toxicity in rats (see section 7). According to the Swedish Rapporteur for DEHP, this consequently leads to a change in the $PNEC_{oral,\ mammal}$. Following the provisions of the TGD for PNEC derivation from a NOAEL, the Rapporteur calculated a $PNEC_{oral,\ mammal}$ of 3.2 mg/kg food (see section 6.1.3).

The lowest PNEC is used for the derivation of a quality standard for prey (biota tissue) with respect to secondary poisoning of top predators as objective of protection.

**$QS_{secpois.biota}$ = 3.2 mg DEHP / kg prey (wet weight)**

Since DEHP is readily adsorbed onto organic surfaces and particles in the water column and to sediment, the highest bioaccumulation factors are obtained for zooplankton (Acartia sp.) with a high surface/weight ratio, for Gammarus sp., a sediment dwelling amphipod, 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.

Because fish show relatively low BCF values compared to invertebrates, invertebrate eating animals are probably a more critical target group. Therefore, in the RAR [1] the following BCF values are recommended to represent / assess realistic worst case conditions: 840 (fish as prey); 2500 (mussel as prey); 2700 (crustaceans as prey)

New data evaluated by the Swedish Rapporteur for the DEHP RAR indicate that DEHP does not biomagnify up the aquatic food web [8, 9, 10, 11, 13]. Hence, in accordance with the recommendation by the Rapporteur-MS (see section 6.1.3), biomagnification is not considered for the calculation of the $QS_{secpois.water}$.

This results in the following QSs for freshwater and saltwater:

| | | | |
|---|---|---|---|
| $QS_{secpois.water}$ | = 3.2 [mg/kg] / BCF 840 | = | 3.8 µg DEHP /l (fish as prey) |
| **$QS_{secpois.water}$** | **= 3.2 [mg/kg] / BCF 2500** | **=** | **1.3 µg DEHP /l (mussels as prey)** |
| $QS_{secpois.water}$ | = 3.2 [mg/kg] / BCF 2700 | = | 1.2 µg DEHP /l (crustaceans as prey) |

Thus, protection of predators from secondary poisoning requires lower quality standards than the "true" water solubility of DEHP.

Because mammals that almost exclusively prey on small pelagic or benthic crustaceans are unknown, it appears justified in account of the precautionary principle to choose the $BCF_{mussel}$ for the calculation of the concentration in water that corresponds to the $QS_{secpois.biota}$ (≈ $PNEC_{food}$ for top predators). Bioaccumulation in aquatic insects is much lower (in the range of the $BCF_{fish}$) [1] than in crustaceans so that mammals like water shrews (e.g. *Neomis fodiens)* feeding on a mixed diet of small water animals such as insect larvae, crustaceans, molluscs, amphibia etc. should be protected by the mussel scenario. The $PNEC_{oral}$ of birds and fish is much higher than that derived for mammals (see section 6.1.2). Therefore, representatives of these groups preying predominantly on crustaceans will be protected by the $PNEC_{oral.mammal}$ / $BCF_{mussel}$ scenario as well.

As the log $Kp_{susp}$ is >3, the $QS_{secpois.water}$ is additionally given as concentration in SPM of the TGD standard water (15mg/l SPM in freshwater, 3 mg/l SPM in marine water; see section 4.3.1 of the Manual [4]):

$$\mathbf{QS_{secpois.SPMfreshw}} = \frac{QS_{secpois.water}\ [1.3\ \mu g/l]}{C_{SPM}\ [15\ mg/l] * 10^{-6}\ [kg/mg] + Kp^{-1}\ [(16{,}500\ l/kg)^{-1}]} = \mathbf{17.2\ mg/kg}\ \text{SPM (dry wt)}$$

$$\mathbf{QS_{secpois.SPMsaltw}} = \frac{QS_{secpois.saltwater}\ [1.3\ \mu g/l]}{C_{SPM}\ [3\ mg/l] * 10^{-6}\ [kg/mg] + Kp^{-1}\ [(16{,}500\ l/kg)^{-1}]} = \mathbf{20.4\ mg/kg}\ \text{SPM (dry wt)}$$

### 8.4 QS referring to food uptake by Humans

The lowest value identified in the risk assessment [7, 8] as relevant for the assessment of impacts on human health is a $NOAEL_{oral}$ of 4.8 mg/kg bw $d^{-1}$ for developmental toxicity in rats.

If the usual assessment factor of 100 is applied to extrapolate from animal to man the $NOAEL_{oral.human}$ is 48 µg/kg bw $d^{-1}$ (≈ 3360 µg $d^{-1}$ for a person with 70 kg body weight as relevant threshold level).

In the Manual [4] (section 4.3.2.6) it is suggested that the relevant threshold level may not be exhausted for more than 10% by consumption of food originating from aquatic sources (i.e. 336 µg $d^{-1}$).

The average fish consumption of an EU citizen is 115 g $d^{-1}$ (TGD [3]). Thus, 115 g fish (or fishery products) must not contain more than 336 µg DEHP.

$$\mathbf{QS_{hh.food}} = \frac{336\ \mu g\ DEHP}{115 g\ fish\ consumption} * 1000\ g = \mathbf{2920\ \mu g\ DEHP\ /\ kg\ fishery\ products}$$

Given the $BCF_{fish}$ mentioned in the risk assessment [1] (842 l/kg, biomagnification is apparently not relevant [8, 9]), a tissue concentration of 2920 µg DEHP per kg fish results in a water concentration of:

$$\mathbf{QS_{hh.food.water}} = \frac{2920\ \mu g/kg}{BCF\ (842\ l/kg)} = \mathbf{3.5\ \mu g\ DEHP\ /\ l}$$

The $BCF_{mussel}$ is considerably higher than the $BCF_{fish}$. 2500 was identified in the risk assessment [1]. With the $QS_{hh.food}$ of 2920 µg/kg, the $BCF_{mussel}$ of 2500 this results in a corresponding water concentration of 1.7 µg/l.

Thus, the quality standard required to protect human health form adverse effects due to the ingestion of fishery products contaminated by DEHP may only slightly be higher than the standards required to protect predators from secondary poisoning. However, the QS derived for the protection of predators from secondary poisoning might also protect humans from adverse health effects due to the ingestion of fishery products.

## 8.5 QS for drinking water abstraction

No "guide values" or quality standards have been set in the context of Council Directives 75/440/EEC or 98/83/EC. Therefore, a provisional drinking water quality standard is calculated based on the recommendations given in the TGD (section 4.3.3 of the Manual [4]).

The lowest relevant value identified in the risk assessment is a $NOAEL_{oral}$ of 4.8 mg/kg bw $d^{-1}$ [7, 8]. If the usual assessment factor of 100 is applied to extrapolate from animal to man the $NOAEL_{oral.human}$ is 48 µg/kg bw $d^{-1}$.

The provisional quality standard for drinking water is calculated with the provision that uptake by drinking water should in any case not exceed 10% of the threshold level for human health [3].

$$QS_{DW.provisional} = \frac{0.1 * TL_{HH} * BW}{Uptake_{DW}} = < 168\ \mu g\ DEHP\ /l$$

with:

| | |
|---|---|
| $QS_{DW.provisional}$ | provisional quality standard for drinking water (mg/l) |
| $TL_{HH}$ | threshold level for human health (48 µg DEHP /kg bw per day) |
| BW | body weight (70 kg) |
| $Uptake_{DW}$ | uptake drinking water (2 l per day) |

Overall, it can be concluded that a quality standard addressing drinking water abstraction is not required as the standards necessary to protect human health and top predators from adverse effects due to ingestion of food originating from aquatic environments provide a sufficient margin of safety for drinking water uptake.

## 8.6 Overall Quality Standard

As consequence of its very low water solubility, DEHP does apparently not exert direct toxic effects on the pelagic communities of freshwater and saltwater ecosystems, respectively. However, due to its bioaccumulation potential DEHP could pose top predators or human health at risk (uptake of contaminated fishery products). In order to avoid such risks for predators and humans,

respectively, a very low quality standard in the range of the "true" water solubility of DEHP is required.

The proposed overall annual average quality standard of 1.3 µ/l is referring to secondary poisoning of predators. It is also stringent enough to protect human health form adverse effects upon the ingestion of fishery products contaminated by DEHP. Possible effects of DEHP on endocrine regulation should be covered by these standards as well, because the $PNECs_{food}$ used for their calculation cover effects on development, reproduction and sexual differentiation.

## 9. References

[1] European Union Risk Assessment Report: bis(2-ethylhexyl)phthalate, CAS No: 117-81-7, , Consolidated Final Report: September 2001. (Files: R042_0109_env_hh_0-3.doc (Chapters 0-3); R042_0109_env_hh_4-6.doc (chapters 4-6)). The (final) report will be available from the internet-site of the European Chemicals Bureau: http://ecb.jrc.it/existing-chemicals/

[2] COM(2001)262 final: Communication from the Commission to the Council and the European Parliament on the implementation of the Community Strategy for Endocrine Disrupters – a range of substances suspected of interfering with the hormone system of humans and wildlife.

[3] Technical Guidance Document on Risk Assessment in Support of Commission Directive 93/67/EEC on Risk Assessment for New Notified Substances and Commission Regulation (EC) No 1488/94 on Risk Assessment for Existing Substances and Directive 98/8/EC of the European Parliament and the Council Concerning the placing of biocidal products on the market. Part II. European Commission Joint Research Centre, EUR 20418 EN/2, © European Communities 2003. Available at the internet-site of the European Chemicals Bureau: http://ecb.jrc.it/existing-chemicals/

[4] Manual of the Methodological Framework Used to Derive Environmental Quality Standards for Priority Substances of the Water Framework Directive. Peter Lepper, Fraunhofer-Institute Molecular Biology and Applied Ecology, 15 November 2004.

[5] Technical Guidance Document on Risk Assessment in Support of Commission Directive 93/67/EEC on Risk Assessment for New Notified Substances and Commission Regulation (EC) No 1488/94 on Risk Assessment for Existing Substances and Directive 98/8/EC of the European Parliament and the Council Concerning the placing of biocidal products on the market. Part II. European Commission Joint Research Centre, EUR 20418 EN/2, © European Communities 2003

[6] Personal communication. E-mail by Lars Andersson, Swedish National Chemicals Inspectorate, 14.05.2003

[7] European Union Risk Assessment Report: bis(2-ethylhexyl)phthalate, CAS No: 117-81-7, , Consolidated Final Report: February 2004. Chapters 4-6 (File: R042_04102_env_hh_4-6.doc)

[8] Personal communication. E-mails by Lars Andersson, Swedish National Chemicals Inspectorate, 17.03.2004 and 30.06.2004

[9] Updating the Water Framework Directive Substance Data Sheet for Di(2-ethylhexyl) phthalate (DEHP). Position Papers by ECPI submitted after EAF 6 and after EAF 7.

[10] Mackintosh CE, Maldonado J, Hongwu J, Hoover N, Chong A, Ikonomou MG and Gobas FA., Distribution of phthalate esters in a marine aquatic food web: comparison to polychlorinated biphenyls. Environmental Science and Technology (2004) 38(7):2011-20.

[11] Norman A, H Börjeson, F David, B Tienport and L Norrgren: Dietrary exposure of atlantic salmon (Salmo salar) to di-2-ethylhexyl phthalate (DEHP) – kinetics and gonad morphology. Sumbmitted to the Journal of Environmental Toxicology and Chemistry.

[12] Caunter JE, TD Williams, N Shillabeer (2004): Di-2-Ethylhexylphthalate: Multi-generation study with the fathead minnow (Pimephales promelas). Study No. AJ0172. Performed by Brixham environmental laboratory Astra Zeneca UK Ltd, sponsored by European Council for Plasticisers and Intermediates.

[13] DEHP – Addendum on fish toxicity for TC NES I, 2005. File: TEST042_env_S2.doc, 2005-02-09

[14] Draft minutes on DEHP; TEC NES I 2005. File: mi_042_tc0501_env_dr1-1.doc.

[15] Norman A (2003): Dietary exposure of Atlantic salmon (Salmo salar) to di-2-ethylhexyl phthtalate (DEPH) – kinetics and morphology. Manuscript.

[16] Norrgren, L, A Blom, PL Andersson, H Börjesson, DGJ Larsson, and P-E Olsson, (1999): Effects of potential xenoestrogens (DEHP, nonylphenol and PCB) on sexual differentiation in juvenile Atlantic salmon (*Salmo salar*). Aquatic Ecosystem Health and Management, Vol 2/3. pp.311-317

[17] Opinion of the Scientific Committee on Toxicity, Ecotoxicity and the Environment (SCTEE) on "The Setting of Environmental Quality Standards for the Priority Substances included in Annex X of Directive 2000/60/EC in Accordance with Article 16 thereof", adopted by the CSTEE during the 43rd plenary meeting of 28 May 2004, European Commission Health & Consumer Protection Directorate General, Brussels. http://europa.eu.int/comm/health/ph_risk/committees/sct/documents/out230_en.pdf