Environmental hazard assessment: Di-(2-ethylhexyl)phthalate
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This environmental hazard assessment document was prepared for the Toxic Substances Division of the Directorate for Air, Climate and Toxic Substances in the Department of the Environment. The document is intended to help develop an understanding of the behaviour of a chemical in the environment. It is a contribution towards government policy on the chemical concerned but is not intended to be a statement of that policy.

The document reviews and assesses the distribution, fate and effects of chemicals in the environment. Potential routes to man are included in the assessment but are not studied exhaustively. Effects on man are not within the scope of the assessments.

In one section of the document, a simple model has been used to predict the environmental distribution of the chemical. The data provided by the model are compared with the other available information to help identify where more data may be needed. The model only gives a crude estimate of the environmental distribution.

Information in the document was collected and evaluated by the Building Research Establishment and the Institute of Terrestrial Ecology. The document was circulated in draft form and has been revised to take account of comments on the draft. These comments were submitted by a number of interested organisations and their contribution is very much appreciated.
Summary

**Properties:** Di-(2-ethylhexyl)phthalate (DEHP) is a non-volatile liquid which is used extensively in the plastics processing industry as a plasticiser (increasing the flexibility of plastics, mainly PVC).

**Production:** Nine suppliers of phthalic acid esters were identified in the United Kingdom, but no information was available on the actual number of sites of DEHP production in this country. An estimated 33,000 tonnes of octyl phthalates, i.e. DEHP and di-isooctyl phthalate, were produced in the UK in 1989, and an estimated 66,000 tonnes of octyl phthalates were used in the UK in the same year.

**Release:** DEHP is released into the UK environment as a result of its production and its later use in plastics processing. DEHP will also be released from plasticised products during their use and after their disposal. This release pattern ensures that DEHP is distributed throughout the whole environment. A worst case annual release of 1,220 tonnes DEHP was estimated for the UK from all sources. Any natural sources of DEHP were considered to be negligible compared to anthropogenic sources.

**Fate:** The fate of DEHP in the environment is influenced by its low volatility and its high potential for accumulation. Biodegradation of DEHP occurs fairly rapidly under aerobic conditions; it is not thought that abiotic processes (hydrolysis, photolysis etc) contribute greatly to the overall removal of DEHP from the environment. DEHP readily accumulates in aquatic sediments, suspended particulate material, and in the lipid tissues of aquatic biota. DEHP is also strongly held by soil solids and organic material in the terrestrial environment.

**Effects:** The acute toxicity of DEHP to aquatic organisms is generally low, with 96h LC50s of > 10 mg/l, although more recent acute toxicity values have been found which are lower than this; more sensitive indicators, such as reproduction and biochemical changes, have also been affected at lower concentrations. The acute toxicity of DEHP is low for micro-organisms, amphibians, plants, birds and mammals. The maximum measured environmental levels of DEHP in freshwater are greater than those at which both acute effects and marginal chronic effects on aquatic organisms have been observed in laboratory studies. It is therefore possible that both acute and chronic effects on aquatic organisms may be observed as a result of DEHP exposure in the real environment. Elevated levels of phthalates have been measured on sediments, and adverse effects may be noted on sediment-dwelling organisms.
Summary

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1. INTRODUCTION

1.1 Regulatory action

Di-(2-ethylhexyl)phthalate (hereafter referred to as DEHP) is not one of the chemicals identified by the Commission of the European Communities as being a potential List I compound under the Dangerous Substances Directive (76/464/EEC), nor does it appear on the UK Red List of priority water pollutants or in the priority candidate category for inclusion on the UK Red List.

1.2 Physico-chemical properties

DEHP is a colourless or yellow oily liquid; it is the diester formed between 1,2-benzenedicarboxylic (o-phthalic) acid and 2-ethylhexanol. Some of its physico-chemical properties are listed in Table 1.1 below.

<table>
<thead>
<tr>
<th>Property</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>CAS Registry Number</td>
<td>117-81-7</td>
</tr>
<tr>
<td>Molecular Weight</td>
<td>391.0</td>
</tr>
<tr>
<td>Boiling Point</td>
<td>370°C at 760 mmHg\textsuperscript{a}; 236°C at 10 mmHg\textsuperscript{a}</td>
</tr>
<tr>
<td>Pour Point (Melting Point)</td>
<td>-50°C\textsuperscript{a}</td>
</tr>
<tr>
<td>Vapour Pressure</td>
<td>3.4 \times 10^{-7} mmHg at 23°C\textsuperscript{b}</td>
</tr>
<tr>
<td>Water Solubility at 25°C</td>
<td>340 μg/l or 45 μg/l\textsuperscript{c}</td>
</tr>
<tr>
<td>Octanol-water Partition Coefficient (Log Kow)</td>
<td>4.88\textsuperscript{b}</td>
</tr>
<tr>
<td>Density (kg/litre)</td>
<td>0.985</td>
</tr>
</tbody>
</table>

All data originated from HWC (1980) unless stated below:

\textsuperscript{a} = HSE (1986);
\textsuperscript{b} = RCEC/TOC (1985);
\textsuperscript{c} = ECETOC (1985) - DEHP may form colloidal dispersions which lead to higher values for solubility.

The chemical structure of DEHP is shown below.

\[
\begin{align*}
\text{C} & \text{- O - CH}_2 \text{CH(CH}_3\text{)} \text{CH}_2 \text{CH}_2 \text{CH}_2 \text{CH}_3 \\
\text{C} & \text{- O - CH}_2 \text{CH(CH}_3\text{)CH}_2 \text{CH}_2 \text{CH}_2 \text{CH}_3 \\
\end{align*}
\]
2. PRODUCTION AND USES

2.1 Production methods

The same general method is used to manufacture all phthalic acid esters, so the comments and figures in this section do not refer specifically to DEHP. Information for this section (production methods) was taken from Cibula (1984).

The esters are produced by the reaction of phthalic anhydride (rather than the acid) with the appropriate alcohol (in the case of DEHP, the alcohol used is 2-ethylhexanol) in the presence of a catalyst; either an acid catalyst (e.g. sulphuric acid, p-toluene sulphonic acid) or tetra-alkyl titanate is used. The process has a high conversion efficiency due to the use of excess alcohol and the continuous removal of the water produced in the reaction; unconverted alcohol is recycled back to the reactor. No process water is used, so although the process is carried out in batch, losses are low.

After the reaction is complete, the excess alcohol is driven off under vacuum, and the product is treated with a neutralising slurry of alkali and charcoal, which removes the excess acid in the form of salts adsorbed by the charcoal. It is estimated that only 0.1% of the acid used is lost as unreacted material. The product is dried and filtered in a rotary vacuum filter; the ester is then passed to a steam-stripping column where any residual alcohol is removed. The main loss of product is in the charcoal, which may contain up to 5% of the material. The charcoal is removed from the drum filter, and about 75% of it can be recycled within the process. The remainder is sent to specialist recoverers, who use the ester content (about 1.3% of ester product) in lower grade plasticisers, and dump the waste charcoal to landfill. Some ester is also lost when the slurry of alkali and charcoal is removed (0.6% ester loss) and again, some ester is lost (about 0.3%) in the steam-stripping process. These losses find their way to the effluent stream, along with all the wastewater from the process, i.e. the water of reaction, the water removed in drying and the steam-stripping condensate. Volatile losses are claimed to be low.

2.2 World production volumes

ECETOC (1985) estimated that globally, about $2.7 \times 10^6$ tonnes/year of total phthalates are produced, of which the non-plasticiser (dimethyl and diethyl) phthalates represent a very small fraction. Of the plasticiser phthalates, DEHP accounts for well over 50% of the tonnage. The contributions of the remaining phthalates to the total production figure range from about 1% to 10% each.

The production of DEHP has been increasing since the compound was first used commercially in 1949. During the period 1950-1954, the US production figure for DEHP was 106,000 tonnes; by the period 1965-1969, the figure had risen to 650,000 tonnes (Peaskill, 1975). A later report (ATSDR, 1987) gave a considerably reduced figure of about 130,000 tonnes for 1986 US DEHP production and estimated this to be a third of total US phthalate production for that year.

The total Western European production of all phthalate plasticisers was estimated at nearly $1 \times 10^6$ tonnes in 1979 (IARC, 1982). MAFF (1987) estimated a similar overall phthalate consumption figure for the European Community of ~ 800,000 tonnes in 1984.

An unpublished Dutch Government report (quoted in Wams, 1987) gave a global DEHP production figure of 3-4 million tonnes for the mid-1980s. This figure is by far the largest DEHP production estimate found in the available literature and it was assumed that Wams referred to total phthalate production rather than to DEHP production.

2.3 United Kingdom production

A total of nine UK manufacturers/suppliers of DEHP were identified in 1983 (Cibula, 1984). These companies are listed below, however it is unlikely that more than one or two of these companies
are actual UK manufacturers of DEHP:

- BASF Aktiengesellschaft
- Bayer UK Ltd
- BP Chemicals Ltd
- Ciba-Geigy UK Ltd
- Dynamit Nobel UK Ltd
- Esso Chemical Ltd
- Hüls UK Ltd
- ICI plc
- Shell Chemicals UK Ltd

Cibula (1984) quoted a value of 80,000 tonnes for the annual production of 11 phthalate esters in the UK in the early 1980s. And another report, OEC7 (1987), quoted a value of 65,000 tonnes for the 1985 production of dioctyl phthalates in the UK. MAFF (1987) quoted a higher total phthalate consumption figure of ~ 100,000 tonnes for the UK in 1984.

The figure of 80,000 tonnes of total phthalate produced per year was used in Cibula's report (Cibula, 1984) to estimate United Kingdom consumption and release figures for DEHP.

More recent information on the production and consumption of phthalates in the UK was provided in a communication from the British Plastics Federation (BPF, 1990). The BPF estimated a total UK consumption of all phthalate esters of 122,000 tonnes in 1989, of which 66,000 tonnes were thought to be DEHP and di-iso-octyl phthalate (DIOP). An estimated 33,000 tonnes of DEHP and DIOP were produced in the UK in 1989. The BPF assumed for calculation that the entire 66,000 tonnes consumption was of DEHP alone, and this figure is used as a maximum consumption figure in Section 3 to estimate releases of DEHP to the UK environment.

2.4 Uses

The largest usage of phthalate esters in general is as plasticising agents for resins and polymers such as polyvinyl chloride (PVC), the cellulosics and certain types of elastomers. Other lesser uses include: as defoaming agents in paper manufacture, in cosmetic products as a vehicle for perfumes, in lubricating oils and in insect-repellent formulations.

Over 95% of phthalates are used as plasticisers, with those of higher molecular weight (ester groups > C₆) being used in flexible PVC (CEFIC (1989) reported that ~ 90% of all plasticisers used in Western Europe were used for this purpose). Plasticisers are materials incorporated into a plastic in order to increase its workability and distendability. Phthalates are physical plasticisers in that they are not bound to the resin polymer, but are dispersed in the matrix of polymer chains, decreasing the interaction forces between adjacent chains and hence promoting chain mobility and flexibility.

Plasticiser content is typically of the order of 40 parts plasticiser per 100 parts resin (phr), but can vary from 10 to > 100 phr. The lack of a chemical bond between the plasticiser and the resin means that it is possible for the plasticiser to be released from the polymer, especially at elevated temperatures such as those used in the manufacture of some PVC goods and forms.

DEHP is the most common of the phthalate esters and has been widely used since 1949. CEFIC (1989) stated that DEHP constitutes 50% of Western European plasticiser usage, and DEHP is quoted as being the most important plasticiser in PVC manufacture. DEHP is also used in cellulose esters, synthetic elastomers and industrial paints, although this latter use is diminishing. MAFF (1987) stated that, unlike many other plasticisers, DEHP was not used in the production of PVC films for food contact use, although use in bottle closure sealant compounds was reported.

DEHP has been used in the past as a dielectric fluid in condensers and as an orchard acaricide.
3. ENVIRONMENTAL RELEASES

It seems likely that the vast bulk of phthalate esters released to the environment arises from human activity and not from natural sources. Some dialky1 esters may be found in coals, crude oil and shales, while others may have plant origins but most originate either directly or indirectly from industrial processes.

The following sections refer to both phthalate release in general and to DEHP in particular. Specific United Kingdom information was received in a communication from the British Plastics Federation (BPF, 1990) based on UK industry estimates and information gathered by the European Chemical Industry Federation (CEFIC) and is included below.

3.1 Point source releases

3.1.1 Release from phthalate production and distribution

In Section 2.1, estimates were made of the percentage amount of phthalates released into the effluent stream at each stage of the manufacturing process. These estimates of production releases were made by a UK phthalate manufacturer, who quoted an overall loss during production of 2.3%; this estimate included losses during filtration and purification and the DEHP adsorbed onto the charcoal during neutralisation of the crude product (Cibula, 1984).

In a recent communication from the British Plastics Federation (BPF, 1990), a lower estimated emission from production was quoted. The BPF stated that the production process quoted by Cibula (1984) (and in Section 2.1 of this report) apparently did not take into account modern production practices, whereby effluents are returned to a central recovery system before disposal. The low water solubility of the ester results in high recovery levels and on this basis a maximum production release of 0.01% of phthalate production was estimated.

ECETOC (1985) also stated that the controlled nature of modern production processes and the low water solubility of phthalates combined to make it unlikely that any significant loss of phthalate to the environment would occur during production, either in aqueous effluents or to the atmosphere. However, ECETOC (1985) concluded that the older production methods, which involve sulphuric acid catalysis, could result in losses of the order of 1% of production to the aqueous environment.

Other estimates of release during the production and distribution of phthalates have been made by other authors for other countries. Berndtsson (1982) (in ECETOC, 1985) suggested a release figure of 0.05% of production for losses sustained during drum and tank cleaning occurring in the distribution process. BPF (1990) estimated a maximum loss during distribution of 5 kg DEHP per 25 tonne lot, i.e. a 0.02% loss rate. This figure represents the quantity of DEHP which cannot easily be drained from a tanker. The estimate ignores the fact that the use of dedicated tankers reduces the amount of cleaning necessary, and that modern steam cleaning processes would not allow the discharge of contaminated washings straight to effluent. The occurrence of spillages and accidents during transport of DEHP cannot easily be included in this estimate.

Leah (1977) quoted values of 0.1-0.3% release of phthalates from production, which had been agreed by the relevant sectors of the Canadian chemical industry: Leah recommended that these estimated figures should be followed up by monitoring. US EPA (1980) proposed a figure of 0.2% phthalate loss by volatilisation during the production process.

These emission estimates can be summarised as follows:

Release from production of DEHP/phthalates: 0.01% of phthalate production [BPF, 1990];
0.1-0.3% of phthalate production [Leah, 1977];
0.2% of phthalate production [US EPA, 1980];
1% of phthalate production [ECETOC, 1985];
2.3% of phthalate production [Cibula, 1984].
Release from distribution of DEHP/phthalates: 0.02% of phthalate production [BPF, 1990]; 0.05% of phthalate production [Berndtsson, 1982].

If these emission factors are applied to the maximum UK DEHP production figure quoted by the BPF of 33,000 tonnes in 1989 and the maximum UK DEHP consumption figure of 66,000 tonnes (assumed to be the amount distributed in the UK), the following DEHP release figures are obtained:

Release from production:
- 3.3 tonnes DEHP/year [BPF, 1990];
- 33-99 tonnes DEHP/year [Leah, 1977];
- 66 tonnes DEHP/year [US EPA, 1980];
- 330 tonnes DEHP/year [ECETOC, 1985];
- 759 tonnes DEHP/year [Gibula, 1984].

Release from distribution:
- 13 tonnes DEHP/year [BPF, 1990];
- 33 tonnes DEHP/year [Berndtsson, 1982].

3.1.2 Release from plastics processing

The British Plastics Federation supplied a breakdown of the end use of processed plastics containing DEHP. The information was originally gathered by the European Chemical Industry Federation and refers to European end uses in general, however, there is no reason to presume that UK end uses differ dramatically from those for Europe as a whole. The following table, Table 3.1, presents this data and estimates the tonnage of DEHP used in these products in the UK. It was assumed that the maximum UK annual consumption of 66,000 tonnes DEHP was entirely used in plastics processing (BPF, 1990).

Table 3.1: END USES OF PLASTICS CONTAINING DEHP (BPF, 1990)

<table>
<thead>
<tr>
<th>End use</th>
<th>Percentage end use</th>
<th>Quantity of DEHP used in the UK per category (in kilotonnes)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Film and sheet</td>
<td>16%</td>
<td>10.6</td>
</tr>
<tr>
<td>Coated products</td>
<td>13.5%</td>
<td>8.9</td>
</tr>
<tr>
<td>Flooring</td>
<td>17%</td>
<td>11.2</td>
</tr>
<tr>
<td>Hose and profile</td>
<td>10%</td>
<td>6.6</td>
</tr>
<tr>
<td>Wire and cable</td>
<td>26%</td>
<td>17.2</td>
</tr>
<tr>
<td>Other</td>
<td>17.5%</td>
<td>11.5</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>66,000 tonnes</td>
</tr>
</tbody>
</table>

BPF (1990) also gave a breakdown of the processing mode used in the production of the above groups of articles. This breakdown is given in Table 3.2 overleaf.
### Table 3.2: Extent to Which the Different Plastics Processing Modes Are Used to Produce Articles Containing DEHP (BPF, 1990)

<table>
<thead>
<tr>
<th>End use</th>
<th>Calendering</th>
<th></th>
<th>Plastisol</th>
<th></th>
<th>Extrusion</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>%</td>
<td>Kilotonnes</td>
<td>%</td>
<td>Kilotonnes</td>
<td>%</td>
<td>Kilotonnes</td>
</tr>
<tr>
<td>Film and sheet</td>
<td>70</td>
<td>7.4</td>
<td>30</td>
<td>3.2</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>Coated products</td>
<td>10</td>
<td>0.9</td>
<td>90</td>
<td>8.0</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>Flooring</td>
<td>30</td>
<td>3.4</td>
<td>70</td>
<td>7.8</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>Hose and profile</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>100</td>
<td>6.6</td>
<td></td>
</tr>
<tr>
<td>Wire and cable</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>100</td>
<td>17.2</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>—</td>
<td>50</td>
<td>5.7</td>
<td>50</td>
<td>5.8</td>
<td></td>
</tr>
</tbody>
</table>

Note: — = This process was not used in the production of these commodities.

BPF (1990) supplied DEHP emission factors for the processing of plastics by the following methods, calendering (a separate emission factor was included for the production of calendered floors), spread coating and plastisol formation and extrusion/injection moulding. Table 3.3 below uses these emission factors and the information in Table 3.2 to calculate DEHP emissions from the plastics processing industry in the UK. The emission factors used take into account the nature of the different processes and the use of fume elimination equipment.

### Table 3.3: DEHP Losses from the UK Plastics Processing Industry (BPF, 1990)

<table>
<thead>
<tr>
<th>Process used</th>
<th>Loss of DEHP from process (%)</th>
<th>DEHP used in process (in kilotonnes)*</th>
<th>Loss of DEHP from process (in tonnes)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calendering</td>
<td>0.2%</td>
<td>8.3&lt;sup&gt;a&lt;/sup&gt;</td>
<td>16.6</td>
</tr>
<tr>
<td>Calendered floors</td>
<td>0.03%</td>
<td>3.4&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1.0</td>
</tr>
<tr>
<td>Spread coating</td>
<td>0.04 or 1.0%&lt;sup&gt;c&lt;/sup&gt;</td>
<td>19.0&lt;sup&gt;d&lt;/sup&gt;</td>
<td>7.6 or 190</td>
</tr>
<tr>
<td>Other plastisols</td>
<td>0.4%</td>
<td>5.7&lt;sup&gt;e&lt;/sup&gt;</td>
<td>22.8</td>
</tr>
<tr>
<td>Extrusion/injection moulding</td>
<td>0.01%</td>
<td>29.6&lt;sup&gt;f&lt;/sup&gt;</td>
<td>3.0</td>
</tr>
</tbody>
</table>

Notes: * - Figures from Table 3.2;  
<sup>a</sup> - Total DEHP used in film and sheet and coated products (calendered);  
<sup>b</sup> - Total DEHP used in flooring (calendered);  
<sup>c</sup> - Loss depends on whether or not fume elimination technology is installed;  
<sup>d</sup> - Total DEHP used in film and sheet, coated products and flooring (plastisol);  
<sup>e</sup> - Total DEHP used for other purposes than ‘d’ (plastisol);  
<sup>f</sup> - Total DEHP used in all extruded products.

From these calculations, a DEHP release figure is estimated of between 51 and 233 tonnes/year.
from the UK plastics processing industry. The worst case figure assumes no fume elimination technology is used at plants employing spread coating processing methods.

A number of other authors have given estimates of the release of phthalate esters from plastics processing and other uses. The most detailed emissions information comes from Leah (1977) and was estimated by the Canadian plastics industry. Table 3.4 presents the emissions as percentages of phthalate use within individual categories and as percentages of overall Canadian phthalate usage.

Table 3.4: RELEASE OF PHTHALIC ACID ESTERS FROM PLASTICS PROCESSING, CANADA, 1973 (after Leah, 1977)

<table>
<thead>
<tr>
<th>Activity</th>
<th>% release (of category use)</th>
<th>% release (of total usage)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vinyl flooring manufacture</td>
<td>1.0 - 5.0</td>
<td>0.16 - 0.83</td>
</tr>
<tr>
<td>Extruded PVC film and sheet</td>
<td>0.1 - 0.3</td>
<td>0.03 - 0.09</td>
</tr>
<tr>
<td>Wire and cable insulation*</td>
<td>0.1 - 0.2</td>
<td>0.01 - 0.03</td>
</tr>
<tr>
<td>Plastisols production</td>
<td>5 - 10</td>
<td>1.6 - 3.2</td>
</tr>
<tr>
<td>Other vinyl extrusions*</td>
<td>0.1</td>
<td>0.01</td>
</tr>
<tr>
<td>Adhesives*</td>
<td>2.0</td>
<td>0.01</td>
</tr>
<tr>
<td>Cellulose film coatings*</td>
<td>0.1</td>
<td>0.01</td>
</tr>
<tr>
<td>Other*</td>
<td>1.0</td>
<td>0.01</td>
</tr>
</tbody>
</table>

Note: * - None of these activities were thought by Leah (1977) to use DEHP as a plasticiser.

These figures give an overall phthalate release to the environment of ~2-4% of the total amount of phthalate supplied for use.

In the UK, the Rubber and Plastics Research Organisation considered that average emissions from plastics processing were likely to be 0.5-1% by weight of the plasticisers used (Cibula, 1984). And, a lower average phthalate release from plastics processing was estimated by Berndtsson (1982) at 0.8% of phthalate usage.

If the emission factors given by Leah (1977), Cibula (1984) and Berndtsson (1982) are applied to the maximum total DEHP consumption figure of 66,000 tonnes, DEHP releases from plastics processing can be calculated for the UK in 1989 as follows:

- 330-660 tonnes DEHP/year [Cibula, 1984];
- 528 tonnes DEHP/year [Berndtsson, 1982];
- 1,320-2,540 tonnes DEHP/year [Leah, 1977].

3.2 Disperse source releases

3.2.1 Release during use of plasticised products

Phthalate esters can be released to the environment at the consumer level from finished goods in use. Leah (1977) identified volatilisation of phthalates from articles during their useful lifetime as...
one source of release in this category.

Losses from articles during their useful lifetime can also occur via leaching (eg from water hoses). Peakall (1975) estimated that articles in direct contact with water lose a maximum of 1% of their phthalate content per year, whilst articles in contact with air (eg flooring, weather stripping, etc) may lose 0.1% per year; this estimate was based on laboratory studies on the loss of phthalates from PVC flooring. Articles with a lower surface contact (eg wire and cable) were arbitrarily assigned a release figure of 0.01% of phthalate content per year; these percentage losses were estimated to occur over the lifetime of the product. Leah (1977) considered that the contribution of such sources to the overall environmental burden of phthalic acid esters was at that time unknown and indeed difficult to estimate. However, Leah commented that bearing in mind the large quantity of articles in use containing phthalates, the contribution to the environmental burden from this source could be considerable. If the BPF (1990) consumption figure of 66,000 tonnes DEHP/year is used along with Peakall’s estimated maximum release of 1% of phthalate content from articles in use, an overall annual release of 660 tonnes DEHP is obtained.

ECETOC (1985) quoted a Swedish study (Berndtsson, 1982) which estimated that 0.35% of annual phthalate consumption would be released to the atmosphere from articles in use, with a further 0.15% entering aquatic systems. If the BPF consumption figure of 66,000 tonnes DEHP/year is used, then the maximum release of DEHP from articles in use would be 330 tonnes/year according to Berndtsson (1982).

Pierce et al (1980) estimated that 1% of the phthalate content of articles placed on the market would be released during their usage; it was stressed that this release would occur over a period of several years.

The British Plastics Federation (BPF, 1990) estimated a low loss of DEHP from products with interior uses. Loss from products not exposed to weather would be mainly by volatilisation and was predicted to be low at 0.025% of the DEHP content because of low vapour pressure. Outdoor use is much less easy to define and a loss of 0.5% DEHP was estimated as a worst case example. Applying these emission factors to the total consumption of 66,000 tonnes DEHP/year gives an interior end use emission of 16.5 tonnes and an exterior emission of 330 tonnes. These DEHP emissions are assumed to occur over the lifetime of the products in use.

3.2.2 Release after disposal of plasticised products

Phthalate esters can also be released to the environment from finished goods after their disposal. Leah (1977) identified two routes for such releases, via the disposal of plasticised articles to landfill and via the incineration of such products.

The amount of phthalic acid esters leached from a landfill situ, if any, will depend upon a number of factors, many of which are specific to the site. ECETOC (1985) considered that phthalates would be slowly leached from plasticised articles, and could in principle reach the aquatic environment via the tip leachate, depending on whether degradation or adsorption to soil occurred. However, the low solubility of these compounds and their high affinity for organic soil particles combine to make it unlikely that major amounts of phthalates would enter the environment by this route. Articles dumped in an uncontrolled manner, i.e. as litter, would be more likely to lead to volatilisation of phthalates to the atmosphere than to contamination of the aquatic environment.

Ghassemi et al (1984) reported the results of analysis on the leachate from hazardous waste disposal sites in the USA. DEHP was found in leachates from two different types of landfill site - Case 1: sites taking mixed industrial wastes; and Case 2: sites taking both municipal and industrial wastes. In both cases leachate samples were taken from above the landfill liners. DEHP was found at a level of 0.20 mg/l in one of 8 leachate samples from case 1 landfill sites. DEHP was also found twice at 8 case 2 sites at a maximum level of 0.11 mg/l.

Wams (1987) estimated that approximately 100,000 tonnes of DEHP were present in Dutch landfills at the time of writing the report. On the basis of an estimated migration rate of 1% per
year, it was calculated that about 1,000 tonnes of DEHP migrate from Dutch landfills into percolating water (leachate) per year. This figure would constantly be changing as the amount of plasticised products disposed of in landfill would increase with time, and the amount of DEHP available to the leachate would also increase. This release figure constitutes a potentially large source of groundwater contamination.

Leah (1977) identified three possible routes into the environment arising from incineration: release with the stack gases, release in the scrubbing water from the stack clean-up (if present), and release in the solid waste residue after incineration, though this latter route was thought unlikely because of the volatile nature of phthalates under incinerator conditions. ECETOC (1985) commented that around 30% of household waste is disposed of by incineration (probably on a European rather than a UK basis), and that in a high temperature incinerator virtually complete combustion of the phthalates is expected. In lower temperature, less well-controlled incinerators, losses to the atmosphere of up to 25% of the phthalates in the incinerated material could occur. Leah (1977) estimated that in Canada in 1973, out of the total material discarded, including residential, commercial and non-specific industrial wastes, approximately 3.5% consisted of plastics. Slightly less than 5% of this amount was PVC, and half of this fell into the flexible vinyls category; this material was assumed to contain 40% plasticisers by weight. Thus, the quantity of phthalates disposed of by landfill or incineration in Canada in 1973 amounted to around 10% of the total phthalic acid ester production figure.

3.3 Natural sources

Phthalates have been reported in a wide variety of substances (oil, soil, plants and animals) over a wide geographic area. Many have anthropogenic origins but some could be naturally produced. The problem is further increased by the fact that sampling techniques often lead to contamination of samples, i.e. contamination from plastic bags or bottles.

Mathur (1974a) critically reviewed this problem and concluded that the possibility of the phthalic acid esters found in biological and geochemical samples being of biosynthetic origin could not be ruled out. Both Mathur (1974a) and Peakall (1975) gave examples of surveys in which phthalates were detected in samples and no anthropogenic source could be found. More recent studies by Manandhar et al (1979) and Paré et al (1981) have shown residues of phthalates in biological samples where the source seems to be natural; di-(2-n-propyl)pentyl phthalate and n-butyl-4-ol n-propyl phthalate were found in the two sets of samples respectively. Peterson and Freeman (1982) suggested that some of the phthalates found in older samples, from the 1920s and 1930s, of sediment cores from Chesapeake Bay, USA, may have been of natural origin.

An ECETOC task force concluded that, although knowledge of naturally produced phthalates is limited or uncertain, it seems unlikely that this contribution to overall phthalate release is of great significance (ECETOC, 1985). No specific mention of the natural occurrence of DEHP was made by either Mathur (1974a) or Peakall (1975).
3.4 Summary

Table 3.5 below summarises the emissions estimates made in the previous sections.

Table 3.5: ESTIMATED ANNUAL EMISSIONS OF DEHP TO THE UNITED KINGDOM ENVIRONMENT

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Production</td>
<td>3.3 tonnes</td>
<td>330 tonnes</td>
<td>3.3 - 759 tonnes</td>
</tr>
<tr>
<td>Distribution</td>
<td>13 tonnes</td>
<td>33 tonnes</td>
<td>13 - 33 tonnes</td>
</tr>
<tr>
<td>Plastics processing</td>
<td>51 - 233 tonnes</td>
<td>528 tonnes</td>
<td>51 - 2,640 tonnes</td>
</tr>
<tr>
<td>Products:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>During use</td>
<td>347 tonnes</td>
<td>330 tonnes</td>
<td>330 - 660 tonnes</td>
</tr>
<tr>
<td>After disposal</td>
<td>U</td>
<td>U</td>
<td>U</td>
</tr>
<tr>
<td>Total emission</td>
<td>414 - 596 tonnes</td>
<td>1,221 tonnes</td>
<td>397 - 4,092 tonnes</td>
</tr>
</tbody>
</table>

Notes: * - Estimates from ECETOC (1985) include those reported in Berndtsson (1982); U - Unknown emission value.

The estimates of overall annual DEHP release to the United Kingdom environment ranged from 397-4,092 tonnes/year. The lower BPF (1990) estimates were based on information received from UK phthalate manufacturers and therefore may be thought to be more accurate and relevant to the UK than the other estimates based on European, Canadian and American emission factors. The higher figure of ~ 1,220 tonnes will however be used as a UK environmental release figure for the modelling exercise in Section 5.5 of this report as it lies near the middle of the range of release values estimated above.

4. ENVIRONMENTAL FATE

4.1 Environmental distribution

Section 3 shows that phthalates can enter the environment by volatilisation to the atmosphere, by direct entry to surface waters, and also by indirect entry to groundwater. DEHP is a ubiquitous environmental pollutant and transport mechanisms exist which distribute DEHP to all phases in the environment in areas distant from the point of release.

4.1.1 Volatilisation

DEHP has a low vapour pressure, and as such will only volatilise slowly from any media in which it is found.

Klöpfer et al (1982) estimated the half-life for the volatilisation of DEHP from water by extrapolating laboratory data on the process under defined conditions. A value of 146 days was obtained, although on purely theoretical grounds a value of only 25 days was calculated. Wolfe et al (1980a), using the Exposure Analysis Modelling System (EXAMS), calculated that at equilibrium the loss of DEHP via volatilisation from a river, a pond, an eutrophic lake and an
oligotrophic lake would be 0%, 2.8%, 2.2% and 2.3% respectively, over a period of 1 hour, 30 days, 200 days and 200 days respectively. The DEHP carried by the river water would be expected to be carried out of the section of river considered by the model within the hour equilibrium period studied, therefore there is no volatilisation of DEHP from the river.

Evaporation of DEHP from plant material is also slow. Løkke and Bro-Rasmussen (1981) treated the leaves of Sinapis alba with a mixture of di-iso-butyl phthalate (DiBP), di-n-butyl phthalate (DBP) and DEHP at a rate of 2.5 μg/cm². Only very small amounts of DEHP evaporated from the leaf during the 15 day experiment, compared with DiBP (71% loss) and DBP (43% loss) (the exact percentage of DEHP evaporating from the leaves was not stated).

4.1.2 Precipitation and dry deposition

ECETOC (1985) suggested that the DEHP which is released to the atmosphere (either directly or via volatilisation from other media) would be distributed throughout the environment by either dry deposition or in precipitation.

ATSDR (1987) commented that because of its high boiling point and low vapour pressure, atmospheric DEHP would have a strong tendency to adhere to atmospheric particulate matter, especially organic matter and soot, and be removed from the atmosphere by rain-out.

4.1.3 Adsorption onto soil and sediment

The solubility of DEHP in water is low (around 0.3-0.4 mg/l at 25°C). However, the amount present in surface water may be higher than this 'real' solubility as a result of adsorption onto organic particles (Taylor et al, 1981) and interactions with dissolved high molecular weight organic matter, such as humic and fulvic acids (Matsuda and Schnitzer, 1971). Infra-red studies indicate that no chemical bond is formed between the phthalate and fulvic or humic acid, but that adsorption to the surface of the acid molecule takes place.

DEHP has a high octanol-water partition coefficient, and so the equilibrium between water and an organic-rich soil or sediment will be very much in favour of the soil or sediment. Adsorption of DEHP by soil or sediment will also be enhanced by van der Waals type bonds with natural soil minerals, promoted by the presence of benzene rings and carbonyl groups, and also by the low solubility of DEHP (ECETOC, 1985).

Al-Omran and Preston (1987) observed that DEHP was adsorbed onto suspended particulate matter fairly rapidly, being most actively adsorbed by small particles; the adsorption of DEHP and five other phthalate esters was enhanced in salt water. This effect was also noted by Sullivan et al (1902) who observed that increasing the salinity of the solution increased the amount of phthalate bound to clay minerals, calcite, a sediment sample and glass tubes; adsorption to and desorption from these substrates in seawater was observed to be rapid. Adsorption appeared to be reversible with mineral and calcite samples, but some retention of the phthalate was seen with the sediment sample, showing that this might act as a final sink for DEHP.

From results with other organic substances, Wams (1987) estimated that 90% of available DEHP would be readily adsorbed by organic soil particles in an ecosystem.

4.1.4 Bioaccumulation

DEHP is highly lipophilic, with an octanol-water partition coefficient of around 4 to 5, and is moderately persistent. The accumulation of DEHP is also dependent on the capability of an organism to metabolise it.

It should be noted that many of the exposure concentrations of DEHP quoted in the following bioaccumulation studies are in excess of the compound’s water solubility. In these cases it is
expected that undissolved DEHP will be adsorbed to various surfaces within the test apparatus, for example to the walls of the test chamber, or in cases where sediment is incorporated into the test ecosystem, to suspended and settled particulate matter. It is also possible that excess undissolved DEHP will collect at water surfaces during the test. All of these processes were observed by Södergren (1982) in the course of his bioaccumulation study. In many of the studies, nominal levels of DEHP are quoted for the exposure period; it is of course preferable for studies to have continuously monitored the exposure levels throughout the duration of the test, and to use a measured DEHP level for the calculation of the bioaccumulation factor.

4.1.4.1 Model ecosystems

Metcalf et al. (1973) studied the distribution of DEHP in a variety of aquatic organisms in a model ecosystem after application of 5 mg of the 14C-labelled compound to Sorghum plants at the terrestrial end of the system. The organisms studied were the alga Oedogonium, the snail Physa sp., the mosquito larvae Culex pipiens quinquefasciatus, and the fish Gambusia sp. The mosquito larvae showed the highest bioconcentration factor (BCF) and the fish the lowest. At the end of the 33 day experiment, the water contained 0.34 µg/l DEHP, the mosquito larvae 36.61 mg/kg (a BCF of 107,680), the alga 18.32 mg/kg (a BCF of 53,880), the snails 7.30 mg/kg (a BCF of 21,470), and the fish 0.044 mg/kg (a BCF of 130). The BCFs quoted here were calculated for this report and not by Metcalf et al. in their original document.

Södergren (1982) exposed fish, aquatic invertebrates and plants to 14C-labelled DEHP at a theoretical concentration of 1.43 mg/l for 27 days under static conditions. After 5 days, one-fiftieth of the added amount of DEHP was still present in the water. At the end of the experiment, 62% of the 14C-DEHP and its metabolites were recovered from the various surfaces, i.e. the walls of the test vessel, the sediment and the surface layer of water. All organisms accumulated DEHP and its metabolites. The amphipod, Gammarus pulex, larvae of trichopterans and the snail, Planorbis corneus, accumulated the 14C-compounds to the highest degree with BCFs ranging from 17,000 to 24,000. The submerged plants Mentha aquatica and Chara corallina also showed uptake and storage of large amounts of DEHP and its metabolites, BCFs of 18,000 were found, whilst the fish, stickleback Pungitius pungitius and the minnow Phoxinus phoxinus, did not accumulate the 14C-compounds to such a great extent (BCFs were 300 or less). Larger accumulations of DEHP and its metabolites occurred in organisms inhabiting and/or feeding at the surface or on the sediment at the bottom than in those living in the free water volume. As these BCFs were calculated using the levels of both 14C-DEHP and its metabolites (the monoethylhexyl ester and phthalic acid) in the organisms after 27 days exposure, it is to be expected that the true BCFs for DEHP will be lower than those quoted above.

4.1.4.2 Aquatic organisms

Geyer et al. (1984) exposed the green alga Chlorella fusca to 14C-labelled DEHP at a concentration of 50 µg/l for 24 hours. A BCF of 5,400 (wet weight) was determined for this organism.

Brown and Thompson (1982a) exposed Daphnia magna to nominal water concentrations of 3.2, 10, 32 and 100 µg/l of 14C-labelled DEHP for 21 days. Bioconcentration factors (BCFs) were 166, 140, 261 and 268 based on the four exposure concentrations respectively.

Brown and Thompson (1982b) exposed mussels, Mytilus edulis, to labelled DEHP at mean measured concentrations of 4.1 and 42.1 µg/l. At both concentrations equilibrium was reached within 14 days, with an associated average BCF of 2,500. Exposure ceased on day 28 and the mussels were monitored for a further 14 days, a half-life for the loss of DEHP over this period was calculated to be 3.5 days.

Laughlin et al. (1978) exposed grass shrimp, during larval development, to nominal initial DEHP concentrations of up to 1 mg/l for 28 days. DEHP was not detectable in shrimp tissue at or above the 2 mg/kg level.
Streufert et al (1980) exposed midge larvae, *Chironomus plumosus*, to water concentrations of 0.2 µg/l radioactively labelled DEHP. The larvae accumulated 292 times the water concentration within 2 days. DEHP levels in the midge larvae reached a plateau level after 7 days at a BCF of 408. When some of the larvae were transferred to clean water after 4 days, by which time they had accumulated 56 µg/kg DEHP, a half-life for loss was measured at 3.4 days.

After 9 weeks exposure to sediment containing approximately 600 mg/kg DEHP, dragonfly larvae had taken up 14.7 mg/kg DEHP, significantly more than controls which were exposed to a less contaminated reference sediment containing 0.4 mg/kg DEHP; the control larvae contained DEHP at a level of 2.9 mg/kg after the exposure period (Woin and Larsson, 1987).

The possibility of DEHP uptake from food as well as from water was investigated by Macuk et al (1979). *Daphnia magna* and bluegill sunfish, *Lepomis macrochirus*, were exposed separately to 14C-DEHP under continuous flow conditions to establish the level of bioconcentration from water. *Daphnia magna* were exposed to a mean measured aqueous concentration of 5.4 ± 1.3 µg/l DEHP for 24 hours, after which time a 14C-DEHP residue of 2.8 ± 0.7 mg/kg was found in the organisms. Bluegill sunfish were exposed to a mean measured aqueous concentration of 5.7 ± 0.9 µg/l DEHP for 35 days (equilibrium was attained within 3 days), after which time a 14C-DEHP residue of 0.64 ± 0.11 mg/kg was found in the fish. BCFs of 518 and 112 were calculated for the *Daphnia* and bluegill respectively. Fish subjected to both dietary and aqueous exposure to DEHP, via *Daphnia* contaminated with 2.8 mg/kg DEHP and water containing 0.56 µg/l DEHP, were found to contain 0.73 ± 0.11 mg/kg 14C-DEHP residues after 35 days exposure, a residue not significantly different to the residues found after aqueous exposure alone. Fish exposed to DEHP only via contaminated *Daphnia magna* (containing 2.8 mg/kg DEHP) were found to contain 0.21 mg/kg DEHP residues after 35 days exposure; this equilibrium level of around 10% of the DEHP level in the *Daphnia* was reached after 14 days. These studies indicate that the effects of exposure to DEHP via dietary and aqueous routes are not additive, that is, the observed increment in DEHP residues due to diet should have been equal to the actual DEHP residues measured in fish exposed to dietary DEHP alone, i.e. 0.21 mg/kg, but the actual observed increment was only 0.09 mg/kg. The authors concluded that bioconcentration through the food chain did not appear to be as important as that direct from aqueous contact.

Tarr et al (1990) exposed three groups of rainbow trout (*Oncorhynchus mykiss*) with different average body weights to 14C-DEHP in a static system. The measured BCFs were found to decrease from 51.5 to 1.6 as the average body weights of the trout increased from 2.9 g to 441 g.

Mehrlie and Mayer (1976) exposed rainbow trout eggs, *Oncorhynchus mykiss*, from 12 days prior to hatching to 24 days post-hatch, to 14C-labelled DEHP at concentrations of 5, 14 and 54 µg/l. BCFs were 78, 113 and 42 for the three dose levels respectively.

Freitag et al (1985) exposed golden orfe (*Leuciscus idus melanotus*) to 14C-labelled DEHP at a concentration of 50 µg/l for 3 days. A BCF of 40 (wet weight basis) was determined for this exposure.

Mayer (1976) exposed fathead minnow, *Pimephales promelas*, to DEHP concentrations ranging from 1.9 to 62 µg/l for 56 days, under flow-through conditions. Bioconcentration factors, measured after 56 days, ranged downwards from 569 to 91 as the exposure concentrations increased. Equilibrium was attained from 28 days at the lowest dose up to 56 days at the highest dose. After exposure the fish were placed in clean water for 28 days. Half-lives for loss of DEHP ranged from 6.2 days (at 2.5 µg/l) to 18.3 days (at 62 µg/l).

The results of some other bioconcentration studies carried out for a variety of aquatic organisms are given in Table 4.1 overleaf.
<table>
<thead>
<tr>
<th>Organism</th>
<th>Stat/flow</th>
<th>Exposure period</th>
<th>Concen. (µg/l)</th>
<th>Bioconcentration factor</th>
<th>Ref.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>FRESHWATER ORGANISMS:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Canadian pondweed</td>
<td>stat</td>
<td>24 hours</td>
<td>10*</td>
<td>274.8</td>
<td>a⁺²</td>
</tr>
<tr>
<td><em>(Elodea canadensis)</em></td>
<td>stat</td>
<td>12 hours</td>
<td>10,000*</td>
<td>133.8</td>
<td>a⁺²</td>
</tr>
<tr>
<td>snail</td>
<td>stat</td>
<td>48 hours</td>
<td>10*</td>
<td>857.5</td>
<td>a⁺²</td>
</tr>
<tr>
<td><em>(Physa sp.)</em></td>
<td>stat</td>
<td>6 hours</td>
<td>10,000*</td>
<td>402</td>
<td>a⁺²</td>
</tr>
<tr>
<td>scud</td>
<td>flow$</td>
<td>7 days</td>
<td>0.1*</td>
<td>13,600</td>
<td>b⁺²</td>
</tr>
<tr>
<td><em>(Gammarus pseudolimnaeus)</em></td>
<td>flow$</td>
<td>7 days</td>
<td>0.1*</td>
<td>3,900</td>
<td>c⁺²</td>
</tr>
<tr>
<td>water flea</td>
<td>flow$</td>
<td>7 days</td>
<td>0.3*</td>
<td>5,200</td>
<td>b⁺²</td>
</tr>
<tr>
<td><em>(Daphnia magna)</em></td>
<td>flow</td>
<td>7 days</td>
<td>0.3*</td>
<td>420</td>
<td>c⁺²</td>
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<td></td>
<td>stat</td>
<td>1 hour</td>
<td>10*</td>
<td>421</td>
<td>a⁺¹</td>
</tr>
<tr>
<td></td>
<td>stat</td>
<td>48 hours</td>
<td>10,000*</td>
<td>155.1</td>
<td>a⁺¹</td>
</tr>
<tr>
<td>sowbug</td>
<td>flow$</td>
<td>21 days</td>
<td>62.3*</td>
<td>250</td>
<td>b⁺²</td>
</tr>
<tr>
<td><em>(Asellus brevicaudus)</em></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>mosquito</td>
<td>stat</td>
<td>12 hours</td>
<td>10*</td>
<td>1,320.2</td>
<td>a⁺¹</td>
</tr>
<tr>
<td><em>(Culex pipiens)</em></td>
<td>stat</td>
<td>24 hours</td>
<td>10,000*</td>
<td>1,187.3</td>
<td>a⁺¹</td>
</tr>
<tr>
<td>quinquefasciatus</td>
<td>stat</td>
<td>24 hours</td>
<td>10*</td>
<td>20.3</td>
<td>a⁺¹</td>
</tr>
<tr>
<td></td>
<td>stat</td>
<td>48 hours</td>
<td>10,000*</td>
<td>434.6</td>
<td>a⁺¹</td>
</tr>
<tr>
<td>midge larvae (3rd instar)</td>
<td>flow$</td>
<td>7 days</td>
<td>0.3*</td>
<td>3,100</td>
<td>b⁺²</td>
</tr>
<tr>
<td><em>(Chironomus plumosus)</em></td>
<td>flow</td>
<td>7 days</td>
<td>0.3*</td>
<td>350</td>
<td>c⁺²</td>
</tr>
<tr>
<td>mayfly</td>
<td>flow$</td>
<td>7 days</td>
<td>0.1*</td>
<td>2,300</td>
<td>b⁺²</td>
</tr>
<tr>
<td><em>(Hexagenia bilineata)</em></td>
<td>flow</td>
<td>7 days</td>
<td>0.1*</td>
<td>575</td>
<td>c⁺²</td>
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<tr>
<td>mosquito fish</td>
<td>stat</td>
<td>48 hours</td>
<td>10*</td>
<td>265.3</td>
<td>a⁺¹</td>
</tr>
<tr>
<td><em>(Gambusia affinis)</em></td>
<td>stat</td>
<td>12 hours</td>
<td>10,000*</td>
<td>129.4</td>
<td>a⁺¹</td>
</tr>
<tr>
<td>fathead minnow</td>
<td>flow</td>
<td>14 days</td>
<td>1.9*</td>
<td>458</td>
<td>c⁺²</td>
</tr>
<tr>
<td><em>(Pimephales promelas)</em></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>MARINE ORGANISMS:</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eastern oyster (muscle)</td>
<td>stat</td>
<td>24 hours</td>
<td>100</td>
<td>11.2</td>
<td>n d</td>
</tr>
<tr>
<td><em>(Crassostrea virginica)</em></td>
<td>stat</td>
<td>24 hours</td>
<td>500</td>
<td>6.9</td>
<td>n d</td>
</tr>
<tr>
<td>brown shrimp</td>
<td>stat</td>
<td>24 hours</td>
<td>100</td>
<td>10.2</td>
<td>n d</td>
</tr>
<tr>
<td><em>(Penaeus aztecus)</em></td>
<td>stat</td>
<td>24 hours</td>
<td>500</td>
<td>16.6</td>
<td>n d</td>
</tr>
<tr>
<td>sheepshead minnow</td>
<td>stat</td>
<td>24 hours</td>
<td>100</td>
<td>10.7</td>
<td>n d</td>
</tr>
<tr>
<td><em>(Cyprinodon variegatus)</em></td>
<td>stat</td>
<td>24 hours</td>
<td>500</td>
<td>13.5</td>
<td>n d</td>
</tr>
</tbody>
</table>

Notes and references for this table on facing page.
NOTES AND REFERENCES FOR TABLE 4.1:

Notes: static - static conditions (water unchanged for the duration of the test);
flow - flow-through conditions (DEHP concentration in water continuously maintained);
flow$ - intermittent flow-through conditions;
bioconcentration factor = concentration of DEHP in organism / concentration of DEHP in water;
* - bioconcentration factor calculated using a radioactive isotope;
n - calculations of BCFs are based on nominal initial concentrations of DEHP in water;
1 - these BCFs were calculated for this report and not quoted in the original reference;
2 - BCF calculations based on measurement of total radioactivity in organisms and water, i.e. $^{14}$C-DEHP and any $^{14}$C-labelled metabolites of DEHP.

References: a = Metcalf et al (1973);
b = Sanders et al (1973);
c = Mayer and Sanders (1973);

4.1.4.3 Amphibians

Larsson and Thurén (1987) exposed Moorfrog eggs to sediment containing DEHP at concentrations ranging from 10 to 800 mg/kg. The eggs hatched after about 3 weeks and the tadpoles were analysed after 60 days. The DEHP was released from the sediment to the overlying water, the losses to the water increasing linearly with increasing levels in the sediment, from 0.89 to 187.4 $\mu$g/l. DEHP accumulated in tadpoles at concentrations ranging from 0.28 to 246.8 mg/kg fresh weight. The accumulation increased with increasing levels of DEHP in both sediment and the water.

4.1.4.4 Plants

Løkke and Bro-Rasmussen (1981) applied DEHP at a rate of 2.5 $\mu$g/cm$^2$ (in a mixture also containing di-iso-butyl and di-n-butyl phthalates) to the leaves of Sinapis alba. Residues of DEHP in the leaves immediately after application were 2.7 $\mu$g/cm$^2$. After 15 days, DEHP levels were measured at 0.8 $\mu$g/cm$^2$, but when the growth of the plant was taken into account, no significant loss of DEHP over this period was found. Løkke and Rasmussen (1983) also found little loss of DEHP, over a 15 day period, when they applied the chemical to Achillea at a rate of 3.5 $\mu$g/cm$^2$ or to Sinapis at 3.1 $\mu$g/cm$^2$. Residues ranged from 120 to 155 $\mu$g per plant, on both occasions DEHP was applied in a mixture with di-n-butyl phthalate. Approximately 80% of the DEHP applied remained on the surface of the leaf.

O'Connor (1988) studied the uptake and accumulation of three organic chemicals by plants supplied with the substances in municipal sewage sludge. The chemicals in the sludge were DEHP, Aroclor 1248 (a polychlorinated biphenyl) and pentachlorophenol. There was little bioaccumulation of DEHP by the plants studied (fescue, lettuce, carrots and chile), the maximum BCF found was 0.53. A further study by Aranda et al (1989) using the same plants and the same method of exposure to DEHP also found low BCFs (0.01-0.03 on a fresh weight basis).

Kloskowski et al (1981) treated a humus-sand (organic carbon content 2.89%) with $^{14}$C-labelled DEHP at a concentration of about 2 mg/kg. Summer barley was sown into this soil and exposed to the chemical for a week. At the end of the week, the total radioactivity recovered from the plant, soil and gaseous phase was > 95%, the correspondingly low plant BCF was calculated at 1.1.

These studies suggest strongly that little or no intact DEHP would persist in plants grown in DEHP-contaminated soil and that any DEHP taken up by the plants would be metabolised rapidly.
4.1.4.5 Birds

Ishida et al (1982) fed hens on a diet containing 5 or 10 g/kg DEHP for up to 230 days. DEHP was detected in all tissue monitored except the brain, with residues ranging from not detected to 42.9 mg/kg. Adipose tissue, 192.7 to 899.6 mg/kg DEHP, and feathers, 513.1 to 1,165.2 mg/kg, accumulated the highest concentrations. A similar pattern of uptake was observed in hens fed 2 g/kg for 25 days, although the amount of DEHP accumulated was much lower. At this dose level no accumulation had occurred after 5 days in tissues other than the liver and feathers.

Belisle et al (1975) fed mallard ducks, *Anas platyrhynchos*, on a diet containing 10 mg/kg DEHP for a period of 5 months. No DEHP was detected in fat tissue; levels of 0.1 and 0.15 mg/kg (wet weight) were found in breast muscle and lung tissue respectively.

O’Shea and Stafford (1980) exposed starlings, *Sturnus vulgaris*, to a dietary concentration of 25 or 250 mg/kg DEHP for 30 days. One of the eight birds fed 25 mg/kg contained detectable residues (1.6 mg/kg) after 30 days exposure. Five of the eight birds fed 250 mg/kg contained an average of 1.8 mg/kg DEHP after 30 days exposure, and still contained detectable residues (an average of 1.3 mg/kg) 14 days after dosing had finished.

4.2 Abiotic degradation

4.2.1 Photolysis

There is a general lack of data available concerning the photolysis of DEHP. One report (Fraunhofer Gesellschaft, 1984) was found which indicated that DEHP undergoes rapid photodegradation in the atmosphere, with a half-life of less than one day (no further experimental information was available). However, Freitag et al (1983) reported slower degradation of DEHP in a study simulating abiotic processes in the atmosphere. DEHP was sorbed to silica gel and exposed to 17 hours irradiation at λ > 290 nm. DEHP was reported to undergo 1.6% degradation in this time.

Wolfe et al (1980a), using the Exposure Analysis Modelling System (EXAMS), calculated that at equilibrium, loss via photolysis from a river, a pond, an eutrophic lake and an oligotrophic lake would be 0%, 1.8%, 1.4% and 13.7% respectively, over a period of 1 hour, 30 days, 200 days and 200 days respectively.

4.2.2 Photo-oxidation

There is a lack of quantitative data available concerning the photo-oxidation of DEHP in the atmosphere, however, it is reported to occur only very slowly.

US EPA (1982) quoted rate constants for the oxidation of DEHP in aquatic systems at 25°C by singlet oxygen (\( ^1\)O\(_2\) ) and peroxy radicals (RO\(_2\) ) as much less than 360 M\(^{-1}\) hour\(^{-1}\) and 7.2 M\(^{-1}\) hour\(^{-1}\) respectively.

Its resistance to photo-oxidation in general is one of the characteristics of DEHP that makes the substance suitable as a plasticiser in durable plastic products (Wams, 1987).

4.2.3 Hydrolysis

A half-life of over 100 years in water was found for DEHP by Wolfe et al (1980b) at pH 8 and 30°C. This is considered too low a rate to affect aqueous concentrations significantly. Wolfe et al (1980a), using EXAMS (see Section 4.2.1 above), calculated that at equilibrium, 0.2% DEHP would be lost via hydrolysis from an eutrophic lake (over a time period of 200 days), and 0% would be lost from a river, a pond or an oligotrophic lake (over a time period of 1 hour, 30 days and 200 days respectively).
4.3 Biodegradation

4.3.1 Aerobic degradation

A number of studies on the biodegradation of DEHP have indicated that it is degradable under aerobic conditions. ECETOC (1985) commented that at high dilutions in rivers and in seawater, or in the river die-away test, short-chain phthalates are degraded fairly quickly, whereas those with longer chains, such as DEHP, are only 40–90% degraded after 10–35 days (primary degradation). In a river die-away test reported by Saeger and Tucker (1973a, 1973b) a concentration of 1 mg/l was observed to undergo 50% primary degradation after 2-5 weeks. At higher concentrations, such as those in sewage treatment works or simulations of sewage treatment works, degradation again seemed to occur under aerobic conditions. In an acclimatised 'shake-flask' (CO₂ evolution) test, DEHP gave evidence of more than 90% primary degradation and more than 55% ultimate degradation. The half-life for ultimate degradation was less than 28 days (Sugatt et al., 1984). Graham (1973) used a semi-continuous activated sludge system (SCAS), in which sludge from domestic sewage was mixed with DEHP and with a synthetic sewage mixture with the desired concentration of suspended solids. At a feed rate of 5 mg DEHP in 48 hours, 91% of the chemical was degraded within that time (type of degradation unstated). However, Freitag et al (1985) found only 4% degradation of DEHP after 5 days incubation in activated sludge taken from a municipal sewage treatment plant and treated with 0.05 mg/l DEHP.

Saeger and Tucker (1976) found that 60% of the DEHP that they added to Mississippi River water underwent primary biodegradation within 5 weeks. Rapid primary degradation was also found when DEHP was added to activated sludge at the rate of 5 mg per 24 hours. Between 70% and 78% degradation was found, depending on the source of the sludge. To monitor whether complete biodegradation was being achieved, Saeger and Tucker measured carbon dioxide (CO₂) evolution. Within the 14 day incubation period, DEHP had essentially achieved complete degradation to CO₂ and water under the conditions of this test.

Taylor et al (1981) demonstrated the presence of significant populations of taxonomically distinct bacteria, which grew on a range of phthalic acid esters including DEHP, in the water and sediments of the Mississippi River region. Taylor et al (1981) also mentioned that in surface waters, DEHP was strongly adsorbed onto organic particles (see Section 4.1.3) and Baughman et al (1980) commented that adsorption onto organic matter would tend to reduce the amount of DEHP available for degradation by water-borne micro-organisms.

Johnson and Lulves (1975) studied cultures consisting of freshwater hydrosol with an overlying layer of pond water incubated under aerobic conditions. The cultures were treated so that the average ¹⁴C-DEHP content in both hydrosol and water was 1 ppm. DEHP underwent 53% primary degradation after 14 days incubation at 22°C, a much slower rate of degradation than that found with di-n-butyl phthalate (DBP), 98% of DBP undergoing ultimate degradation within 5 days. Engelhardt et al (1977) found that the fungus Penicillium lilacinum degraded approximately half of the available DEHP within 30 days, and Kurane et al (1977a, 1977b) reported that the bacterium Pseudomonas acidovorans completely degraded DEHP at a media concentration of 5,000 mg/kg within 72 hours.

Tabak et al (1981) studied the aerobic degradation of DEHP using settled domestic wastewater as the microbial inoculum. The incubation flasks also contained salts and yeast extract. The original culture was incubated for 7 days with either 5 or 10 mg/l of DEHP and was subjected to three weekly subcultures which were introduced to fresh media after extraction from the original culture. DEHP was significantly biodegraded, but required a gradual 3 week adaptation period before 95% and 93% DEHP loss (from the 5 and 10 mg/l cultures respectively) was achieved at the end of the third subculture incubation period. After the first subculture, only 43% and 47% loss of DEHP respectively was observed.

Thomas et al (1986) found ¹⁴C-DEHP to be mineralised by mixed cultures of bacteria obtained from a sewage treatment plant (primary effluent). Rates of mineralisation were determined by trapping ¹⁴CO₂ evolved from flasks containing the inoculated medium. The mineralisation of DEHP was first detected after 28 hours, before this activity was detected the previously continuous film of
DEHP on the surface of the liquid was found to have dispersed into fine droplets. After 96 hours, 66% of the DEHP had been mineralised.

Johnson et al (1984) looked at the influence of environmental factors on the primary biodegradation of phthalates, including DEHP, in water-containing sediments from a lake in Missouri, USA. They concluded that phthalates degraded optimally at concentrations close to their solubility limit and at temperatures above 22°C in nutrient-rich systems (eg sewage treatment plants, eutrophic lakes, and streams) in the summer. It was thought possible that phthalates in general could accumulate on sediments in the winter. Fish et al (1977) reported that in lake sediments, 20% of a 14C-labelled amount of DEHP appeared as 14CO₂ after 28 days under aerobic conditions at 20°C. Ring cleavage was thought to have occurred. No significant degradation of DEHP occurred at temperatures lower than 20°C.

Mathur (1974b) reported that the aerobic degradation of DEHP in soil was dependent upon temperature. A loam soil was incubated with DEHP at 4°C, 10°C, 22-25°C and 32°C. Soil respiration rates were measured after 14 weeks. Increased rates of respiration, showing that microbial degradation was taking place, were found at all temperatures. However, results indicated that only marginal degradation was taking place at 4°C and 10°C.

Engelhardt and Wallnöfer (1978) commented that aerobic biodegradation of phthalates in the upper layers of soil proceeded as for that occurring in surface water, but that further down degradation was virtually non-existent.

Shanker et al (1985) incubated garden soil containing DEHP at a concentration of 500 mg/kg. Within 20 days, 75% of the DEHP had been primarily degraded and after 30 days, more than 90% had disappeared. Again, the rate of degradation was much slower than that found for either di-n-methyl or di-n-butyl phthalate. The authors found no degradation when sterilised soil was used.

The degradation of 14C-labelled DEHP was monitored in three soils from New Mexico amended or not with anaerobically digested sewage sludge (Fairbanks et al, 1985). Evolution of 14CO₂ was the only observed mechanism of DEHP loss from the soil (no volatilisation was observed) and it was assumed that this indicated complete degradation of the chemical. 50% DEHP disappearance occurred in 8 to 72 days, depending on the soil type, the concentration of the chemical and the sludge pre-incubation time (prior to being applied to the soil). After 146 days, 76-93% of the DEHP had been released as 14CO₂. In general, DEHP persistence increased with increase in the initial loading of the soil. Degradation was also more complete in the two sandy loam soils tested than in the clay soil.

In general, aerobic degradation of DEHP is achieved rapidly and fairly completely in the environment. Adsorption of the chemical onto sediments was considered to reduce the amount of DEHP available for aerobic degradation under some experimental conditions.

4.3.2 Anaerobic degradation

In contrast to its behaviour under aerobic conditions, DEHP appears to be relatively stable under anaerobic conditions.

Johnson and Lulves (1975) found DEHP to be completely resistant to microbial attack under anaerobic conditions. After 30 days, no significant loss of 14C-DEHP activity in freshwater hydrosoils under a nitrogen atmosphere was found. In a later study, Johnson et al (1984) found slow primary degradation of DEHP under anaerobic conditions in water-containing sediments from a lake in Missouri, USA, with approximately 10% of the DEHP degrading over a period of 28 days. However, Schwartz et al (1979) found no signs of degradation after 14 days in sediment samples from Dutch rivers.

Laboratory studies by Shelton et al (1984) indicated that DEHP would not be mineralised at a significant rate in municipal sludge anaerobic digesters, although shorter chain phthalates would be. O'Connor et al (1989) also studied the degradation of DEHP in a medium containing municipal
digester sludge under anaerobic conditions. They found that biotransformation occurred at all three levels of DEHP dosage, i.e. at 20, 100 or 200 mg/l. Biotransformation removed 100%, 69% and 54% of DEHP respectively at the three concentrations quoted. DEHP was the only carbon source present; however, complete biodegradation to carbon dioxide and methane was minimal.

Battersby and Wilson (1989) studied the degradation potential of DEHP (amongst other chemicals) under methanogenic conditions in an anaerobic digesting sludge from the south-east of England. Degradation was assessed in terms of net gas production (methane and carbon dioxide), expressed as a percentage of theoretical gas production. DEHP was observed over more than 11 weeks and no evidence of degradation was seen, i.e. little effect on gas production was observed when compared to the sterile controls.

Shanker et al (1985) found that the degradation of DEHP was much reduced in anaerobic soil, achieved by flooding the soil with sterile water. After 30 days incubation, 33% of the DEHP had been primarily degraded compared with more than 90% in aerobic soil.

Indirect evidence for the stability of DEHP and other phthalates under anaerobic conditions comes from a study of anaerobic sediment cores from Chesapeake Bay, Baltimore, USA. Peterson and Freeman (1982) found that the DEHP content of cores from successively deeper levels correlated with the production volumes from the corresponding time periods. This suggests that degradation of DEHP under anaerobic conditions in sediment is indeed slow.

4.3.3 Metabolism

The aerobic metabolic pathway of the biodegradation of DEHP can be summarised as follows: the di-ester is hydrolysed into the mono-ester by esterases with low substrate specificity (Kurane et al., 1980; and Taylor et al., 1981). The mono-ester is then converted into phthalic acid (Engelhardt et al., 1975). Degradation to pyruvate and succinate and then to carbon dioxide and water is similar to the metabolic pathway of benzoic acid. Kurane et al (1984) suggested that this is probably why the biodegradation of phthalic acid esters is so widespread. It appears that mixed populations of micro-organisms are the most successful at completely degrading DEHP (Engelhardt et al, 1975; Kurane et al, 1978; and Kurane et al, 1979).

Two separate studies exposing fish to DEHP continuously for 24 hours in a static test have been reported; Stalling et al (1973) used channel catfish (Ictalurus punctatus) whilst Melancon and Lech (1976) used rainbow trout (Oncorhynchus mykiss). In both cases, more than 85% of the DEHP absorbed was degraded to intermediate moieties. The main product with the catfish was mono-ethylhexyl phthalate, whilst that with the trout was a conjugate of the mono-ester; these differences were considered by Pierce et al (1980) to have arisen from differing experimental protocols, and not from the differences in species or in DEHP concentrations used.

4.4 Summary

The environmental fate of DEHP is influenced strongly by its physical and chemical properties. It is a high boiling liquid with a low water solubility and a vapour pressure of $3.4 \times 10^7$ mmHg (at 25°C). The compound is lipophilic, and has a log octanol-water partition coefficient of 4.88.

There is a general lack of data for DEHP on the occurrence of abiotic degradation processes in the environment, i.e. photolysis, photo-oxidation, etc, and it is not known what effect these processes may have on the overall fate of DEHP.

The biodegradation of DEHP will occur fairly rapidly under aerobic conditions, but DEHP will degrade very much more slowly, if at all, in anaerobic systems. DEHP released to the atmosphere can be removed from this phase by dry deposition or rain-out. It is not known what role photolysis and photo-oxidation reactions play in the removal of DEHP from this compartment.

In the aquatic environment, DEHP accumulates in sediments, on suspended solid materials and in
the lipid tissues of aquatic biota. In soil, DEHP is strongly held by soil solids and organic material such as fulvic acid, from which it is not readily leached.

5. ENVIRONMENTAL LEVELS

DEHP is widespread and found in most environmental samples, including air, precipitation, water, sediment, soil and biota; residues have also been detected in food and in human tissue.

In many cases, there is uncertainty as to the origin of the DEHP measured in environmental samples. Firstly, the ubiquitous presence of plasticisers in laboratory equipment (tubing etc) makes the prevention of contamination extremely difficult. Secondly, it has been suggested that phthalate esters may occur naturally in the environment.

5.1 Occurrence in air

Atmospheric DEHP levels have been measured in only a few areas of the world. Background levels have been measured in some remote areas, but no levels were found for the United Kingdom atmosphere.

The levels of DEHP found in remote areas were ≤ 5 ng/m³; whereas those in urban areas were higher at up to 30 ng/m³. A high level of 77 ng/m³ was measured in Sweden (Thurén and Larsson, 1990). This figure may have been due to the influence of industrial effluents which tend to elevate DEHP measurements; indeed, concentrations of up to 300 ng/m³ have been reported in polluted areas (see Table 5.1).

Other studies reported the levels of DEHP found in airborne particulate matter alone. Cautreels et al (1977) sampled the air at two different sites for DEHP in particulate matter. Two 42 day air samples collected at a remote site near La Paz, Bolivia, were found to contain 17 and 20 ng/m³ DEHP in particulate matter; and four 14-day samples collected in the residential city of Antwerp, Belgium, were found to contain higher levels of DEHP in the airborne particulate matter of between 26 and 132 ng/m³ air sampled.

A later study by Cautreels and Van Cauwenberghe (1978) found a sample of air collected from an unspecified polluted atmosphere to contain DEHP in particulate matter at a mean level of 54.1 ng/m³.
### Table 5.1: Concentrations of DEHP in the Atmosphere

<table>
<thead>
<tr>
<th>Location</th>
<th>Concentration (ng/m³)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gulf of Mexico</td>
<td>Av: 0.4; Range: &lt; 0.4 - 2.3</td>
<td>Giam et al (1978)</td>
</tr>
<tr>
<td>North Atlantic</td>
<td>Av: 2.9; Range: 1.4 - 4.1</td>
<td>Giam et al (1978)</td>
</tr>
</tbody>
</table>
| Gulf of Mexico; 1977 | Av: 1.16 (57%)
Range: 0.33 - 1.92 (100 - 51%) | Giam et al (1980) |
| Enewetak Atoll, North Pacific Ocean; 1979 | Av: 1.4; Range: 0.32 - 2.68² | Atlas and Giam (1981) |
| Sweden; 1984-85³ | Av: 2.0; Range: 0.28 - 77.0² | Thurén and Larsson (1990) |
| Great Lakes ecosystem | 0.5 - 5.0⁴ | Eisenreich et al (1981) |
| New York, 1975; Queens, Staten Island and Brooklyn (urban areas) | Average concentrations: 10.20 - 16.79⁴
| Hamilton, Ontario, air near a water works | 300 | Thomas (1973) |
| Polluted air; 1976 (unspecified location) | 127² | Cautreels and Van Cauwenberghe (1978) |

Notes: ND - Not detected, detection limit not stated;
1 - This figure includes DEHP measured in airborne particulate matter collected simultaneously with the air sample. The figure in brackets represents the percentage of DEHP found in the vapour phase alone, i.e. the true air phase concentrations are: Av: 0.66 ng/m³; Range: 0.53-0.98 ng/m³;
2 - Vapour phase concentration only;
3 - 14 sites sampled for 4 consecutive 3 month periods, 53 samples collected in total;
4 - Includes DEHP measured in airborne particulate matter collected simultaneously with the air sample.

#### 5.2 Occurrence in Water and Sediment

##### 5.2.1 Surface water (fresh)

The only data found concerning levels of DEHP in freshwater in the UK were reported by Fatoki and Vernon (1990). They measured DEHP levels in two rivers in an industrial area of Greater Manchester in 1984. Levels measured in the River Irwell ranged from not detected up to 0.4 µg/l, a higher maximum level of 1.6 µg/l DEHP was found in the River Etherow (detection limit not stated).

Table 5.2 overleaf gives details of freshwater monitoring surveys carried out in other countries.

Cole et al (1984) carried out a survey of the concentrations of pollutants in the urban stormwater runoff from 19 American cities. The runoff would eventually be channelled back into the fresh water rivers and lakes of the regions studied. DEHP was not found frequently enough for its mean level to be calculated, i.e. there were not enough measured values to calculate a statistically valid mean. Out of 86 samples taken, DEHP had a 13% frequency of detection. DEHP was detected at
levels of 7-39 µg/l in four cities - Long Island, New York; Knoxville, Tennessee; Austin, Texas; and Denver, Colorado.

Table 5.2 : CONCENTRATIONS OF DEHP IN WATER

<table>
<thead>
<tr>
<th>Location</th>
<th>Concentration (µg/l)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>THE NETHERLANDS</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>River Rhine; 1977</td>
<td>0.3 - 1.7</td>
<td>Schouten et al (1979)</td>
</tr>
<tr>
<td>River IJssel; 1977</td>
<td>0.6 - 2.6</td>
<td>Schouten et al (1979)</td>
</tr>
<tr>
<td>River Meuse; 1977</td>
<td>0.4 - 4.0</td>
<td>Schouten et al (1979)</td>
</tr>
<tr>
<td>River Rhine; 1982</td>
<td>ND - 4.0</td>
<td>Wams (1987)</td>
</tr>
<tr>
<td>River Rhine; 1983</td>
<td>ND - 1.2</td>
<td>Wams (1987)</td>
</tr>
<tr>
<td>River Meuse; 1983</td>
<td>&lt; 0.1 - 3.5</td>
<td>Wams (1987)</td>
</tr>
<tr>
<td>Lake Yssel (inland lake); 1986</td>
<td>&lt; 0.1 - 0.3</td>
<td>Ritsema et al (1989)</td>
</tr>
<tr>
<td><strong>SWEDEN</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>River Ronnebyan:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>upstream of industrial discharge near discharge point</td>
<td>0.32 - 1.78</td>
<td>Thurén (1986)</td>
</tr>
<tr>
<td>near discharge point</td>
<td>0.66 - 3.10</td>
<td>Thurén (1986)</td>
</tr>
<tr>
<td>River Svartan:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>upstream of industrial discharge near discharge point</td>
<td>0.39 - 0.52</td>
<td>Thurén (1986)</td>
</tr>
<tr>
<td>downstream of discharge point</td>
<td>1.98</td>
<td>Thurén (1986)</td>
</tr>
<tr>
<td></td>
<td>0.39</td>
<td>Thurén (1986)</td>
</tr>
<tr>
<td><strong>CANADA</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lake Superior, Ontario, rural/industrial area</td>
<td>300</td>
<td>Mayer et al (1972)</td>
</tr>
<tr>
<td><strong>USA</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Missouri River, Missouri</td>
<td>4.9</td>
<td>Mayer et al (1972)</td>
</tr>
<tr>
<td>Lake Huron, Michigan</td>
<td>5.0</td>
<td>Mayer et al (1972)</td>
</tr>
<tr>
<td>Unidentified river receiving wastes from chemical plants</td>
<td>1 - 50</td>
<td>Jungclaus et al (1978)</td>
</tr>
<tr>
<td>Mississippi River, entire length</td>
<td>ND - 0.72</td>
<td>DeLeon et al (1986)</td>
</tr>
<tr>
<td><strong>JAPAN</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Various rivers</td>
<td>ND - 3.1</td>
<td>Kodama et al (1975)</td>
</tr>
<tr>
<td>Various cities; 1974</td>
<td>0.1 - 2.19</td>
<td>Goto (1979)</td>
</tr>
</tbody>
</table>

Note : ND = Not detected, detection limits not reported.

5.2.2 Seawater

One DEHP level was reported for a non-industrialised UK estuary, the Crouch estuary in Essex, by Waldock (1983). The range of levels measured was 58.5-78.3 ng/l. A study of the dissolved and particulate levels of phthalate esters in the Mersey Estuary, considered to be one of the most polluted estuary systems in the United Kingdom, was carried out in 1985 by Preston and Al-Omran (1986). Sixteen samples of seawater were collected from sites from the Walton railway bridge in Warrington down river to a site in the Crosby Channel out in Liverpool Bay. The samples were analysed for DEHP content in both the aqueous phase and the suspended particulate phase. There seemed to be a general trend of decreasing concentration with increasing salinity. Levels of DEHP in seawater fell within the range 83-335 ng/l. The concentration of DEHP in the suspended particulate fraction ranged from 182-702 µg/kg; a particularly high DEHP level of 1,700 µg/kg was found in a sample collected from the river bank in Runcorn. The levels found in seawater reported
above agree with levels found in other regions reported in Table 5.3 below.

**Table 5.3: CONCENTRATIONS OF DEHP IN SEAWATER**

<table>
<thead>
<tr>
<th>Location</th>
<th>Concentration (ng/l)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>North Atlantic Ocean</td>
<td>0.1 - 6.3</td>
<td>Giam et al (1978)</td>
</tr>
<tr>
<td>Gulf of Mexico, near coast</td>
<td>6 - 316</td>
<td>Giam et al (1978)</td>
</tr>
<tr>
<td>Gulf of Mexico, open Gulf</td>
<td>6 - 97</td>
<td>Giam et al (1978)</td>
</tr>
<tr>
<td>West Germany, various estuaries</td>
<td>ND - 300</td>
<td>Weber and Ernst (1983)</td>
</tr>
<tr>
<td><strong>USA</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mississippi Delta</td>
<td>23 - 225</td>
<td>Giam et al (1978)</td>
</tr>
<tr>
<td>Galveston Bay, Texas, 8 sites; 1978/70</td>
<td>Av: 600 Range: &lt; 2 - 12,000</td>
<td>Murray et al (1981a)</td>
</tr>
</tbody>
</table>

Note: ND = not detected, detection limit not reported.

5.2.3 *Groundwater*

A survey by the Water Research Centre (Kenrick et al, 1985) sampled 32 public and private supply boreholes in four UK aquifers for trace organics during the period March to August 1983. A total of 43 samples were analysed; DEHP was found in only one of the aquifers monitored in 7 out of 11 samples. These samples, from a sandstone aquifer, were found to contain an average DEHP level of 0.07 µg/l (the average of the seven occurrences) and a maximum level of 0.1 µg/l.

Other reported levels are few. Wams (1987) reported that contaminated groundwater in The Netherlands was found to contain between 20 and 45 µg/l DEHP, and Rao et al (1985) found higher DEHP levels of up to 170 µg/l in New York state groundwater.

Hutchins et al (1985) studied groundwater contamination at rapid infiltration sites in the USA. These sites offer an opportunity for the land treatment of municipal wastes, but can lead to groundwater contamination. DEHP levels were measured in wastewater at three infiltration sites and in the groundwater at the same sites as follows:

1. **Fort Devens, Massachusetts, 1978** - Wastewater: 5.6 µg/l; Groundwater: 1.4 µg/l.
2. **Boulder, Colorado, 1978**  - Wastewater: 0.95 µg/l; Groundwater: 0.052 µg/l.
3. **Phoenix, Arizona, 1980**  - Wastewater: 0.058 µg/l; Groundwater: 0.098 µg/l.

As can be seen, DEHP did appear in the groundwater at these sites; Hutchins et al (1985) stated that in samples taken from both wastewater before infiltration and groundwater at the infiltration sites, on average phthalate esters were detected in both sets of samples 86% of the time.
5.2.4 Precipitation

Atlas and Giam (1981) monitored the amount of DEHP in rainfall at Eniwetok Atoll in the North Pacific Ocean between April and August 1979. DEHP levels ranged from 5.3 to 213 ng/l, with an average of 55 ng/l. These levels are fairly high for what might be considered a background site for pollutant measurement. These samples were however analysed whole, i.e. the values represent both total dissolved DEHP and particulate DEHP in the samples (both wet and dry deposition were measured).

Both wet and dry deposition were also measured by Thurén and Larsson (1990) in a study of DEHP levels in the precipitation at 14 sites in Sweden. Precipitation was monitored for 4 consecutive 3 month periods between October 1984 and October 1985. In total, 56 samples were taken, and a median DEHP level of 48 ng/l was determined (range = 8.3-429 ng/l).

Lower levels of DEHP were found in cases where wet deposition only was measured. Eisenreich et al (1981) reported that between 4 and 10 ng/l DEHP were measured in precipitation falling on the Great Lakes ecosystem. Although it is not stated, it seems fair to assume that Goto (1979) measured both wet and dry deposition in his survey of the rainfall in various Japanese cities, since relatively high mean DEHP concentrations of 0.65 to 3.16 µg/l were reported. Ligocki et al (1985) found lower levels of DEHP in rainfall measured in Portland, Oregon, USA. Precipitation was collected over a 1-5 day period during 7 rain events in Spring 1984. The average DEHP level found was 49 ng/l (range of values from 15-100 ng/l).

Løkke and Rasmussen (1983) analysed snow samples from near to a plasticiser production plant. Levels of DEHP ranged from 0.7-4.7 µg/m²/day, with the highest levels being within 150 metres of the plant and the lowest levels at least 600 metres away.

5.2.5 Municipal water, sewage treatment and industrial effluents

Fatoki and Vernon (1990) reported the presence of DEHP in the final effluent from the Prestwich sewage treatment plant in Greater Manchester, UK. The effluent, sampled in 1984, contained 1.9 µg/l DEHP.

DEHP was also found to occur in American sewage treatment works as reported by Bennett (1989). DEHP was detected in 94% of the influent samples taken in the range 2-390 µg/l and also in 95% of the untreated sewage sludge samples in the range 1-42,300 mg/kg (wet weight). DEHP was also found at fairly high levels in two surveys of American sewage sludges as follows (Jacobs et al., 1987):

1. DEHP found at levels greater than the detection limit for the study (not stated) in 84% of the 234 sludge samples tested.
   Range: 0.42-58,300 mg/kg (dry weight); Median: 168 mg/kg (dry weight).

2. DEHP found at levels greater than the detection limit for the study (not stated) in 95% of the 437 sludge samples tested.
   Range: 2-47,000 µg/l.

5.2.6 Sediment

DEHP, being lipophilic, tends to be adsorbed onto sediment which acts as a sink for the chemical. UK levels were reported for the River Usk, where Eglinton et al (1978) detected 30,000 µg DEHP/kg sediment (on a dry weight basis), and for the estuary of the River Crouch, Essex, where Wallock (1983) reported finding DEHP at 11.2-26.2 µg/kg sediment (wet weight). DEHP levels found in sediment samples from countries other than the United Kingdom are given in Table 5.4.
### Table 5.4: CONCENTRATIONS OF DEHP IN SEDIMENT

<table>
<thead>
<tr>
<th>Location</th>
<th>Concentration (µg/kg)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>THE NETHERLANDS</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>River Meuse; 1977</td>
<td>1,000 - 17,000 dw</td>
<td>Schwartz et al (1979)</td>
</tr>
<tr>
<td>River Ijssel; 1977</td>
<td>2,500 - 52,500 dw</td>
<td>Schwartz et al (1979)</td>
</tr>
<tr>
<td>River Rhine; 1977</td>
<td>6,500 - 70,500 dw</td>
<td>Schwartz et al (1979)</td>
</tr>
<tr>
<td>Lake Yssel, inland lake, six samples;</td>
<td>12,000 - 25,000spm</td>
<td>Ritsema et al (1989)</td>
</tr>
<tr>
<td><strong>SWEDEN</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>River Ronnebyan:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>upstream of industrial discharge</td>
<td>1,200 - 8,030 dw</td>
<td>Thurén (1986)</td>
</tr>
<tr>
<td>near discharge point</td>
<td>79,200 - 628,000 dw</td>
<td>Thurén (1986)</td>
</tr>
<tr>
<td>River Svartan:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>upstream of industrial discharge</td>
<td>1,520 - 3,520 dw</td>
<td>Thurén (1986)</td>
</tr>
<tr>
<td>near discharge point</td>
<td>1,480,000 dw</td>
<td>Thurén (1986)</td>
</tr>
<tr>
<td>downstream of discharge point</td>
<td>150 dw</td>
<td>Thurén (1986)</td>
</tr>
<tr>
<td><strong>CANADA</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lake Superior, Ontario,</td>
<td>200</td>
<td>Mayer et al (1972)</td>
</tr>
<tr>
<td>rural/industrial area</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>USA</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chester River, Maryland; 1978</td>
<td>&lt; 45 dw</td>
<td>Peterson and Freeman (1984)</td>
</tr>
<tr>
<td>Tributary of Chester River; 1978¹</td>
<td>20 4,800 dw</td>
<td>Peterson and Freeman (1984)</td>
</tr>
<tr>
<td>Unidentified river receiving</td>
<td>200 - 56,000 ww</td>
<td>Jungclaus et al (1978)</td>
</tr>
<tr>
<td>waste from chemical plants; 1976</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chesapeake Bay, Maryland; 1978</td>
<td>110 dw</td>
<td>Peterson and Freeman (1984)</td>
</tr>
<tr>
<td>Nueces estuary system, Texas,</td>
<td>Av: 2,500 dw</td>
<td>Ray et al (1983b)</td>
</tr>
<tr>
<td>industrial area; 1980</td>
<td></td>
<td>Ray et al (1983a)</td>
</tr>
<tr>
<td>Estuary at Portland, Maine,</td>
<td>Range: 40 - 16,000 dw</td>
<td></td>
</tr>
<tr>
<td>industrial area; 1980</td>
<td>Av: 1,500 dw</td>
<td></td>
</tr>
<tr>
<td>Mississippi Delta</td>
<td>Range: 60 - 7,800 dw</td>
<td></td>
</tr>
<tr>
<td>Gulf of Mexico, near coast</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gulf of Mexico, open Gulf</td>
<td>&lt; 0.1 - 248</td>
<td></td>
</tr>
<tr>
<td>Galveston Bay, Texas (Gulf of</td>
<td>3.4 - 14.2</td>
<td>Giam et al (1978)</td>
</tr>
<tr>
<td>West Galveston Bay, Texas (Gulf of</td>
<td>Av: 22</td>
<td>Giam et al (1976)</td>
</tr>
<tr>
<td></td>
<td>Av: 94</td>
<td>Murray et al (1981b)</td>
</tr>
<tr>
<td></td>
<td>Range: 13 - 170 dw</td>
<td></td>
</tr>
<tr>
<td><strong>JAPAN</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Various rivers; 1974</td>
<td>80 - 1,360 dw</td>
<td>Goto (1979)</td>
</tr>
</tbody>
</table>

**Notes:**
- **dw** - dry weight;
- **ww** - wet weight;
- **spm** - suspended particulate material;
- ¹ - Sample collected 2 km downstream of a phthalate ester manufacturing plant outfall.
5.3 Occurrence in soil

Contaminated soil from The Netherlands was found to contain up to 1.5 mg/kg DEHP (dry weight) (Wams, 1987). Residues of DEHP in soil collected in the vicinity of a DEHP manufacturing plant contained up to 0.5 mg/kg DEHP (dry weight) (Persson et al, 1978). Diercsens and Tarradellas (1987) found no detectable DEHP in farmland soil from Epenses, Switzerland. After the application of sewage sludge (containing 16,610 mg DEHP/kg dry weight) the soil contained 157 mg/kg DEHP. No DEHP was detectable one month after application.

No information was found on levels of DEHP in UK soil.

5.4 Occurrence in biota

5.4.1 Human tissue

It was recognised by Trimble et al (1966) and by Guess and Haberman (1968) that DEHP can be leached from certain medical devices and can be detected in the fluids and substances passing through them or stored in them. Marcel and Noel (1970) reported the presence of phthalate esters in human plasma that had been stored in plastic blood bags.

Jaeger and Rubin (1972) found that DEHP was extracted from PVC plastic blood bags by human blood at the rate of 0.25 mg/100 ml/day at 4°C. DEHP was found in lipid-containing and lipid-free fractions of plasma; red cells contained only minor amounts. Lung tissue from 7 out of 12 patients who were autopsied and had received blood transfusions contained detectable levels of DEHP, ranging from 13.4 to 91.5 mg/kg (dry weight). In the patient with the highest lung tissue levels of DEHP, 69.5 and 25.3 mg/kg were found in the liver and spleen respectively. Mes et al (1974) analysed human adipose tissue, they found the level of DEHP in most samples to be in the 0.3 to 1.0 mg/kg range.

5.4.2 Aquatic organisms

Table 5.5 gives some levels of DEHP found in a range of biota. Further details of some of these and other studies are given below.

Ray et al (1983a) found DEHP levels of up to 490 μg/kg (dry weight) in Neanthes virens and up to 170 μg/kg (dry weight) in clams collected from the area of Portland, Maine, USA. The levels in the invertebrates collected did not seem to reflect the sediment levels or local pollutant sources.

Mayer et al (1972) analysed for DEHP in various aquatic biota collected in both agricultural and industrial areas of North America. Levels of DEHP varied from 200 μg/kg in dragonfly naïads to 3,200 μg/kg in channel catfish.

Glam et al (1978) collected various biota, mainly fish, from the Gulf of Mexico. DEHP residues ranged from < 1 to 135 μg/kg. Musial and Uthe (1980) collected various fish species from the Gulf of St. Lawrence, Canada, and analysed for DEHP in fish lipid extracts, levels of up to 6.5 mg/kg on a wet weight basis (51.3 mg/kg on a fat weight basis) were found in mackerel muscle and up to 7.2 mg/kg (47.1 mg/kg on a fat weight basis) in herring muscle. Lower levels of 0.37 mg/kg (wet weight) were found in eels, and in both plaice and redfish levels were less than 0.001 mg/kg.

Persson et al (1978) collected aquatic organisms from the vicinity of a DEHP factory in Finland. Aquatic invertebrates contained up to 0.1 mg/kg DEHP (dry weight). Fish collected in the area also contained DEHP, for example, 1.1 mg/kg (dry weight) in roach muscle and 2.3 mg/kg (dry weight) in pike liver. Thureén (1986) analysed biota in two Swedish rivers near to industrial effluent discharge points. In the River Ronneby, organisms contained up to 14.4 mg/kg DEHP (freshweight basis in Asellus aquaticus) upstream of the discharge point, and in the River Svartan, Odonata sp. (dragonfly larvae) were found to contain 5.3 mg/kg at the discharge point.
<table>
<thead>
<tr>
<th>Organism</th>
<th>Location</th>
<th>Concentration (µg/kg)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>molluscs; 2 species digestive glands</td>
<td>UK; Crouch estuary, Essex</td>
<td>9.2 - 214</td>
<td>Waldock (1983)</td>
</tr>
<tr>
<td>dragonfly naiads</td>
<td>USA; Iowa (industrial)</td>
<td>200</td>
<td>Mayer et al (1972)</td>
</tr>
<tr>
<td>channel catfish</td>
<td>USA; Miss. and Ark. (industrial)</td>
<td>3,200</td>
<td>Mayer et al (1972)</td>
</tr>
<tr>
<td>channel catfish</td>
<td>USA; Iowa (industrial)</td>
<td>400</td>
<td>Mayer et al (1972)</td>
</tr>
<tr>
<td>various fish species</td>
<td>Japan</td>
<td>70 - 450</td>
<td>Kodama and Takai (1974)</td>
</tr>
<tr>
<td>various fish species</td>
<td>Japan</td>
<td>&lt; 50 - 1,800</td>
<td>Kamata et al (1978)</td>
</tr>
<tr>
<td>various fish species</td>
<td>Japan; various cities; 1974</td>
<td>50 - 720</td>
<td>Goto (1979)</td>
</tr>
<tr>
<td>various species of aquatic organism</td>
<td>Gulf of Mexico</td>
<td>&lt; 1 - 135</td>
<td>Giam et al (1978)</td>
</tr>
<tr>
<td>dab; muscle (M) and liver (L)</td>
<td>UK; Crouch estuary, Essex</td>
<td>M: 13.8</td>
<td>Waldock (1983)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>L: 2.0 - 2.4</td>
<td></td>
</tr>
<tr>
<td>dab, plaice and whiting (liver)</td>
<td>UK; Tees Bay</td>
<td>43 - 85.9</td>
<td>Waldock (1983)</td>
</tr>
<tr>
<td>dab, plaice and whiting (muscle)</td>
<td>UK; Tees Bay</td>
<td>13 - 51.3</td>
<td>Waldock (1983)</td>
</tr>
<tr>
<td>walleye</td>
<td>Canada; Lake Superior, Ontario¹</td>
<td>800</td>
<td>Mayer et al (1972)</td>
</tr>
<tr>
<td>tadpoles</td>
<td>USA; Iowa (industrial)</td>
<td>300</td>
<td>Mayer et al (1972)</td>
</tr>
<tr>
<td>common seal pup (blubber)</td>
<td></td>
<td>10,600</td>
<td>Zitko (1972)</td>
</tr>
</tbody>
</table>

Notes:  
ww - wet weight;  
lw - lipid weight;  
¹ - rural/industrial area.
5.4.3 Terrestrial organisms

Persson et al (1978) analysed soil arthropods from near to a DEHP factory in Finland. Residues of 2.8 mg/kg DEHP were found.

5.4.4 Food

DEHP has been found in many fish and shellfish samples (see Section 5.4.2 and Table 5.5). DEHP has been found in milk (at a level of 80 mg/l in one sample) (Cerbulis and Ard, 1967), bovine pineal gland (Taborsky, 1967), bovine heart muscle (Nazir et al, 1971) and chicken eggs (Ishida et al, 1981). Perkins (1967) isolated a substance similar to DEHP from corn oil. ATSDR (1987) quoted an FDA (US Food and Drug Administration) survey of various foods in 1974 that showed levels in most foods of less than 1 mg/kg DEHP; foods surveyed included margarine, cheese, meat, cereal, eggs, milk, white bread, canned corn, corn meal and baked beans.

Ishida et al (1981) collected and analysed chicken eggs commercially available in Japanese markets. DEHP levels in egg white ranged from 0.05 to 0.4 mg/kg. No DEHP was detected in the egg yolks collected.

Mayer et al (1972) analysed catfish from the national fish hatchery, they were found to contain 0.4 mg/kg DEHP. Commercial fish food contained levels of up to 7 mg/kg. Zitko (1972) detected DEHP in hatchery-reared juvenile Atlantic salmon at 13 to 16 mg/kg lipid; fish food contained 8 to 9 mg DEHP/kg lipid.

Williams (1973) analysed fish available to the Canadian consumer. Only 6 of the 21 samples contained measurable amounts of DEHP. Levels of 0.1 mg/kg were found in unprocessed eels, and levels of up to 0.16 mg/kg DEHP were found in processed canned tuna fish.

Due to its use in closure seals for glass containers, DEHP has been found in a few samples of bottled beverages. MAFF (1987) found combined levels of DEHP and di-isooctyl phthalate (DIOP) in bottled beers, ciders and soft drinks at levels of < 0.01-0.1 mg/kg and in draught beers at lower levels of < 0.01-0.04 mg/kg.

5.5 Estimation of the environmental distribution of DEHP by modelling

This section includes a modelling exercise which attempted to estimate the way in which DEHP distributes itself in the United Kingdom environment once it has been released there as a result of human activities. The computer model used by the Building Research Establishment has been developed from Mackay’s original fugacity models (Mackay, 1979; and Mackay and Paterson, 1981), and uses a UK ‘model world’ in which all the environmental compartments (air, water, soil, sediment, suspended sediment and biota) have the approximate dimensions appropriate to the UK (not including Northern Ireland).

In terms of hazard assessment for potential environmentally dangerous chemicals, modelling exercises of this sort can pinpoint the possible major sinks likely for the chemical in question (if they are not already known or apparent). Once the major sinks have been pinpointed, then it can be seen whether sufficient data on environmental levels in the appropriate compartment already exist for an assessment to be made, or if more surveys for the chemical in one particular compartment are necessary before assessing its potential hazard.

The model used was at Level II in Mackay’s development of his fugacity models. The Level II model incorporates degradation rates and a continuous rate of input of the chemical, derived from the annual release figure for the UK environment. In the case of DEHP, an annual UK release figure of 1,220 tonnes was used (see Section 3 for its estimation).

Mackay’s models are used quite frequently to estimate the environmental distribution of chemicals, since only a small amount of usually readily available information is required for their
operation. The information needed is listed here: molecular weight, water solubility, vapour pressure (at 20°C), octanol-water partition coefficient, the UK annual release figure and as many of the transformation and degradation rate constants for the chemical in the environment as can be derived. The data for DEHP are presented in Table 5.6.

Table 5.6: INFORMATION USED IN THE MODELLING EXERCISE ON DEHP

<table>
<thead>
<tr>
<th>Information</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Molecular weight</td>
<td>391</td>
</tr>
<tr>
<td>Water solubility</td>
<td>$4.5 \times 10^{-2} \text{ mg/l } (1.15 \times 10^{-4} \text{ mol/m}^3)$</td>
</tr>
<tr>
<td>Vapour pressure (at 20°C)</td>
<td>$3.4 \times 10^{-7} \text{ mmHg } (4.54 \times 10^{5} \text{ Pa})$</td>
</tr>
<tr>
<td>Octanol-water partition coefficient</td>
<td>4.88 (Log value); 7.59 x 10^6 (non-log value)</td>
</tr>
<tr>
<td>Amount of DEHP released in the United Kingdom per year</td>
<td>1,220 tonnes (356 moles/hour)</td>
</tr>
<tr>
<td>Rate constants:</td>
<td></td>
</tr>
<tr>
<td>River : Die-away</td>
<td>$8.7 \times 10^{-4} - 7 \times 10^{-3}/\text{hour (t}_{1/2} = 7 - 5 \text{ weeks)}$</td>
</tr>
<tr>
<td>Soil : Biodegradation</td>
<td>$9.3 \times 10^{-4}/\text{hour (t}_{1/2} &lt; 1 \text{ month)}$</td>
</tr>
<tr>
<td>Water : Hydrolysis</td>
<td>$8 \times 10^{-7}/\text{hour (t}_{1/2} = 100 \text{ years)}$</td>
</tr>
<tr>
<td>Air : Photolysis</td>
<td>$2.9 \times 10^{-2}/\text{hour (t}_{1/2} &lt; 1 \text{ day)}$</td>
</tr>
<tr>
<td>Sediment : Biodegradation</td>
<td>$9.3 \times 10^{-4}/\text{hour (t}_{1/2} &lt; 1 \text{ month)}$</td>
</tr>
</tbody>
</table>

The rate data were derived from information given in Sections 4.2 and 4.3. Three different sets of rate data were used in the model. The first set included the lowest value for each rate where a range of values was found, and excluded the photolysis rate in air as it was not very well defined; this gave a worst case situation. The second set used the highest values in each range, again excluding photolysis; finally the third set used high rate values and the air photolysis rate.

Table 5.7 below gives the range of results obtained from the three modelling exercises.

Table 5.7: ESTIMATED DISTRIBUTION OF DEHP IN THE ENVIRONMENT

<table>
<thead>
<tr>
<th>Compartment</th>
<th>Amount/moles</th>
<th>Concentration/mol m(^3)</th>
<th>Concentration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Air</td>
<td>5.150 - 8.880</td>
<td>$2.75 \times 10^{-12} - 4.75 \times 10^{-12}$</td>
<td>1.08 - 1.86 ng/m(^3)</td>
</tr>
<tr>
<td>Water</td>
<td>8.75 - 15.1</td>
<td>$1.70 \times 10^{-8} - 2.93 \times 10^{-8}$</td>
<td>6.65 - 11.5 ng/l</td>
</tr>
<tr>
<td>Soil</td>
<td>$2.22 \times 10^5 - 3.83 \times 10^5$</td>
<td>$3.96 \times 10^{-8} - 6.83 \times 10^{-8}$</td>
<td>10.3 - 17.8 µg/kg</td>
</tr>
<tr>
<td>Sediment</td>
<td>140 - 241</td>
<td>$9.52 \times 10^{-5} - 1.64 \times 10^{-4}$</td>
<td>24.8 - 42.7 µg/kg</td>
</tr>
<tr>
<td>Suspended sediment</td>
<td>0.25 - 0.42</td>
<td>$9.52 \times 10^{-5} - 1.64 \times 10^{-4}$</td>
<td>24.8 - 42.7 µg/kg</td>
</tr>
<tr>
<td>Biota</td>
<td>0.80 - 1.38</td>
<td>$6.19 \times 10^{-5} - 1.07 \times 10^{-4}$</td>
<td>24.2 - 41.7 µg/kg</td>
</tr>
</tbody>
</table>
The general agreement between the model predictions and the measured levels of DEHP in the UK environment was good. The predicted levels for the sediment and biota compartments were in very good agreement with those levels measured in the UK. Levels of DEHP found in the aquatic environment of the UK tended to be about 100-1,000 times greater than those levels predicted by the model. The predicted air levels were of the same order as those background levels measured in remote areas of the world; no comparisons could be made with UK air levels as no data could be found for this compartment. The soil levels predicted by the model were less than those found in contaminated areas of the environment.

6. ENVIRONMENTAL EFFECTS

6.1 Aquatic toxicity

6.1.1 Invertebrates

Data on the acute toxicity of DEHP to aquatic invertebrates are summarised in Table 6.1. Most studies have determined nominal values for acute toxicity tests in excess of 10 mg/l, values which give a low toxicity rating for DEHP. However, the solubility of DEHP is 45 μg/l (340 μg/l if colloidal dispersions are formed). Therefore, concentrations in excess of 340 μg/l will be unstable emulsions, and the actual exposure of the organisms will be impossible to determine.

A single, recent study showed Daphnia pulex to be considerably more sensitive than previous studies, with a nominal 48h LC50 of 0.133 mg/l (133 μg/l) (Passino and Smith, 1987). This contrasts with 11 mg/l for a 48h LC50 on another species of water flea Daphnia magna (LeBlanc, 1980).

Table 6.1: TOXICITY OF DEHP TO AQUATIC INVERTEBRATES

<table>
<thead>
<tr>
<th>Organism</th>
<th>Age</th>
<th>Stat/flow</th>
<th>Temp °C</th>
<th>Hardness (mg/l)</th>
<th>pH</th>
<th>Parameter</th>
<th>Conc. (mg/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>water flea (Daphnia pulex)</td>
<td>&lt; 24h</td>
<td>stat</td>
<td>17</td>
<td></td>
<td></td>
<td>48h - LC50</td>
<td>0.133</td>
</tr>
<tr>
<td>water flea (Daphnia magna)</td>
<td>&lt; 24h</td>
<td>stat</td>
<td>21-23</td>
<td>173</td>
<td>7.4-9.4</td>
<td>48h - LC50</td>
<td>&gt; 68b</td>
</tr>
<tr>
<td></td>
<td>&lt; 24h</td>
<td>stat</td>
<td>21-23</td>
<td>173</td>
<td>7.4-9.4</td>
<td>48h - LC50</td>
<td>&gt; 11b</td>
</tr>
<tr>
<td>crayfish (Oncorhynchus nais)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>96h - LC50</td>
<td>&gt; 10c</td>
</tr>
<tr>
<td>harpacticoid (Nitocra spinipes)</td>
<td>adult</td>
<td>stat</td>
<td>20-22</td>
<td>7s</td>
<td>7.8</td>
<td>96h - LC50</td>
<td>&gt; 300d</td>
</tr>
<tr>
<td>scud (Gammarus pseudolimnaeus)</td>
<td></td>
<td>stat</td>
<td></td>
<td></td>
<td></td>
<td>96h - LC50</td>
<td>&gt; 32e</td>
</tr>
<tr>
<td>midge (Chironomus plumosus)</td>
<td>larvae</td>
<td>stat</td>
<td>21-23</td>
<td>270</td>
<td>7.4</td>
<td>48h - LC50</td>
<td>&gt; 18f</td>
</tr>
</tbody>
</table>

Notes: stat - static conditions - water not changed during the exposure; s - salinity (%/oo), if salinity not given then test performed in freshwater; n - nominal concentrations.
References used in Table 6.1:  
a = Passino and Smith (1987);  
b = LeBlanc (1980);  
c = Mayer and Sanders (1973);  
d = Lindén et al (1979);  
e = Sanders et al (1973);  
f = Streufert et al (1980).

Brown and Thompson (1982a) found no mortality of *Daphnia magna* over a period of 48h exposure to nominal concentrations of up to 320 µg/l DEHP. However, at 180 µg/l or more DEHP, the Daphnids were seen to float in the surface layer. The authors suggested that this is connected with the solubility of DEHP (DEHP tends to precipitate out at above 180 µg/l and may precipitate onto the daphnids causing them to float). Stephenson (1983) found no mortality in *Gammarus pulex* after 96h exposure to DEHP concentrations in excess of its solubility at 400 µg/l and after 24h in toxicant free water.

Laughlin et al (1978) exposed grass shrimp, *Palaemonetes pugio*, to DEHP concentrations of up to 1 mg/l (well above the solubility limit of the ester). They found no significant difference in mortality between the controls and treated shrimp during the 28 day exposure period. DEHP had no significant effect on the development rate of larvae from hatching to moult. No apparent effects were noted when mussels, *Mytilus edulis*, were exposed to a nominal concentration of 50 µg/l DEHP for 28 days (Brown and Thompson, 1982b).

Hobson et al (1984) found no significant mortality in penaeid shrimps, *Penaeus vannamei*, fed a diet containing up to 50 g/kg DEHP for 14 days, representing 4% of body weight per day. Histological examination revealed no changes and no significant dose-related effects were observed on moult ing.

Sanders et al (1973) and Mayer and Sanders (1973) exposed *Daphnia magna* for a complete life cycle (21 days) to 3, 10 or 30 µg/l DEHP in an intermittent-flow system. Reproduction was significantly inhibited at all concentrations (60, 70 and 83% inhibition at the 3 doses respectively). In contrast, Brown and Thompson (1982a) found no effect on the reproduction of *Daphnia magna* at concentrations up to 100 µg/l. These latter authors commented on the differences in the numbers of young born to the controls in the two studies as a possible explanation for the difference, i.e. 11 offspring per parent (Mayer and Sanders, 1973) compared with 170 per parent (Brown and Thompson, 1982a). Knowles et al (1987) exposed *Daphnia magna* to measured DEHP concentrations of between 12 and 811 µg/l for up to 21 days, under flow-through conditions (the reproduction rate was approximately 200 offspring per adult). Survival was not affected up to and including 158 µg/l DEHP. At 811 µg/l (the next highest dose), survival was significantly reduced after both 7 and 21 days. The mean number of young per surviving adult was also reduced at this highest dose. Major biochemical components such as protein, RNA, DNA and glycogen were all significantly reduced after 7 days at 811 µg/l DEHP, but all except glycogen were unaffected after 21 days. The authors state, therefore, that the maximum acceptable toxicant concentration (MATC), based on survival and reproduction, was between 158 and 811 µg/l. A no effect concentration (NOEC) of 72 µg/l was identified by both DNA content and RNA/DNA ratio at day 7 and by surface behaviour of *Daphnia* at 7 (at higher concentrations the *Daphnia* were observed to come to the water's surface, and although they appeared to be trapped at the surface, they appeared to be healthy and continued to feed).

In flow-through chronic toxicity tests with DEHP in both sand and hydrosoil, concentrations as high as 360 µg/l for sand and 240 µg/l for hydrosoil had no significant effect on growth and development of midge larvae *Chironomus plumosus* over a 35 day period. The continuous exposure of first generation midge eggs in sand substrate to mean DEHP concentrations of between 140 and 360 µg/l had no significant effect on hatchability (Streufert et al, 1980).

Woin and Larsson (1987) found that dragonfly larvae (*Aeshna* sp.) caught significantly fewer prey (*Chaoborus* larvae) per effort when exposed to sediment DEHP concentrations of approximately 600 mg/kg for between 3 and 9 weeks.
6.1.2 Fish

The acute toxicity of DEHP to fish is summarised in Table 6.2. Most acute studies with fish were unable to attain 50% mortality at the concentrations used, all 96h LC50s were, therefore, greater than a nominal concentration of 10 mg/l, with a test on bluegill sunfish giving a value of greater than 770 mg/l (undissolved material present) (Buccafusco et al, 1981). The only fully estimated value was a 96h LC50 on rainbow trout at 540 mg/l (Hruday et al, 1976), which was considered to underestimate the actual toxicity of DEHP due to loss of the compound from the test solution (a surface emulsion of undetermined stability was observed to form). However, as in Section 6.1.1, these values are also much higher than the solubility of DEHP. Therefore, the data are difficult to interpret because the actual exposure of the fish is unknown. It seems reasonable to conclude, from the data available, that DEHP is of low acute toxicity to fish.

Table 6.2 : TOXICITY OF DEHP TO FISH

<table>
<thead>
<tr>
<th>Organism - Size/age</th>
<th>Stat/flow</th>
<th>Temp °C</th>
<th>Hardness (mg/l)</th>
<th>pH</th>
<th>Parameter</th>
<th>Conc. (mg/l)</th>
<th>Ref.</th>
</tr>
</thead>
<tbody>
<tr>
<td>bluegill - 0.32-1.2g (Lepomis macrochirus)</td>
<td>stat</td>
<td>21-23</td>
<td>32-48</td>
<td>6.7-7.8</td>
<td>96h-LC50</td>
<td>&gt; 770 n</td>
<td>(b)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>96h-LC50</td>
<td>&gt; 10 n</td>
<td>(a)</td>
</tr>
<tr>
<td>fathead minnow (Pimephales promelas) - 29-34 days</td>
<td>flow</td>
<td>24-26</td>
<td>44-46.4</td>
<td>7.0-8.2</td>
<td>96h-LC50</td>
<td>&gt; 10 n</td>
<td>(a)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>96h-LC50</td>
<td>&gt; 0.33 m</td>
<td>(d)</td>
</tr>
<tr>
<td>channel catfish (Ictalurus punctatus)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>96h-LC50</td>
<td>&gt; 10 n</td>
<td>(a)</td>
</tr>
<tr>
<td>rainbow trout - finger (Oncorhynchus mykiss)</td>
<td>stat</td>
<td>14-16</td>
<td>70-80</td>
<td>7.4-7.8</td>
<td>96h-LC50</td>
<td>540 c</td>
<td>(c)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>96h-LC50</td>
<td>&gt; 10 n</td>
<td>(a)</td>
</tr>
</tbody>
</table>

Notes: stat - static conditions - water not changed during the exposure; finger - fingerling; ! - same as 24h-LC50 value; m - measured concentration; n - nominal concentration; c - calculated value.


Silvo (1974) found no mortality in one year old rainbow trout, Oncorhynchus mykiss, exposed to between 1 and 1,000 mg/l DEHP for 48h. Zitko (1972) found that DEHP caused no deaths among juvenile Atlantic salmon (Salmo salar) at 100 mg/l over 96h.

Mehrle and Mayer (1976) reported no effects on growth or survival of adult fathead minnow, Pimephales promelas, exposed to between 1 and 62 μg/l DEHP for 56 days. They also exposed rainbow trout eggs to DEHP levels of 5, 14 and 54 μg/l for 12 days prior to hatching; no significant effect on hatchability was found. The resulting fry were continuously exposed to DEHP for a further 90 days. The two highest concentrations caused a significant, but not dose-related, increase in mortality of sac fry within 5 days of hatching. After yolk absorption, at 24 days, DEHP caused no significant mortality or effects on growth and development for the remainder of the exposure.
DeFoe et al (1990) carried out chronic toxicity tests on the Red Band strain of rainbow trout \textit{(Oncorhynchus mykiss)} and the Japanese medaka \textit{(Oryzias latipes)}. No significant adverse effects were noted on the hatchability, survival or growth of rainbow trout exposed to a mean DEHP concentration of 0.502 mg/l (the highest concentration tested) in a 90-day embryo-larval test. However, exposure to a mean DEHP concentration of 0.554 mg/l significantly reduced the growth of Japanese medaka during a 168-day larval test.

Mayer and Sanders (1973) studied the effects of DEHP on the reproduction of zebra fish, \textit{Brachydanio rerio}, and guppies, \textit{Poecilia reticulata}. During 90 days dietary exposure to DEHP, zebra fish were fed on 50 or 100 mg DEHP/kg food and guppies were fed 100 mg/kg. Although the treated zebra fish spawned more often than controls, control fish produced more eggs per spawn. Fry survival was significantly reduced by DEHP. Guppies receiving the dosed diet produced fewer fry per adult and had an 8% incidence of abortions compared with none in the control group (no further statistics are given).

Mayer et al (1977) found lowered collagen levels in three fish species exposed to DEHP; the effects were noted after 150 days at 3.7 µg/l in adult brook trout \textit{(Salvelinus fontinalis)}, after 127 days at 11 µg/l for fathead minnow and after 90 days at 5 µg/l for rainbow trout. The authors found that DEHP did not affect fish growth.

The acute toxicity of DEHP to the embryo-larval stages of fish was studied by Birge et al (1978) and the results of tests on four species of fish are given in Table 6.3.

<table>
<thead>
<tr>
<th>Organism</th>
<th>Stat/flow</th>
<th>LC\textsubscript{50} mg/l</th>
<th>95% Confidence Limits</th>
</tr>
</thead>
<tbody>
<tr>
<td>channel catfish \textit{(Ictalurus punctatus)}</td>
<td>stat$</td>
<td>0.69</td>
<td>0.55 - 0.86</td>
</tr>
<tr>
<td>redear sunfish \textit{(Lepomis microlophus)}</td>
<td>stat$</td>
<td>6.18</td>
<td>4.65 - 8.04</td>
</tr>
<tr>
<td>largemouth bass \textit{(Micropterus salmoides)}</td>
<td>flow\textsuperscript{a}</td>
<td>42.1</td>
<td>24.8 - 77.8</td>
</tr>
<tr>
<td></td>
<td>flow\textsuperscript{b}</td>
<td>32.9</td>
<td>19.8 - 58.9</td>
</tr>
<tr>
<td>rainbow trout \textit{(Oncorhynchus mykiss)}</td>
<td>flow\textsuperscript{a}</td>
<td>139.5</td>
<td>123.2 - 165.2</td>
</tr>
<tr>
<td></td>
<td>flow\textsuperscript{b}</td>
<td>149.2</td>
<td>125.8 - 203.8</td>
</tr>
</tbody>
</table>

Notes: stat$ - static conditions, but water renewed every 12 hours [hardness = 90-115 mg/l CaCO\textsubscript{3}; pH = 7.6-8.1];
flow\textsuperscript{a} - flow-through conditions (DEHP concentration in water continuously maintained) [hardness = 45-55 mg/l CaCO\textsubscript{3}; pH = 7.5-8.0];
flow\textsuperscript{b} - flow-through conditions (DEHP concentration in water continuously maintained) [hardness = 190-225 mg/l CaCO\textsubscript{3}; pH = 7.5-8.0].

In the experiments reported in Table 6.3, exposure was initiated 2 to 6 hours after spawning (except for rainbow trout where exposure was initiated 15 minutes after fertilisation) and continued to 4 days post-hatch. Hatching times varied: 22 days for rainbow trout (13.5-14.3°C); 3 days for catfish (29-31°C); and 3-4 days for bass and sunfish (20-24°C).

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6.1.3 Amphibians

The acute toxicity of DEHP to the tadpoles of Fowler's toad and the leopard frog is given in Table 6.4.

**Table 6.4 : TOXICITY OF DEHP TO THE TADPOLES OF AMPHIBIANS** (Birge et al. 1978)

<table>
<thead>
<tr>
<th>Organism</th>
<th>LC50 mg/l</th>
<th>95% Confidence limits</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fowler's toad</td>
<td>3.88</td>
<td>3.08 - 4.84</td>
</tr>
<tr>
<td>(Bufo fowleri)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>leopard frog</td>
<td>4.44</td>
<td>3.65 - 5.37</td>
</tr>
<tr>
<td>(Rana pipiens)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

In the experiments reported in Table 6.4, exposure was carried out under static conditions, but the water was renewed every 12 hours. Exposure was initiated 2 to 6 hours after spawning and continued to 4 days post-hatch. Hatching times varied between 3 and 4 days, therefore, exposure varied between 7 and 8 days. The conditions for the experiment were: Temperature 20-24°C; hardness 90-115 mg/l CaCO3; pH 7.6-8.1.

Larsson and Thurén (1987) exposed eggs of the moorfrog (Rana arvalis) to sediment concentrations of between 10 and 800 mg/kg DEHP (fresh weight). The number of tadpoles hatching decreased with increasing exposure concentration. In the controls, 90% hatched compared with 50% at 150 mg/kg DEHP and < 30% at > 400 mg/kg. After hatching, the survival of tadpoles was not affected. There was no delay in hatching and no abnormalities in the developing tadpoles exposed to the various DEHP concentrations.

Wams (1987) quoted an unpublished Dutch report which states that an exposure of 2 mg/l DEHP caused a reduction in the growth rate of clawed toad, *Xenopus laevis*, larvae.

6.2 Terrestrial toxicity

6.2.1 Toxicity to plants

Phthalate esters, especially dibutyl phthalate, have been identified as being toxic to some plants in areas of restricted ventilation; however, the available test results indicate that DEHP causes only slight toxic effects (if any).

Herring and Bering (1968) grew spinach and pea plants from seed for 14 to 16 days in soil containing 100 mg/kg DEHP. No effect on plant growth, as measured by height, was found. The effect of DEHP on seed germination was observed by placing seed in Petri dishes containing water with 100 mg/l DEHP. A 40% to 50% reduction in the number of seeds germinating was found.

Lekke and Rasmussen (1983) found DEHP to cause no visible effect on white mustard (*Sinapis alba*), rape seed (*Brassica rapus*) and milfoil (*Achillea millefolium*) when sprayed in the field at up to 8.75 µg/cm² (0.875 kg/ha).

Stanley and Tapp (1982) found that DEHP, at a dose level of 1,000 mg/kg soil, had no effect on the growth of *Brassica rapa* (rape) and only a slight effect on that of *Avena sativa* (oats).
6.2.2 Toxicity to insects

Al Badry and Knowles (1980) did not find DEHP to be toxic to female house flies (Musca domestica) when applied topically or by injection at a concentration of 20 µg/fly, which is equivalent to 1,000 mg/kg. When applied simultaneously with various organophosphates, an antagonistic interaction was apparent. Mortality of 85% to 95% was reduced to less than 10% by the DEHP. However, when DEHP was applied 30 minutes before exposure to an organophosphate concentration causing 10% to 30% mortality, the resulting interaction was synergistic with mortality rising to over 60% and in most cases over 80%.

6.2.3 Toxicity to birds

Hill et al (1975) found no mortality in 10 day old ring-necked pheasants or mallard fed up to 5 g/kg DEHP for 5 days, followed by 3 days on a 'clean' diet.

Wood and Bitman (1984) fed broiler hens on a diet containing 2,000 mg/kg DEHP (226 mg DEHP/day/hen) for 4 weeks. Egg production and body weights were significantly decreased. Ichida et al (1982) found that egg production was stopped and an abnormality of the ovaries was observed in laying hens fed 5,000 or 10,000 mg DEHP/kg diet for up to 230 days.

O'Shea and Stafford (1980) fed starlings (Sturnus vulgaris) on a diet containing 25 or 250 mg/kg DEHP for a 30 day period. The treated birds gained significantly more body weight than controls during the exposure period. At the lower dose rate, food consumption was significantly reduced.

Peakall (1974) fed ring doves (Streptopelia risoria) on a diet containing 10 mg/kg DEHP. No effect on eggshell thickness, ashed egg weight, rate of water loss, surface area or permeability of eggs laid was found.

6.2.4 Toxicity to micro-organisms

A concentration of 100 mg/l DEHP did not significantly reduce numbers of the marine dinoflagellate Gymnodinium breve in culture. However, growth of the culture was inhibited, and an EC₅₀ of 31 mg/l was found (Wilson et al. 1978).

Mathur (1974b) found that DEHP at 0.3% on soil inhibited respiration. Addition of the same amount of DEHP to soil pre-incubated for 14 weeks with DEHP or dioctyl phthalate had no effect on respiration rate. The experiments were conducted at 22°C since no degradation of DEHP occurred at 4°C or 10°C.

In an intermittent flow-through hydrosoil microcosm, DEHP (at 1 and 100 mg/l) produced no significant effect on the numbers of physiological groups of organisms monitored, nor on any of the overall activities which were studied (Mutz and Jones, 1977). This study is reported only in abstract and no further details are available.

Larsson et al (1986) studied the effect of DEHP on microbial activity in sediments. Sediment cores were taken from an eutrophic lake together with the overlying water. The sediment was spiked with between 25 and 400 mg/kg DEHP, spread evenly 5 mm below the sediment surface. Uncontaminated sediments had a significantly higher oxygen uptake than sediment containing DEHP. Microbial activity, as estimated from decreased oxygen saturation in the soil column, was inversely correlated with increasing levels of DEHP in the sediment.

Volskay and Grady (1988) measured the respiratory inhibition of activated sludge micro-organisms caused by chemical contaminants. At its solubility limit of 0.4 mg/l, DEHP was found to cause a 14% reduction in the oxygen consumption of the micro-organisms (compared to the controls) after 30 minutes contact.
6.2.5 Toxicity to earthworms

Neuhauser et al (1986) carried out a series of earthworm toxicity contact tests to determine the toxicity of a range of organic chemicals. Contact testing, in which DEHP could be absorbed into the earthworms' bodies from filter paper impregnated with the chemical, yielded an LC₅₀ value of over 25,000 µg/cm² (µg DEHP per cm² of filter paper), the amount of chemical in the test vial at this concentration exceeded the solubility of DEHP in 1 ml water, i.e. there was undissolved DEHP present in the test vial. Neuhauser et al found that the chemicals which were most toxic to earthworms were also most toxic to rats, by comparison between the LC₅₀ values found and LD₅₀ oral rat toxicities.

6.2.6 Toxicity to mammals

6.2.6.1 Acute toxicity (single exposures)

Data on the acute toxicity of DEHP to mammals are given in Table 6.5. The figures show DEHP to be of low toxicity when administered by the oral route. The one report of a positive result for inhalation toxicity contained insufficient details to make an assessment, and no information on toxicity by dermal exposure was found.

Table 6.5: ACUTE TOXICITY OF DEHP TO MAMMALS

<table>
<thead>
<tr>
<th>Route</th>
<th>Species</th>
<th>Concentration</th>
<th>Effect</th>
<th>Comments</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral</td>
<td>Rat</td>
<td>33.23 g/kg bw</td>
<td>LD₅₀</td>
<td></td>
<td>Homrowski and Nikonorow</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(1959)</td>
</tr>
<tr>
<td>Oral</td>
<td>Rat</td>
<td>34.5 g/kg bw</td>
<td>No deaths</td>
<td>Diarrhoea at &gt; 4.5 g/kg</td>
<td>Hodge (1943)</td>
</tr>
<tr>
<td>Oral</td>
<td>Rat</td>
<td>33.8 g/kg bw</td>
<td>LD₅₀</td>
<td></td>
<td>Krauskopf (1973)</td>
</tr>
<tr>
<td>Oral</td>
<td>Rat</td>
<td>0.8-20 g/kg bw</td>
<td>No effects reported</td>
<td>No effects reported</td>
<td>NTP (1982)</td>
</tr>
<tr>
<td>Oral</td>
<td>Mouse</td>
<td>1.25-20 g/kg bw</td>
<td>LD₅₀</td>
<td></td>
<td>NTP (1982)</td>
</tr>
<tr>
<td>Oral</td>
<td>Mouse</td>
<td>31.6 g/kg bw</td>
<td>LD₅₀</td>
<td></td>
<td>Lawrence et al (1974)</td>
</tr>
<tr>
<td>Oral</td>
<td>Mouse</td>
<td>33.5 g/kg bw</td>
<td>LD₅₀</td>
<td></td>
<td>Krauskopf (1973)</td>
</tr>
<tr>
<td>Oral</td>
<td>Mouse</td>
<td>49.7 g/kg bw</td>
<td>LD₅₀</td>
<td></td>
<td>Yamada (1974)</td>
</tr>
<tr>
<td>Oral</td>
<td>Guinea-pig</td>
<td>26.3 g/kg bw</td>
<td>LD₅₀</td>
<td></td>
<td>Krauskopf (1973)</td>
</tr>
<tr>
<td>Inhalation</td>
<td>Rat</td>
<td>23.7 g/m³</td>
<td>No deaths - 1 hr exposure</td>
<td>No deaths - 1 hr exposure</td>
<td>WARF Institute (1976)</td>
</tr>
<tr>
<td>Inhalation</td>
<td>Rat</td>
<td>0.6 g/m³</td>
<td>No deaths - 6 hr exposure</td>
<td>Death within 4 hrs</td>
<td>Pegg (1979)</td>
</tr>
<tr>
<td>Inhalation</td>
<td>Rat</td>
<td>Air saturated</td>
<td>No deaths</td>
<td></td>
<td>Shaffer et al (1945)</td>
</tr>
</tbody>
</table>

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6.2.6.2 Subacute toxicity (repeated exposures)

The main effects of longer exposures to DEHP are seen in the liver and testes. The US Agency for Toxic Substances and Disease Registry (ATSDR, 1987) commented that exposure to DEHP resulted in adverse hepatic effects in laboratory animals, with high concentrations causing functional hepatic damage, and alterations in the activity of energy-linked enzymes and metabolism of lipids and carbohydrates. It has also been shown to be a peroxisome proliferator, other examples of which have been linked to liver hyperplasia and carcinogenesis. Oral doses have been shown to induce dose-related proliferation of peroxisomes, increase mitochondria and endoplasmic reticulum membranes, induce cytochrome P-450 and induce hepatomegaly associated with hepatic hyperplasia.

HSE (1986) commented that DEHP has been shown to produce hepatomegaly in mice and rats. The characteristics of this were peroxisome proliferation, increases in the levels of peroxisomal marker enzymes and changes in the activities of several enzymes in the microsomes and mitochondria. The no effect level for these changes appeared to be below a dietary level of 0.1% for a sixteen-day period (Fukuhara and Takabatake, 1977).

As regards effects on the testes, HSE (1986) commented that high doses of DEHP caused testicular atrophy in rats. No effect levels for rats have been identified in a number of studies; 0.375% in the diet over a 90-day period (Shaffer et al, 1945), and 0.2% in a 17-week study (Gray et al, 1977). The mono-ester, MEHP, also produced similar effects. In hamsters, MEHP produced testicular effects while DEHP did not; mice and marmosets appeared to be relatively unaffected by either compound.

6.2.6.3 Reproductive effects: teratogenicity, fetotoxicity

A number of studies have provided evidence for the effects of DEHP on reproduction in laboratory animals.

Wistar rats were fed 0.34 and 1.7 g/kg/day DEHP by oral gavage for the first 21 days of gestation, resulting in significant reduction in fetal weights (Nikonorov et al, 1973). There were no excess deaths or deformities.

When rats (Fischer 344) were exposed to 0.5, 1, 1.5 and 2% DEHP in their diet on days 0-20 of gestation, reduced fetal weight and maternal toxicity were noted in all but the lowest exposed group. At the highest level, there was an increase in the number of resorptions, and a reduction in the number of live fetuses born. Again, no excess deformities were observed (Wolkowski-Tyl et al, 1983).

In JCL-ICR mice there was a dose-related increase in embryo-lethality when animals were exposed to 0.01, 0.1 and 1% DEHP in their diet for days 0-18 of gestation (Hamano et al, 1977). At the middle dose level there was a 30% increase in the incidence of anomalies; there were not enough fetuses at the highest dose to draw a conclusion as to its effects. No skeletal abnormalities were observed.

A different strain of mice, ddy-slc + x CBA, were exposed to 0.05, 0.1 and 0.99 g/kg DEHP orally on day 7 of gestation only (Tomita et al, 1982; and Yagi et al, 1980). At the low dose, there was a reduction in the weight of the fetuses. At the highest dose, a significant number of late fetal deaths were seen, as well as gross skeletal anomalies; these latter effects were not seen at the lower two doses. HSE (1986) commented that in this study DEHP showed signs of fetotoxicity and teratogenicity at a dose where maternal toxicity would not be expected.

ICR-JCL mice, fed DEHP at a level of 0.05, 0.1, 0.2, 0.4 and 1% of their diet on days 1-18 of gestation, showed a dose-related decrease in maternal weight at the three highest concentrations (Shioti et al, 1980; and Shioti and Nishimura, 1982). The number of resorptions was dose-related at the lowest three levels; complete resorption occurred at the two highest doses, as well as fetal
deaths. A dose-related trend in the number of malformations was also noted.

Lamb et al (1987) studied the reproductive effects of four phthalate esters on male and female CD-1 mice. The mice were given diets containing 0%, 0.01%, 0.1% or 0.3% DEHP, and were dosed for 7 days prior to and during a 98 day cohabitation period. Reproductive function was evaluated during the cohabitation period by measuring the numbers of litters born per pair, the number of live pups per litter, the pup weight and offspring survival rate. Exposure to DEHP at the two highest levels caused dose-dependent decreases in fertility for both sexes and also decreases in the number and proportion of pups born alive. No treatment related clinical signs of toxicity were observed during the breeding phase of the study.

Overall, it appeared that mice were more sensitive to the effects of DEHP than rats. Doses of up to 1.7 g/kg/day led to reductions in the weight of rat fetuses; in food, levels of up to 2% DEHP increased the number of malformations. In contrast, mice suffered increases in embryo-lethality when exposed to levels of 0.1% DEHP in the diet throughout pregnancy. Low doses led to the production of gross and skeletal anomalies. HSE (1986) suggested that a no effect level for DEHP in mice appeared to be around 0.05% in the diet when administered throughout pregnancy.
7. ENVIRONMENTAL HAZARD ASSESSMENT

7.1 Data availability

Basic hazard assessment involves the comparison of the expected environmental concentration of a chemical, or the exposure of organisms to that chemical, with the concentrations at which it has harmful effects. The first stage of an assessment is thus to ascertain whether the data available are sufficient to allow this procedure to be carried out meaningfully.

For DEHP, the previous sections show that there is a fairly good spread of information on the toxic effects of the chemical. A variety of aquatic organisms have been the subject of both short and longer term toxicity tests, with exposure not only via water alone, but also via contaminated sediments and food (in a few cases). Results are available from relevant tests on mammalian toxicity, covering acute toxicity by various routes and effects on reproduction. There are also some data available on the effects of DEHP exposure on terrestrial ecosystems.

It should be noted that in this report, mammalian toxicity data are used only to help formulate an environmental hazard assessment, hence the lack of data regarding studies on the carcinogenic and mutagenic effects of DEHP which are only relevant for human health assessments.

On the exposure assessment side, there is not a great deal of information concerning DEHP levels in the United Kingdom. Some measurements have been made on levels in estuarine waters, freshwater, and sediments, but there are no values for the soil or air compartments. Background levels in air over the Atlantic Ocean are adequate for establishing baseline DEHP levels.

One way to supplement the small amount of environmental levels data is to use simplified mathematical models of the environment to estimate likely concentrations. The use of a simple distribution model was demonstrated in Section 5.5, and appeared to be useful for predicting concentrations of DEHP in the environment.

In general, more work is needed on the monitoring of DEHP levels in the UK; more thorough knowledge of the levels in water, sediments, soils and vegetation is needed for a full and accurate hazard assessment for the UK to be made.

7.2 Aquatic

Most of the acute toxicity values for DEHP in invertebrates and fish are much higher than the solubility of the compound. Such concentrations tend to be unstable emulsions and the actual exposure of the organisms to DEHP is impossible to determine. It would appear from the data available that DEHP is of low acute toxicity to aquatic organisms. However, a recent study with water flea, *Daphnia pulex*, gave a 48h LC50 of 133 μg/l, indicating higher toxicity to an invertebrate species.

In the longer term tests, a number of effects have been noted at lower DEHP levels, i.e. effects on reproduction in Daphnids at 3 μg/l and effects on collagen levels in brook trout at 3.7 μg/l. Taking into account the possible unreliability of the Daphnid value and considering the latter effect as not necessarily harmful, 3-4 μg/l is the lowest recorded effective concentration found for DEHP.

In general, DEHP levels in fresh surface waters not particularly contaminated with the chemical rarely reach 5 μg/l, and a maximum level of 1.6 μg/l was reported for a river in the Greater Manchester area (UK). Levels in fresh surface waters in industrial areas are higher than this, and a level of 300 μg/l has been reported for Lake Superior (Canada).

Comparing the lowest LC50 value for invertebrates (133 μg/l) with the measured water concentrations, it can be seen that the LC50/concentration ratio is of the order of 27 for the general case (5 μg/l) but less than 1 using worst case data (300 μg/l). Using the same water levels and the lowest LC50 for fish (> 330 μg/l), ratios of 66 and about 1 are found.
Comparing the lowest effective concentration in chronic tests of 3 μg/l with measured water levels yields ratios of less than 1 in both the general case and using worst case data.

It can be seen that existing DEHP levels in fresh surface waters could cause both acute and marginal chronic effects on a range of aquatic organisms, especially in industrially affected areas, for example, Lake Superior in Canada.

Adverse effects on reproduction have been reported via ingestion of DEHP at 50 mg/kg diet in zebra fish. This is higher than levels usually found in biota which might be food organisms. However, levels approaching this value (14.4 mg/kg) have been reported in invertebrates near an industrial discharge point in Sweden.

Effects on the hatchability of frog's eggs were seen at levels of 150 mg/kg DEHP in sediment. Levels of DEHP in UK river sediment have been measured at 30 mg/kg, which would suggest a ratio for this toxic effect of 5.

Effects on microbial activity in sediments have been noted at a concentration of 25 mg/kg DEHP, and comparing this effect level with the measured level in UK sediments gives a ratio of about 1.

DEHP is a highly lipophilic chemical, and has been shown to bioaccumulate in aquatic organisms. DEHP has also been shown to accumulate in fish fed with contaminated Daphnia when this was the only source of the chemical; however, when exposure was from both water and food, the levels reached no higher than those from exposure to water alone. Elimination of body burdens of DEHP occurs over a few days upon transfer to clean water, and a number of species are able to metabolise or degrade the chemical. Thus, bioaccumulation would not appear to present any excess hazard over those already described.

7.3 Terrestrial

There are insufficient data available to perform a satisfactory hazard assessment for terrestrial organisms. DEHP levels in air and water are too low to produce any acute effects, and are unlikely to lead to any long-term effects. No measured concentrations of DEHP in soil were found for the UK. One possible route of exposure could be absorption or uptake by plants, leading to ingestion by higher animals. This could lead to biomagnification, although a study on mallard ducks did not show any signs of this. The likelihood of any acute effects via this route is low, as DEHP has a low oral acute toxicity to mammals.

7.4 Comments

The assessments above mainly consider exposure to background levels of DEHP. The potential for higher levels of DEHP exists, with possible discharges from production and processing facilities leading to higher air levels and possibly water and biota levels; there is also the possibility of DEHP leaching from landfill sites (via plasticised products discarded after use) into groundwater and so into drinking water, although the lipophilicity of the chemical will mitigate against this. Under circumstances of direct discharge from industrial sites there is a possible hazard to biota from DEHP exposure.

These assessments have been made on the basis of the information available. They reveal a number of gaps in the required information, which mean that a complete assessment cannot be made with any great confidence.

It would be useful if more specific UK environmental levels were available in the aqueous and terrestrial compartments in particular. Some toxicity data are available for sediment-dwelling aquatic organisms, but considering that DEHP finds a major sink in sediment, it would be useful to have more data for these organisms, and indeed more information on the levels to which the organisms are commonly exposed in the United Kingdom.
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8. REFERENCES

(The publications in which the references were found are given in parentheses after the source details, when necessary.)


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