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Environmental risk evaluation report: Triphenyl phosphate (CAS no. 115-86-6)

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Steve Killeen

Head of Science

Executive summary

An environmental risk assessment has been carried out for triphenyl phosphate (CAS no. 115-86-6) on the basis of available information and using the methods of a European Technical Guidance Document. This substance is mainly used in Europe for printed circuit boards, thermoplastic/styrenic polymers, thermosets and epoxy resins, and photographic film.

Potential risks are identified for all areas of use for the surface water (fresh and marine), sediment (fresh and marine) and soil compartments, and for exposure through the terrestrial food chain for one use.

Emission estimates are based on information from a number of generic sources, including emission scenario documents and other risk assessments, so they could be refined with more specific information for the substance itself. However, some of the risk characterisation ratios are high and it is unlikely that such information would be sufficient to remove all of the risks identified.

The assessment could also be refined by performing toxicity tests. Although there is a data gap, no further chronic testing on aquatic invertebrates is suggested. Testing on sediment and terrestrial organisms would allow the assessments for these compartments to be refined. In each case, it is likely that three long term-studies would be required. The actual need for testing is closely linked with that for the other triaryl and alkyl/aryl phosphates considered as part of this project. A suggested testing strategy for the group as a whole is outlined in a separate overview document.

The risks to waste water treatment plant, air, secondary poisoning through aquatic food chains, and for humans exposed through the environment, are low for all uses of triphenyl phosphate. In addition, a low risk to surface water and soil is expected for production sites and for all compartments at the regional level.

Triphenyl phosphate does not meet the criteria for a persistent, bioaccumulative and toxic (PBT) or very persistent and very bioaccumulative (vPvB) substance.

Introduction

This report is one of a series of evaluations covering a group of related substances that represent the major aryl phosphate ester products used in Europe:

Triphenyl phosphate
Trixylenyl phosphate
Tricresyl phosphate
Cresyl diphenyl phosphate
Tris(isopropylphenyl) phosphate
Isopropylphenyl diphenyl phosphate
Tertbutylphenyl diphenyl phosphate
2-Ethylhexyl diphenyl phosphate
Isodecyl diphenyl phosphate
Tetraphenyl resorcinol diphosphate

A further substance is known to be commercially available, but it has already been assessed under the Notification of New Substances (NONS) Regulations. Information is also available on some (possibly obsolete) triaryl phosphates that are not thought to be supplied in the EU. This information is summarised in Annex A, but the risks from these products have not been assessed. Information for the group as a whole has also been used in this assessment, where appropriate, to fill any gaps in the database for this particular substance. Annex B discusses the read-across of data between the various phosphate esters considered.

This group was highlighted for assessment during preliminary work for a review of flame retardants (eventually published as Environment Agency 2003), particularly because they are potential replacements for other flame retardants that have already been identified as a risk to health or the environment. Regulators need to understand the potential consequences of such market switches before substantial replacement takes place. These assessments are not intended to provide a basis for comparison between the different aryl phosphates themselves; such a comparison would require consideration of a wider range of factors than are included here (such as human health risks, efficacy, recycling potential and costs). The assessments have been produced as part of the UK Coordinated Chemical Risk Management Programme (UKCCRMP) (http://www.defra.gov.uk/environment/chemicals/ukrisk.htm).

The methodology used in the report follows that given in an EU Technical Guidance Document (TGD)[1] for risk assessment of existing substances. The scientific work was mainly carried out by the Building Research Establishment Ltd (BRE), under contract to the Environment Agency. The review of mammalian toxicity data for the assessment of non-compartment specific effects was carried out by the Institute of Environment and Health, under contract to the Department for Environment, Food and Rural Affairs (Defra).

The Organisation for Economic Cooperation and Development (OECD) High Production Volume (HPV) Screening Initial Data Set programme recently reviewed triphenyl phosphate (OECD 2002). Data from the OECD report was used here largely without further review. In some cases, it was necessary to include further details missing from the OECD report to present a full picture for this assessment. The original source has been cited where this has been done. More recent data for triphenyl phosphate have been included alongside the OECD-reviewed information.

[1] This document has recently been replaced by similar guidance for the REACH Regulation.

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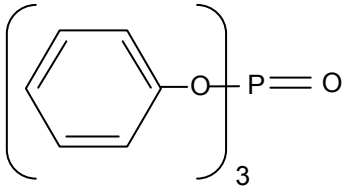
1 General substance information

1.1 Identification of the substance

This assessment considers the following commercial substance.

CAS No:	115-86-6
EINECS No:	204-112-2
EINECS Name:	Phosphoric acid, triphenyl ester
Common Name:	Triphenyl phosphate
Molecular formula:	$C_{18}H_{15}O_4P$
Molecular weight:	326.29 g/mol
Structural formula:	



Other names, abbreviations, trade names and registered trademarks for this substance include the following.

Celluflex TPP®
Disflamoll TP®
Phosflex TPP®
Phosphoric acid, triphenyl ester
Pilabrac 521®
Reofos TPP®
Reomol TPP®
TPP

Some of the trade names and trademarks may refer to older products no longer supplied to the EU, or produced outside the EU, but these are included in the report as they are sometimes referred to in the open literature.

The name triphenyl phosphate is used in this assessment.

1.2 Purity/impurity, additives

1.2.1 Purity/impurities

The purity of triphenyl phosphate is reported to be above 99.6 per cent (OECD 2002).

1.2.2 Additives

Additives are not thought to be present in the commercially supplied products, although some aryl phosphate ester products are sometimes supplied as blends with other (halogenated) flame retardants.

1.2.3 Occurrence in other aryl phosphate esters

Triphenyl phosphate is present in several other commercial aryl phosphate products. Typical levels are given in Table 1.1. The risk evaluation reports of the other aryl phosphate esters in this series give further details of the purity of these products.

Table 1.1 Triphenyl phosphate content of other aryl phosphate esters (data taken from UK environmental risk evaluation reports)

Aryl phosphate type	Triphenyl phosphate content
2-Ethylhexyl diphenyl phosphate	1.5-≤4%
Trixylenyl phosphate	No data
Isodecyl diphenyl phosphate	≤5%-6%
Cresyl diphenyl phosphate	25%
Isopropylphenyl diphenyl phosphate	4-33%
Tricresyl phosphate	0.5%
Tris(isopropylphenyl) phosphate	4%
Tertbutylphenyl diphenyl phosphate	15-20%
Tetraphenyl resorcinol diphosphate	5% maximum 1-2% typically

Triphenyl phosphate is an impurity in these substances because of its presence in the manufacturing process. It is not removed because the product containing it performs the function required. Triphenyl phosphate may also be added deliberately to achieve specific properties. The actual composition will vary between commercial products.

1.3 Physico-chemical properties

Detailed test reports were not available for review, and so the validity of many of the reported values for physico-chemical properties is not always clear.

1.3.1 Physical state (at normal temperature and pressure)

In pure form, the substance is a white or colourless crystalline solid (Ashford 1994, WHO 1991). Commercially supplied products are white flakes or pellets.

1.3.2 Melting point

The melting point has been reported as 49-50°C (Merck Index 1996, IUCLID 2000). A melting point of 49 ± 1°C is given for a commercial triphenyl phosphate product (Bayer 2002) and a melting point of 48°C is reported for another commercial product (Great Lakes Chemical Corporation 2002).

A melting point of 49°C is assumed in this assessment.

1.3.3 Boiling point

Sutton *et al.* (1960) report the boiling point of triphenyl phosphate as 370°C. The boiling point at reduced pressure has been reported as 238°C at 10 mmHg (1.33 kPa) (Boethling and Cooper 1985, Muir 1984), 245°C at 11 mmHg (1.47 kPa) (Merck Index 1996), 220°C-234°C at 5 mmHg (0.67 kPa) (WHO 1991) and 247-250°C at 1.3 kPa (Ashford 1994). Bayer (2002) gives a boiling point of 220°C at 500 Pa for a commercial triphenyl phosphate.

Bayer (2002) report that the decomposition temperature for a commercial triphenyl phosphate was around 500°C. Great Lakes Chemicals Corporation (2002) give the decomposition temperature of another commercial triphenyl phosphate product as above 250°C. Dobry and Keller (1957) reported a decomposition temperature of above 410°C for triphenyl phosphate.

For the purposes of this assessment, the boiling point of triphenyl phosphate at atmospheric pressure is assumed to be 370-500°C.

1.3.4 Density

The density of triphenyl phosphate is 1,190 kg/m^3 at 20°C (Ashford 1994). WHO (1991) reports the relative density of triphenyl phosphate to be 1.185-1.202 at 25°C. Bayer (2002) give a density of 1.205 g/cm^3 at 50°C for a commercial triphenyl phosphate product determined using the DIN 51 757 method.

A relative density of 1.185-1.202 g/cm^3 at 25°C will be used in this assessment.

1.3.5 Vapour pressure

The vapour pressure at ambient temperature is an important physico-chemical property for use in environmental risk assessment, as it is used to estimate both the distribution of a substance in the environment and the volatile releases from products.

Limited reliable data appear to be available for triphenyl phosphate at temperatures around 20-25°C. However, information on vapour pressures at elevated temperature (see above) and boiling points at reduced pressure (see Section 1.3.3) is available.

WHO (1991) reports the vapour pressure of triphenyl phosphate to be 0.15 mmHg (20 Pa) at 150°C and 1.90 mmHg (253 Pa) at 200°C. Sutton *et al.* (1960) reported a vapour pressure of 1.0 mmHg (133 Pa) at 193.5°C. Bayer (2002) report the vapour pressure for a commercial triphenyl phosphate to be below 1 Pa at 20°C and 140 Pa at 200°C. Muir (1994) reports vapour pressures of 1.3 mmHg (173 Pa) at 200°C and 18.2 mmHg (2,426 Pa) at 250°C.

A vapour pressure of 1.5×10^{-6} (no units given) is reported in IUCLID (2000). The original source of this data (Boethling and Cooper 1985) indicates this is an estimated value of 1.5×10^{-6} mmHg (1.2×10^{-4} Pa) that appears to be based on a boiling point of 220°C at 5 mmHg. The paper gives other estimates of 1.2×10^{-6} mmHg and 8.0×10^{-7} mmHg (1.6×10^{-4} Pa and 1.1×10^{-4} Pa) based on boiling points of 238°C at 10 mmHg and 245°C at 11 mmHg.

The vapour pressure of triphenyl phosphate at 25°C was reported to be 5.3×10^{-5} mmHg (7.1×10^{-3} Pa) (Huckins *et al.* 1991 referencing an unpublished report). No further details appear to be available.

The vapour pressure of a pure substance is related to the temperature within a limited temperature range according to the simplified Clapeyron-Clausius equation:

$$\log (\text{vapour pressure}) = [\underline{\Delta}H_v/2.3RT] + \text{constant}$$

where vapour pressure is in Pa
ΔH_v = heat of vapourization in J/mol
R = the universal gas constant 8.314 J/mol K
T = temperature in K

Figure 1.1 shows a plot of log (vapour pressure or reduced pressure (Pa)) against 1/(temperature or boiling point (K)) for the available measured data. This gives a straight line plot corresponding to the following regression equation.

$$\log (\text{vapour pressure (Pa)}) = [-3{,}870.9 \times 1/(\text{temperature (K)})] + 10.598$$

The value of ΔH_v for triphenyl phosphate is estimated to be -74,020 J/mol from the slope of the plot.

Using this equation, the vapour pressure of triphenyl phosphate can be estimated as 2.4×10^{-3} Pa at 20°C, 4.1×10^{-3} Pa at 25°C, 28 Pa at 150°C and 260 Pa at 200°C. These values are in reasonable agreement with the data reported by Dobry and Keller (1957) who estimated the vapour pressure at 20°C to be 4.8×10^{-4} Pa or 8.35×10^{-4} Pa at 25°C for purified triphenyl phosphate, extrapolated from data obtained at elevated temperatures, and the value of 7.1×10^{-3} Pa at 25°C reported by Huckins *et al.* (1991). It Note these estimated values are for the sub-cooled liquid, where triphenyl phosphate exists as a solid at 20°C (melting point 49°C). Also, the value for ΔH_v may vary with temperature and so could introduce further errors in extrapolating the data obtained at elevated temperatures to ambient temperatures.

The TGD provides an equation relating the vapour pressure of the sub-cooled liquid to that of the solid:

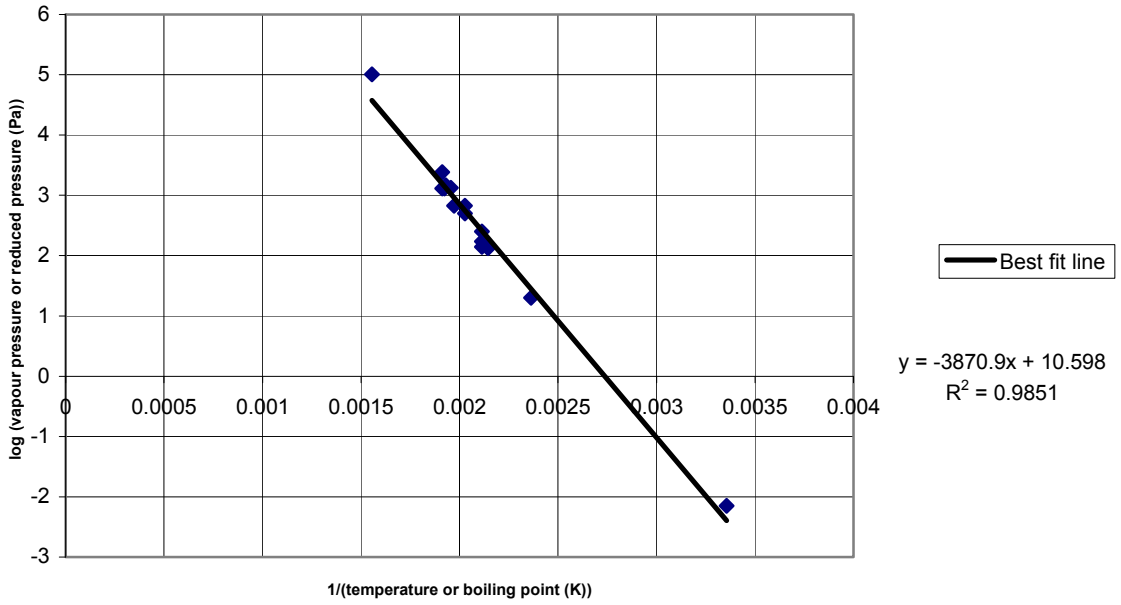
$$VPL = VP / [e^{6.79 \,.\, (1- TEMPmelt/TEMP)}] \quad \text{(Mackay 1991)}$$

where vapour pressure is in Pa
TEMP = environmental temperature in K
TEMPmelt = melting point of substance in K

VPL = sub-cooled liquid vapour pressure in Pa

Using this equation, the vapour pressure for the solid triphenyl phosphate can be estimated as 1.2×10^{-3} Pa at 20°C and 2.4×10^{-3} Pa at 25°C.

Figure 1.1 Plot of log (vapour pressure or reduced pressure (Pa)) against 1/(temperature or boiling point (K))



A vapour pressure (at 25°C) of 2.08×10^{-7} mmHg (2.8×10^{-5} Pa) can be estimated for triphenyl phosphate from its structure using the Syracuse Research Corporation MPBPWIN (version 1.28) software (modified Grain method). Boethling and Cooper (1985) estimated a vapour pressure at 25°C of 8.0×10^{-7} to 1.5×10^{-6} mmHg (1.1×10^{-4} to 2.0×10^{-4} Pa) from the boiling point of triphenyl phosphate (Grain method).

A vapour pressure of 2.4×10^{-3} Pa at 20°C and 4.1×10^{-3} Pa at 25°C for the sub-cooled liquid and 1.2×10^{-3} Pa at 20°C and 2.4×10^{-3} Pa at 25°C for the solid (as derived from the above analysis of all the available data) will be used in this risk assessment.

1.3.6 Water solubility

Saeger *et al.* (1979) determined the solubility of triphenyl phosphate in water using a shake flask method. The substance used was a commercial product consisting of above 90 per cent triphenyl phosphate. In the experiment, 25 ml of the phosphate ester was added to 500 ml of purified water and shaken for 48 hours. The solution was then allowed to stand for one week in the dark before the aqueous phase was centrifuged at 20,000 g for one hour to remove droplets of undissolved substance. The aqueous phase was then extracted twice with methylene dichloride and the extracts were analysed for triphenyl phosphate by a gas chromatography method (the centrifugation/ extraction/analysis steps were carried out in duplicate and gave a mean relative average deviation of 13 per cent). The solubility of the substance tested was determined to be 1.9 mg/l at room temperature.

Anderson *et al.* (1993) reported a water solubility of 2.1 mg/l at 25°C for triphenyl phosphate. No further details were given, but it may be from the Ofstad and Sletten (1985) study below.

Ofstad and Sletten (1985) determined the water solubility of a commercial tricresyl phosphate product (which contained a substantial amount of triphenyl phosphate) using the OECD 105 column elution method using two different flow rates at 25°C. The product tested contained around 25 per cent triphenyl phosphate along with around twenty other triaryl phosphates. The analytical method was able to distinguish between the main

components of the product and the water solubility was determined to be 3.1 mg/l (sum of all components), 0.34 mg/l for the tricresyl phosphate component (all isomers), 2.1 mg/l for the triphenyl phosphate component and 0.11 mg/l for the trixylenyl phosphate component (all isomers). The value obtained in this study for triphenyl phosphate alone agrees well with that determined above by Saeger *et al.* (1979).

Hollifield (1979) estimated a water solubility for triphenyl phosphate of 0.73 mg/l at 24°C using a nephelometric technique. The method involved dissolving the substance in a water miscible solvent (ethanol or acetone) and measuring the turbidity of dilutions of increasing amounts of this solution in water. A turbidity curve was then constructed and extrapolation of this curve to the blank value provided an estimate of the water solubility of the substance.

A water solubility of around 1 mg/l can be estimated for triphenyl phosphate using the Syracuse Research Corporation WSKOW version 1.30 software (the estimate is based on a log K_{ow} of 4.59).

A water solubility of 1.9 mg/l at room temperature is assumed in this assessment.

Triphenyl phosphate is reported to be 'soluble' in organic solvents such as benzene, chloroform, dimethyl ether and acetone, and is 'moderately soluble' in ethanol (WHO 1991).

1.3.7 Octanol-water partition coefficient (log K_{ow})

The octanol-water partition coefficient of a triphenyl phosphate was determined using a shake flask method (Saeger *et al.* 1979). The substance used was a commercial product consisting of above 90 per cent triphenyl phosphate. In the study, the substance was dissolved in n-octanol (at least two concentrations were tested between 100 mg/kg and 10,000 mg/kg) and 100 ml of this solution was shaken with 500 ml of purified water for 48 hours in the dark. The mixture was then allowed to stand for seven days in the dark before the concentration in the water phase (based on the sum of the major components of the product found in the gas chromatography trace) was determined (as only small amounts of the test substance were found to partition into the water phase, the concentration of the substance in the n-octanol phase was taken to be the starting concentration). The K_{ow} obtained was 42,500 (log K_{ow} = 4.63).

Mayer *et al.* (1981 and 1993) reported a K_{ow} value for triphenyl phosphate of 42,000 (log K_{ow} = 4.62). No further details were given although Mayer *et al.* (1981) indicates that the value may have come from the Saeger *et al.* (1979) determination above. A similar value of 4.61 is reported by Kenmotsu *et al.* (1980).

A log K_{ow} value of 3.9 was reported for triphenyl phosphate by Bengtsson *et al.* (1986). The value was from an unpublished source.

Sasaki *et al.* (1981) used a shake flask method to determine the octanol-water partition coefficient. In the test, 50 mg of triphenyl phosphate (purity not given) was added to a separating funnel containing 50 ml of n-octanol (saturated with water) and distilled water. The flask was shaken for two hours and then allowed to stand overnight. The concentration of triphenyl phosphate in the water phase was then measured and the concentration in n-octanol was estimated by difference. The octanol-water partition coefficient determined was 57,260 (log K_{ow} = 4.76) at room temperature.

Renberg *et al.* (1980) determined the octanol-water partition coefficient for a triphenyl phosphate (the same substance as used by Saeger *et al.* 1979 above) using a high performance thin layer chromatography (HPTLC) method. The partition coefficient determined (log value) was 3.15. This value is considerably lower than those found in other studies.

Lo and Hsieh (2000) determined a log K_{ow} value of 3.4 for triphenyl phosphate (purity 98 per cent) using a high performance liquid chromatography (HPLC) method. The reference compounds used in the study were benzene, bromobenzene, biphenyl, bibenzyl and hexachlorobenzene and had log K_{ow} values in the range 2.13 to 6.42.

A log K_{ow} of 4.7 can be estimated for triphenyl phosphate from its structure using the Syracuse Research Corporation Log K_{ow} (version 1.60) software. The software also reports an experimental value of 4.59.

A log K_{ow} value of 4.63 as determined by Saeger *et al.* (1979) is used in this assessment.

1.3.8 Hazardous physico-chemical properties

The flash point of a triphenyl phosphate is reported to be around 220-225°C (WHO 1991). Great Lakes Chemical Corporation (2002) report a flash point above 220°C for a commercial product. Bayer (2002) reports a similar flash point of above 230°C for another commercial triphenyl phosphate product.

No data could be located on autoignition, explosivity or the oxidising properties of this substance. Triphenyl phosphate is expected to decompose at elevated temperatures.

1.3.9 Henry's law constant

A Henry's law constant of 0.21 Pa m^3/mol at around 20°C or 0.41 Pa m^3/mol at around 25°C can be estimated for triphenyl phosphate based on the water solubility of 1.9 mg/l (see Section 1.3.5) and vapour pressure of 1.2×10^{-3} Pa at 20°C or 2.4×10^{-3} Pa at 25°C (see Section 1.3.6) for solid triphenyl phosphate.

A Henry's law constant of 3.98×10^{-8} atm m^3/mol (0.0040 Pa m^3/ mol) at 25°C can be estimated for triphenyl phosphate from chemical structure (bond contribution method) using the Syracuse Research Corporation HENRYWIN (version 3.00) software. The software also gives a value of 3.31×10^{-6} atm m^3/mol (0.33 Pa m^3/mol) at 25°C based on an experimental water solubility of 1.9 mg/l and an experimental vapour pressure of 6.28×10^{-6} mmHg (8.4×10^{-4} Pa) [Note: it is not clear how this value of Henry's law constant is obtained from the reported solubility and vapour pressure. Based on these data, the Henry's law constant should be around 1.4×10^{-6} atm m^3/mol (or 0.14 Pa m^3/mol)].

A Henry's law constant of 0.21 Pa m^3/mol at 20°C and 0.41 Pa m^3/mol at 25°C is used in this assessment, as it is consistent with the vapour pressure and water solubility for triphenyl phosphate used here.

1.3.10 Summary of physico-chemical properties

A summary of physico-chemical data used for this assessment is given in Table 1.2.

Table 1.2 Summary of environmentally relevant physico-chemical properties of triphenyl phosphate

Property	Value
Melting point	49°C
Boiling point (at atmospheric pressure)	370-500°C
Relative density	1.185-1.202 at 25°C
Vapour pressure	2.4×10^{-3} Pa at 20°C or 4.1×10^{-3} Pa at 25°C (sub-cooled liquid) 1.2×10^{-3} Pa at 20°C or 2.4×10^{-3} Pa at 25°C (solid)
Water solubility	1.9 mg/l at room temperature
Octanol-water partition coefficient (log value)	4.63
Henry's law constant	0.21 Pa m^3/mol at 20°C and 0.41 Pa m^3/mol at 25°C

2 General information on exposure

2.1 Production

Triphenyl phosphate is produced by the reaction of phenol with phosphorus oxychloride (Ashford 1994, WHO 1991).

Triphenyl phosphate is usually produced as flakes or as a liquid shipped in heated vessels (Weil 1993).

WHO (1991) reported that around 7,250 tonnes of triphenyl phosphate were produced in the United States in 1977 and around 3,750 tonnes were produced in Japan in 1984.

There are two known European production sites (including Chemtura (formerly Great Lakes), UK) and one additional European supplier. Information on production volume and market size is therefore confidential. It is possible that other companies may supply this substance, but no further information is available for this report.

2.2 Use

2.2.1 General introduction

Aryl phosphate flame retardants were first commercialised in the early twentieth century for use in flammable plastics such as cellulose nitrate and later for cellulose acetate (Weil 1993). Use in cellulose products is still significant, but the largest application is now in plasticized vinyl polymers. The main applications of these products are in wire and cable insulation, connectors, automotive interiors, vinyl moisture barriers, furniture upholstery, conveyor belts (for mining) and vinyl foams.

In addition to their use as flame retardants in polymer systems, triaryl phosphates are also used as flame retardant hydraulic fluids, lubricants and lubricant additives (Weil 1993). Small amounts are also reported to be used as non-flammable dispersing media for peroxide catalysts.

2.2.2 General information on the use of triphenyl phosphate

One of the first applications for triphenyl phosphate was as a flame retardant/plasticizer for cellulose acetate safety film. Current flame retardant/plasticizer applications include cellulose nitrate, various coatings, triacetate film and sheet, rigid urethane foam and engineering thermoplastics such as polyphenylene-high impact polystyrene and acrylonitrile-styrene-butadiene (ABS)-polycarbonate blends (Weil 1993). Other reported uses include as a non-combustible substitute for camphor in celluloid (which is used to render acetylcellulose, nitrocellulose and airplane “dope” stable and fireproof), for impregnating roofing paper, a plasticizer in lacquers and varnishes and as a plasticizer in vinyl automotive upholstery (WHO 1991).

Triphenyl phosphate or diethyl phthalate are the plasticisers most commonly used in cellulose acetates and are used at a loading of 10 to 20 per cent by weight (Williamson *et al.* 1993). A major use of cellulose triacetate is as a photographic film base. Louvet *et al.* (1995) found that triphenyl phosphate was present at 7 to 8 per cent by weight in degraded cellulose nitrate film (including the gelatine layer).

Levchik *et al.* (2000) indicated that aromatic phosphates, including triphenyl phosphate, are the primary flame retardants for polycarbonate/ABS.

Carlsson *et al.* (2000) analysed 18 new computer monitor cases for the presence of organic phosphate esters. Triphenyl phosphate was found to be present in ten of the monitors at around 8 to 10 per cent by weight (in four monitors) or 0.3-0.5 per cent by weight (in six monitors). No other phosphate esters were found in the monitor housing and no triphenyl phosphate (or other organic phosphate esters) were found in the plastic material of the computer chassis or the computer printed circuit board.

The substance is also used as an extreme pressure additive in lubricants and hydraulic fluids (Ashford 1994, WHO 1991). Hydraulic fluid formulations containing tricresyl phosphate and/or triphenyl phosphate as the major component have been used by the United States air force since the 1970s (David and Seiber 1999).

A breakdown of the use of triphenyl phosphate in Japan in 1984 is available (WHO 1991). Out of a total of 3,750 tonnes, 3,200 tonnes (85 per cent) were used as a flame retardant in phenolic and phenylene oxide-based resins for the manufacture of electrical and automobile components, around 500 tonnes were used as a flame-retardant plasticizer in cellulose acetate for photographic films and around 50 tonnes were used for other miscellaneous applications.

2.2.3 Current use of triphenyl phosphate in the EU

Information on the sales of triphenyl phosphate into the EU has been provided by the relevant supplier companies for the year 2005. The specific figures are confidential but the major current areas of use include printed circuit boards, thermoplastic/styrenic polymers, thermosets and epoxy resins, and photographic film.

These areas of application will be considered in the risk assessment. In addition, a small amount appears to be used in flea collars for dogs (VMD 2008). This use is not addressed in this assessment.

3 Environmental exposure

This assessment has been prepared in accordance with the principles of Council Regulation (EEC) 793/93 (the Existing Substances Regulation or ESR)[2] and the methods laid down in Commission Regulation (EC) 1488/94[3] , which is supported by a technical guidance document or 'TGD' (EC 2003). The European Union System for the Evaluation of Substances (EUSES) computer program[4] (v2.0.3) implements the TGD models. The EUSES output file for this assessment is confidential because of the information it contains on tonnage and use pattern; it may be obtained on request to the Environment Agency.

The assessment is generic, representing a *realistic worst case approach* for a hypothetical environment that broadly reflects average European conditions. It uses a number of assumptions (such as a fixed river dilution level), and further details can be found in the TGD. The assessment is based on estimated sales figures for Europe and some site-specific information. Since these are confidential, the calculations are presented in the Confidential Annex, but they are discussed qualitatively in the report as appropriate.

3.1 Environmental fate and distribution

3.1.1 Degradation

Abiotic degradation

Atmospheric photooxidation

A rate constant for reaction of triphenyl phosphate with atmospheric hydroxyl radicals of 10.8×10^{-12} cm^3/molecule s can be estimated from its structure using the Syracuse Research Corporation AOP (version 1.86) software. This program implements the method recommended in the TGD for estimating the rate constant.

Using an atmospheric hydroxyl radical concentration of 5×10^5 molecules/cm^3, a half-life for the reaction in air is estimated to be 36 hours.

Hydrolysis

The hydrolysis of triphenyl phosphate (no information on purity) at various pHs was studied by Howard and Deo (1979). Saturated solutions of the test substance were prepared by shaking an excess of the substance with buffered distilled water or natural lake/river water for two hours, followed by filtration (11 μm) to remove the undissolved material. The concentration of triphenyl phosphate in the solution was then determined. The solution was incubated at 21°C. The concentration of the test substance present and the main hydrolysis products formed was determined at various time periods. The rate of hydrolysis to diphenyl phosphate was found to be higher in alkaline solution than acid solution. The measured half-life for hydrolysis was 1.3 days at pH 9.5 and 7.5 days at pH 8.2. The half-lives at pH 6.7 and pH 4.5 were too long to be determined reliably by the method used. The diphenyl phosphate formed was thought to be relatively stable under

[2] O.J. No L 084, 05/04/1993 p. 0001–0075.
[3] O.J. No L 161, 29/06/1994 p. 0003–0011.
[4] Available from the European Chemicals Bureau, http://ecb.jrc.ec.europa.eu/.

these experimental conditions, as no monophenyl phosphate was found to be present in the studies at any pH.

Experiments using natural waters (from Lake Onondaga (pH 7.8), Lake Ontario (pH 8.2) and Seneca River (pH 8.2)) all showed a lag period of around two days prior to the onset of degradation of the triphenyl phosphate. After this lag phase, the triphenyl phosphate was found to degrade rapidly in all three waters, at a rate higher than would be expected based on the above experiments with buffered distilled water. For example, the half-life was around one day in Lake Ontario water and two days in Seneca River water. This indicates that microbial degradation was probably the dominant degradation process in these samples (although the water samples were filtered prior to use, the size of the filter (11 μm) was chosen so as not to remove microorganisms from the water).

Howard and Deo (1979) also investigated the degradation of several commercial products (Kronitex R, Santicizer 140, Phosflex 41P and Fyrquel GT) some of which may contain small amounts of triphenyl phosphate. These tests were carried out in Lake Ontario water in the same way as above and again indicated that degradation was slow for the first two days, followed by a rapid degradation. Again, these results may reflect biodegradation rather than hydrolysis.

Wolfe (1980) developed linear free-energy relationships to estimate the rate constants for neutral and alkaline hydrolysis of triaryl phosphates using published information on hydrolysis. Under alkaline conditions, the second-order reaction rate constant for hydrolysis was found to correlate with the sum of the Hammett (σ) substituent constants for the aryl substitutents and the following equation was derived:

$$\log k = 1.40 \times \sigma - 0.47$$

where k = second-order rate constant for hydrolysis at 30°C (l/mol s)
σ = sum of Hammet substituent constants

For triphenyl phosphate, σ equals zero and so the second-order hydrolysis rate constant is 0.33 l/mol s. Using this value, the hydrolysis half-life at any alkaline pH can be estimated. For example, at pH 8 the concentration of hydroxyl anions present is 10^{-6} mol/l and so the pseudo first-order hydrolysis reaction rate constant for triphenyl phosphate at this pH is around 3.3×10^{-7} s^{-1}. This is equivalent to a hydrolysis half-life of around 24 days.

For hydrolysis under neutral conditions, the following equation was derived:

$$\log k = -0.95 \times pKa - 1.20$$

where k = first-order rate constant for neutral hydrolysis at 25°C (s^{-1})
$pKa = -\log_{10}$ {acid dissociation constant for the phenolic leaving group}

For triphenyl phosphate, the pKa of the leaving phenol group is around 10. This leads to an estimated value for the rate constant for the neutral hydrolysis of 2×10^{-11} s^{-1} and an estimated half-life for hydrolysis of 1,100 years. Note that alkaline hydrolysis would also occur at this pH, with a half-life estimated to be greater than 240 days.

Great Lakes Chemical Corporation (2003) indicate that hydrolysis of aryl phosphates can also occur under acidic conditions and packages are usually added to hydraulic fluids to delay the onset of hydrolysis during the service life of the products. The standard test for such fluids is the ‘coke bottle’ test. In this test, the fluid is placed in a coke bottle with distilled water (sometimes with catalytic metals such as copper) and the degradation is followed by an increase in acidity (the initial hydrolysis product would be diaryl phosphates (diesters of phosphoric acid), which are acidic). The rate of hydrolysis generally increases as the acidity increases during the test. However, although this indicates that hydrolysis of aryl phosphates can occur at acidic pHs, the autocatalysis seen in the test as a result of the formation of acidic products (resulting in an increase in acidity) is unlikely to occur in the environment, owing to its natural buffering capacity.

The hydrolysis of triphenyl phosphate (no information on purity) was investigated by Mayer *et al.* (1981). The experiments were carried out at 25°C using sterile 0.05 M buffered water at pH 5 (potassium hydrogen phthalate/sodium hydroxide), pH 7 (potassium orthophosphate/sodium orthophosphate) and pH 9 (boric acid/sodium hydroxide). The test substance was added as solution in methanol to give an initial concentration of 50 μg/l and the hydrolysis half-life was determined to be above 28 days at pH 5, 19 days at pH 7 and three days at pH 9. The products from the hydrolysis were not determined but it was thought that the mechanism involved cleavage of the P-O bond to form phenol.

David and Seiber (1999) carried out a number of experiments investigating the abiotic degradation of triphenyl phosphate (purity above 99 per cent) in water and soil-water slurries. Sodium perborate was used in some of the experiments as a source of hydroperoxyl ions, which are known to react as nucleophiles at the phosphorus centre of phosphate esters at a rate of 10 to 100 times that of the hydroxyl ion under alkaline conditions.

The hydrolysis studies in water were carried out by adding 1 ml of a solution of the phosphate ester to 1 to 2 litres of deionised tap water. The initial concentration present was then measured (the initial concentration was always below the water solubility of the substance) and the degradation reaction was started by adding either sodium carbonate directly to the test vessel to give a 0.007 M solution (pH 10.7) or sodium perborate to give a 0.03 M solution (pH 10.3). Hydrolysis was then followed by analyses of water samples at various time points for the disappearance of the starting phosphate ester. Each experiment was carried out in triplicate. The half-life for hydrolysis was found to be 920 minutes in the experiments with sodium carbonate (hydroxyl ion acting as the nucleophile) and four minutes in the experiments with sodium perborate (hydroperoxyl ion acting as a nucleophile). Diphenyl phosphate was the hydrolysis product formed in both sets of experiments, and was relatively stable to further hydrolysis (no decrease in concentration was seen in the sodium perborate solutions over three hours).

The soil used in the soil-slurry experiments was a loamy sand soil consisting of 82 per cent sand, ten per cent silt and eight per cent clay with an organic matter content of 0.3-0.35 per cent and a water content of below 3 per cent. The soil was spiked with a mixture of organophosphate chemicals, including triphenyl phosphate, tributyl phosphate and a mixture of tricresyl phosphate isomers (*ortho*-, *meta*- and *para*- isomers) to give a total concentration of 50-100 mg/kg. The soil-water slurries were prepared by adding two grams of the spiked soil to 100 ml of deionised tap water and shaking for 72 hours. After this equilibration period, the degradation experiments were started by adding a solution of sodium carbonate (to give a final concentration of 0.007 M and a pH of 10.4) or sodium perborate (to give a final concentration of 0.03 M and a pH of 10.1). Controls at pH 6.8 were also run. The slurries were then shaken for between four and 408 hours and the concentrations of test substances determined at various intervals. Each experiment was carried out in duplicate. The degradation of phosphate esters seen did not fit a first- or second-order kinetic model. The results were best explained in terms of the rate-limiting step being desorption of the phosphate esters from the solid phase, followed by a rapid hydrolysis of the desorbed substance. The time for 90 per cent degradation was estimated on the basis of this model to be under eight hours for triphenyl phosphate in the experiments with sodium perborate. The amount of triphenyl phosphate remaining after ten days was determined to be zero per cent in the sodium perborate experiments, two per cent in the sodium carbonate experiments and 79 per cent in water at pH 6.8.

IUCLID (2000) reports further results on the hydrolysis of triphenyl phosphate from experiments carried out by Ishikawa *et al.* (1985a). The experiments were carried out using 0.1 g/l solutions of triphenyl phosphate in water (acetone may have been used as a cosolvent) and the solutions also appeared to have had chlorine present at 3 mg/l to ensure sterile conditions. The extent of hydrolysis/degradation in the experiments was 100

per cent after ten minutes at pH 13, three per cent after 24 hours and five per cent after 48 hours at pH 7, and 20 per cent after 48 hours at pH 3. Experiments with higher concentrations of chlorine (1,000 mg/l) at pH 7 showed more rapid degradation, with 20 per cent degradation seen after 24 hours and 25 per cent degradation after 48 hours.

Hydrolysis is considered further in Annex C.

Photolysis

IUCLID (2000) reports results from experiments carried out by Ishikawa *et al.* (1985a) on the photolysis of solutions of triphenyl phosphate using ultraviolet (UV) light. Experiments were carried out using 0.1 g/l solutions of triphenyl phosphate in water (acetone was used as a cosolvent) using both low pressure (15W) and high pressure (100W) mercury lamps as the UV source. The experiments showed 100 per cent degradation of the triphenyl phosphate after one hour irradiation using the low pressure lamp and 100 per cent degradation after 20 minutes exposure using the high pressure lamp. Given that these experiments were carried out using UV radiation, and acetone (a known photosensitiser) was also present, it is not possible to use these results to predict the rate of photodegradation of triphenyl phosphate in the environment.

Further photolysis experiments using UV radiation were carried out by Ishikawa *et al*., (1992). In these experiments, triphenyl phosphate was added to 100 ml of water and was then dissolved/dispersed by ultrasonication for one hour. The solution was mixed with 1,900 ml (giving a final concentration of around 0.1 mg/l) for 30 minutes. After adjustment to the desired pH with hydrochloric acid or sodium hydroxide, the solution was then irradiated with a 15 W low-pressure mercury lamp and samples were collected from the solution periodically and analysed for the presence of triphenyl phosphate. The disappearance of triphenyl phosphate from the system was found to be rapid and to follow first-order kinetics. The rate constant for degradation was above 40 h^{-1} at pH 3 and at pH 10 (corresponding to half-lives of under one minute at both pHs). Further experiments were carried out using higher concentrations of the test substance (3×10^{-4} mol/l = 0.1 g/l), to identify the photodecomposition products. These experiments were carried out in water without pH adjustment and in water with an initial pH of 12. The products formed were identified as phosphoric acid and phenols (only found in the pH 12 experiment). The yield of phenol after three hours irradiation under the alkaline conditions was found to be around 23 per cent and the phenols were found to be decomposed by further irradiation. Under more acidic conditions, the decomposition of phenol was thought to be more rapid than its formation.

Muir (1984) and Boethling and Cooper (1985) reported that triphenyl phosphate formed some diaryl products (around two per cent yield) when irradiated using a medium pressure mercury arc lamp in ethanol (concentration 0.02 M).

Biodegradation

Dearden and Cronin (1996) reported the result of a MITI I ready biodegradation test (equivalent to OECD 301C) with triphenyl phosphate. The degradation, as determined by biological oxygen demand (BOD) at the end of the study, was 90 per cent. No experimental details of the specific test were given. This is likely to be the same study as reported in the Japanese Chemicals Evaluation and Research Institute database on degradation data (CERI 2003). This database indicates that the test was carried out at 25°C using an activated sludge concentration of 30 mg/l and a triphenyl phosphate concentration of 100 mg/l. The biodegradation was monitored using three methods: biochemical oxygen demand (BOD), total organic carbon (TOC) and parent compound analysis by HPLC. The percentage degradation seen in the study based on BOD determination was 83 per cent, 92 per cent and 94 per cent in three replicates, giving an

average degradation of 90 per cent. The results based on TOC and parent compound analysis showed 95 per cent and 96 per cent degradation respectively.

Bayer (2002) reports that more than 70 per cent degradation of a commercial triphenyl phosphate occurred in an unpublished closed bottle test.

IUCLID (2000) indicates that 94 per cent degradation of triphenyl phosphate was obtained in an unpublished coupled units aerobic sewage treatment simulation test (OECD 303A). The inoculum used in the test was an adapted synthetic mixture of activated sludge from a sewage treatment plant, river water/sediment and garden soil. The results were reported to indicate that the substance was inherently biodegradable.

IUCLID (2000) reports results from experiments carried out by Ishikawa *et al.* (1985a) on the biodegradation of triphenyl phosphate using return sludge from a municipal sewage treatment plant. Two concentrations of triphenyl phosphate were used (0.1 and 1.0 mg/l) and the degradation seen was reported to be around 40 per cent after 48 hours incubation at both concentrations tested.

Boethling and Cooper (1985) report the results of an unpublished study using a commercial isopropylated triphenyl phosphate (main components triphenyl phosphate, 2-isopropylphenyl diphenyl phosphate and 4-isopropylphenyl diphenyl phosphate). In this study, activated sludge mixed liquor was acclimated to progressively higher concentrations of the test substance. At the start of the test, the acclimated liquor was diluted 1:10 with a mineral salts medium and the test substance was added as sole source of carbon. All three main components were found to be above 98 per cent degraded within seven days. All components of the same substance were found to be extensively degraded in a river die-away test over seven days (the half-life for triphenyl phosphate was around three days).

Mayer *et al.* (1981) reported the results of standard biodegradation tests for triphenyl phosphate (no information on purity). The results are reported in summary form only and so full details of the specific tests are not available. The ultimate degradation was studied using the Thompson-Durthie-Sturm procedure with an acclimated bacterial seed and this showed 82 per cent mineralisation (expressed as percentage of theoretical CO_2 evolution). Primary degradation was studied using a semi-continuous activated sludge (SCAS) unit and the river die-away procedure. The SCAS unit showed 96 ± 2 per cent primary degradation of triphenyl phosphate and the primary degradation half-life determined in the river die-away test was two to four days (initial test concentration was 1 mg/l).

Triphenyl phosphate was shown to undergo 77 per cent mineralisation (expressed as percentage of theoretical CO_2 evolution) in an unpublished test using a shake flask procedure (personal communication). In the test, 100 ml of acclimated bacterial seed was mixed with 400 ml of standard BOD water and 15 mg of the triphenyl phosphate was added. After aeration, the flasks were sealed and agitated on a rotary shaker at 80 rpm in the dark for up to 28-35 days at ambient temperature.

Mayer *et al.* (1981) also investigated the biodegradation of the triphenyl phosphate component of a commercial product Pydraul 50E (consisting of 36 per cent triphenyl phosphate, 40 per cent nonylphenyl diphenyl phosphate and 22 per cent cumylphenyl diphenyl phosphate) using a microcosm. The microcosm consisted of 40-litre tanks containing water (20 litres) and sediment (8 litres) from the littoral region of a spring-fed freshwater lake. The microcosm was allowed to stabilise for six weeks and then core chambers were created in the tank by inserting 15 glass cylinders through the water column and sediment (each core chamber contained 150 ml of water and 35 cm^3 of sediment). Each chamber was then supplied with carbon dioxide-free air or pure nitrogen and the test substance was added to the chambers as an acetone solution to give an initial concentration of 1 mg/l. Water samples were analysed for the components of the test substance at days 0, 3, 7,zero, three, seven, 14, 21 and 28 of the study and the

exiting gases from the chambers were passed through an absorbent resin to collect any volatile compounds emitted. The half-life for disappearance of the triphenyl phosphate from the water column (due to degradation and adsorption to the sediment) was determined to be three days in the test system versus 7 to 8 days in sterile controls. Light or purge gas (air or nitrogen) appeared to have little effect on the rate of disappearance from the water column. When the amounts of triphenyl phosphate present in the water column and the sediment phase at the end of the 28-day study were taken into account, the primary degradation seen amounted to more than 80 per cent of the initial amount of triphenyl phosphate added to the test system.

Saeger *et al.* (1979) determined the biodegradation of a triphenyl phosphate using various test systems. The substance used was a commercial product consisting of above 90 per cent triphenyl phosphate. The first test investigated the primary degradation of the test substance using a river die-away method. The water used in the test was settled Mississippi River water. The test substance (at a concentration of 1 mg/l) was added to the water and the test vessels (bottles) were sealed with a foil-lined cap and stored in the dark at room temperature. Sterile control solutions (containing the same concentration of test substance) and positive control solutions (containing linear alkyl benzene sulphonate) were also run. At various times during the study, a bottle was removed and the amount of phosphate ester present was determined (the gas chromatographic method used analysed the sum of major components present in the test substance). The results showed that the test substance underwent primary degradation in the test system, with complete degradation in less than seven days. No significant degradation was seen in the sterile controls.

The second part of the study investigated the primary degradation of the test substance using a SCAS unit (Saeger *et al.* 1979). The method used was based on the Soap and Detergent Association procedure (Soap and Detergent Association 1965 and 1969). The activated sludge used in the test was of domestic origin and the vessels used in the test had an operating volume of 1.5 litres. The test substance was added to the unit at a rate of 3 or 13 mg/l per 24-hour cycle. The units were operated for a period of 7 to 12 weeks and samples of the mixed liquor were removed at weekly intervals and the concentration of the phosphate ester determined. The results indicated an equilibrium removal rate of 96 ± 2 per cent at 3 mg/l and 93 ± 11 per cent at 13 mg/l in the test system. To investigate the loss by volatilisation, the off-gases were passed through a series of scrubbers. No significant loss by volatilisation (below 0.5 per cent per cycle) of the phosphate ester was seen in the experiment.

The final part of the study investigated the ultimate mineralisation of the test substance using a degradation method based on the modified Sturm method. An acclimated bacterial seed was prepared by incubation of 100 ml of settled supernatant from a SCAS unit with 20 mg of one of eleven phosphate esters (including the test substance), 50 mg of yeast extract and 900 ml of standard biological oxygen demand (BOD) water for 14 days in the dark at room temperature. At the end of the incubation period, a combined acclimated seed was prepared by mixing samples from each acclimation bottle and this was used as seed for the inherent biodegradation test. In the test, 500 ml of the composite seed was added to 5,500 ml of BOD water and the substance was then added to the bottle (initial concentration 18.3 mg/l). During the test, CO_2-free air was continually bubbled through each bottle and the CO_2 evolved from the system was determined. Control bottles (receiving no test substance) were also run. The amount of CO_2 evolved from the control bottles was around 10-15 per cent of that of the bottles containing the test substance and the results were corrected for this background CO_2 level. The CO_2 evolved from the test substance (expressed as a percentage of the maximum theoretical amount) was 62 per cent after seven days and 82 per cent after 28 days. Therefore, the substance can be considered as inherently biodegradable based on the results of this test.

A similar series of experiments was reported by Carson *et al.* (1990) using a mixture of triphenyl phosphate and butylphenyl diphenyl phosphate. The study found 84 to 93 per cent removal using the SCAS system (concentration tested 3 or 13 mg/l), a half-life of under one day for primary degradation in the river water (concentration tested 20 mg/l), and a ultimate degradation of 82 to 90 per cent based on CO_2 evolution using acclimated bacterial seed (concentration tested 20 mg/l; bacterial seed 500 ml in a total volume of 6 litres of BOD water). The results reported were very similar to those given by Saeger *et al.* (1979) for triphenyl phosphate alone.

Carson *et al.* (1990) also reported the results for the biodegradation of a mixture of triphenyl phosphate and tributylphenyl diphenyl phosphate (no information on purity) in microcosm and outdoor simulation tests. The microcosm tests were carried out using lake water and sediment collected from the littoral region of a spring-fed freshwater lake in 5- or 10-gallon aquaria. The sediment was screened (1.3 cm) and placed to a depth of 8 cm in the aquaria. Twenty-two litres of lake water were then added to the aquaria and the system was allowed to stabilise for six weeks. After this time, core chambers were created within the aquaria by inserting glass cylinders through the water column and sediment and the test substance was added to each chamber at a concentration of 75 or 675 μg/l. Sterile control chambers were created by adding formaldehyde to the chamber. The half-life for primary degradation in the system was found to be 1 to 3 days in the system.

The outdoor simulation test was carried out using the same mixture of triphenyl phosphate and tributylphenyl diphenyl phosphate (Carson *et al.* 1990). The tests were carried out in tanks (10 feet in diameter and 2 feet deep) containing a 10-15 cm depth of Missouri River flood plain soil and 3,000 litres of well water. The tanks were constantly aerated and allowed to stabilise over a three- to four-month period. After this period, core chambers were created within the tanks as before (each core chamber contained a water volume of 9.5 litres) and the test substance was added to the chambers at concentrations of 25, 75 or 675 μg/l. Sterile control chambers were also created by adding formaldehyde to the chamber. The experiment was run for 35 days. The half-life for primary degradation in this test system was determined to be 2 to 3 days.

Hattori *et al.* (1981) reported that triphenyl phosphate was degraded in samples of water from two rivers. The concentration of triphenyl phosphate used was 0.5, 1 or 20 mg/l and the degradation was followed for 14 days. At 20 mg/l, no degradation of triphenyl phosphate was seen over the first two to three days of the experiment but by day 14 both orthophosphoric acid and phenol were found to be present as degradation products. The rate of degradation of triphenyl phosphate was found to be higher at the lower concentrations tested, with almost 100 per cent primary degradation observed within five to seven days in the two river waters (degradation was slightly faster in the more polluted river water). Sterile controls showed no degradation of triphenyl phosphate over the duration of the study. A similar experiment was carried out using seawater from two sites in Osaka Bay (Tomogashima and Senbuku). In the tests using seawater from Tomogashima, no increase in the concentration of orthophosphoric acid was observed during the first seven days of the study, and only a slight increase in its concentration by the end of the study (the amount of orthophosphoric acid present was equivalent to around 9 per cent degradation at day seven and 35 per cent degradation by day 14). In contrast, experiments with seawater from Senbuku showed 100 per cent degradation within seven days. The difference in rate of degradation between the two sites was explained by the fact that seawater from Senbuku contained much higher levels of microorganisms and nutrients than water from Tomogashima, as industrial waste waters were known to discharge into the Senbuku area.

Anderson *et al.* (1993) studied the biodegradation of triphenyl phosphate in soil under aerobic and anaerobic conditions. The substance tested was a ^{14}C-labelled triphenyl phosphate (radiochemical purity 98.4 per cent; mixed with non-labelled triphenyl phosphate (purity 99.46 per cent)). The soil used in the test was recommended for use in

degradation studies for registration of plant protection products (BBA standard soil 2.2) and was a loamy sand soil with an organic carbon content of 2.22 per cent. The soil was sieved (2 mm) prior to use and had a microbial biomass of 373 mg microbial carbon/kg dry weight at the start of the experiment and 262 mg microbial carbon/kg dry weight after 101 days. The test substance was added to a portion of the dried soil as a solution in acetonitrile. The solvent was evaporated and the spiked dry soil was then mixed into the test soil to give a final triphenyl phosphate concentration of 5 mg/kg dry weight. For the test, 100 g portions of the treated soil were placed in 300 ml flasks and the moisture was adjusted to 40 per cent of the maximum water holding capacity (the water content was checked and, if necessary, adjusted every two to four weeks during the experiment). The flasks were incubated in the dark at 20°C for up to 101 days. Carbon dioxide and other volatiles emitted from the flask were collected and analysed and two soil samples were extracted (Soxhlet extracted for 16 hours with methanol: water (9:1)) and analysed for the presence of radioactivity on days 0, 13, 32, 60 and 101. Similar incubations using heat-sterilized soils were carried out as controls. The results of the experiment are shown in Table 3.1.

Table 3.1 Biodegradation of ^{14}C-triphenyl phosphate in aerobic soil

Incubation time	Percentage of applied radioactivity				Degradation products identified (% of applied amount)			
	Soil-extract-able	Soil-bound residue	Carbon dioxide	Total recovery	Triphenyl phosphate[a]	Diphenyl phosphate[a]	Unknown products[a]	CO_2
0 days	95.3±1.3	4.2±1.1	<0.1	99.5±0.2	92.9-95.3	0.8		<0.1
13 days	71.1±0.3	14.8±0	11.6±1.5	97.4±1.8	62.1-69.3	0.6	2.9-3.5	11.6
32 days	51.9±0.1	23.0±0.1	24.8±0.1	99.7±0.3	41.0-46.6	0.6	4.7-5.5	24.8
60 days	35.4±0.6	25.6±0.2	34.2±0.9	96.2±1.3	25.1-30.4	0.3	5.4-6.0	34.2
101 days	26.6±0.6	26.4±0.7	48.3	101.3	15.6-20.2	0.2	6.4-7.1	48.3
Heat sterilized control soil								
13 days	103.4±1.8	2.2±0.3	<0.1	105.6±1.5	92.9-103.4	7.1		<0.1
101 days	85.4±3.9	3.0±0.3	<0.1	88.4±4.2	82.5-85.4	1.4		<0.1

Source: Anderson *et al.* (1993).
Notes: a) Products identified by both HPLC and TLC methods.

As can be seen from the data in Table 3.1, the mass balance and reproducibility in this test is excellent. The results show that triphenyl phosphate was degraded in the system with around 41 to 47 per cent of the initial amount remaining after 32 days. Traces of diphenyl phosphate were also found in the experiment, along with an unidentified product. This product was more polar than diphenyl phosphate and was shown not to be phenol or monophenyl phosphate. Mass spectroscopic analysis of the isolated, silylated product indicated it to be a saccharide with a molecular weight of 918. It was thought that this product resulted from the re-assimilation of $^{14}CO_2$ by microbial processes in the soil. Based on the carbon dioxide formed in the experiment, a mineralisation half-life of around 100 days at 20°C can be inferred from the data.

The experiments under anaerobic conditions were carried out in a similar way with an initial triphenyl phosphate concentration of 5 mg/kg dry weight, except that the incubations were carried out in desiccators under nitrogen atmosphere (Anderson *et al.* 1993). The desiccators contained an oxygen adsorption unit, along with an oxygen test strip, to ensure anaerobic conditions were maintained. The results of the experiment are shown in Table 3.2.

Again, rapid degradation of the triphenyl phosphate was seen with around 50 per cent of the initial amount remaining after 32 days incubation. A small amount of volatile products (other than carbon dioxide) formed during the study, though their identity was not determined. Phenol was the main intermediate product formed in the soil (this also

degraded) but small amounts of other, unidentified intermediate products may also have been present. Based on these data, a half-life for ultimate mineralisation of the order of 100-200 days can be estimated. The mineralisation rate of triphenyl phosphate in soil under anaerobic conditions is similar to that under aerobic conditions.

The effect of temperature and redox potential degradation of several phosphate esters, including triphenyl phosphate, in two natural sediments was investigated by Muir *et al.* (1989). The results of the experiments are summarised in Table 3.3.

The triphenyl phosphate tested was ^{14}C-labelled mixed with a purified non-labelled triphenyl phosphate. The sediment samples used in the study were collected from a eutrophic farm pond and the Red River, Winnipeg (both samples were from farm areas remote from industry). The pond sediment consisted of 75 per cent clay, 24 per cent silt and one per cent sand and had an organic carbon content of nearly 4 per cent and a pH of 7.6; the river sediment consisted of 48 per cent clay, seven per cent sand and 43 per cent silt with an organic carbon content of 2 per cent and a pH of 7.7.

The aerobic sediment experiments were carried out using loosely capped flasks (static test) or in respirometer flasks with air flowing through the system (1-2 ml/minute). The sediments incubated under anaerobic conditions (in respirometer flask under a nitrogen flow (1-2 ml/minute)) were amended with one per cent by weight of microcrystalline cellulose to provide an additional source of carbon. The degradation experiments were carried out using around 10 g (dry weight) of sediment in dechlorinated water (sediment:water ratios of 1:10 (static test) or 1:20 (respirometer flask)). Each sediment sample was pre-incubated for 21 days at the intended experimental temperature prior to the addition of the test substance. The concentration tested was 0.1 mg/l (static test) or 0.05 mg/l (respirometer flasks) and the substance was added as 0.1 ml of a solution in acetone. All experiments were carried out in duplicate for up to 64 days and sterile controls were also run to investigate the abiotic degradation of triphenyl phosphate under the conditions used.

Table 3.2 Biodegradation of ^{14}C-triphenyl phosphate in anaerobic soil

Incubation time	Percentage of applied radioactivity					Degradation products identified (% of applied amount)			
	Soil-extractable	Soil-bound residues	Carbon dioxide	Other volatiles	Total recovery	Triphenyl phosphate[a]	Diphenyl phosphate[a]	Phenol[a]	Carbon dioxide
0 days	112±0.2	1.4±0.1			113.6±0.2	112.1	1.2		
32 days	66.5±1.2	16.8±1.4	12.4	2.6	98.3±0.2	50.2	0.8	15.5	12.4
68 days	45.5±2.1	20.2±2.0	22.9	6.3	94.9±1.6	35.4	0.3	8.2	22.9
102 days	35.8±2.2	24.0±2.0	40.4	1.6	101.8±4.1	31.4	0.5	1.6	40.4

Source: Anderson *et al.* (1993).
Notes: a) Products identified by HPLC and/or TLC methods.

Table 3.3 Effect of temperature and redox potential on degradation of ^{14}C-triphenyl phosphate in sediments

Test system	Sediment	Temp.	Time (days)	Amount of ^{14}C present (% of applied)							Estimated half-life (days)[c]
				Sediment-extractable[a]	Sediment-non-extractable	Water-extractable[b]	Water- non-extractable	CO_2	Total	Percentage of total as parent compound	
Aerobic static test system	Pond	25°C	0.25	102.0	2.6	13.9	Na		116.5		2.8±1.0
			64	3.5	12.4	<0.1	3.0		18.9		
		10°C	0.25	14.1	22.6	2.5	34.4		73.6		2.8±0.7
			64	3.9	14.4	0.4	1.0		19.7		
		2°C	0.25	98.5	3.3	5.6	Na		107.3		11.9±4.9
			6	49.7	3.1	15.2	Na		68.0		
	River	25°C	0.25	84.0	1.6	10.0	109.3				7.0±2.8
			40	26.2	21.0	0.2	17.8		65.3		
Aerobic respirometer	River	25°C	3	85.9	8.8	35.2	Na	0.2	130.1	56.7	
			40	20.5	31.9	9.7	Na	17.4	79.5	13.1	
Anaerobic respirometer	River	25°C	3	68.9	8.2	40.4		0.8	118.2	68.9	
			40	19.5	20.1	25.7		21.9	87.1	10.3	

Source: Muir *et al.* (1989).

Notes: a) Extracted with aqueous methanol to recover undegraded phosphate ester and any diaryl phosphate degradation products.
b) Extracted with dichloromethane to recover undegraded phosphate ester.
c) Half-life estimated based on the data obtained over days zero to six for pond sediment and days zero to ten for river sediment. The half-life refers to the disappearance of the parent compound from the sediment phase.

The aerobic experiments were incubated with a 16:8 hours light:dark photoperiod (using low intensity light) whereas the anaerobic experiments were incubated in darkness. The microbial biomass present in the test systems was between 9×10^6 to 32×10^6 colony forming units (CFU) per gram in the experiments with river sediments. The microbial biomass present in the aerobic pond respirometer sediments was found to decline from 42×10^6 CFU/g to 0.3 CFU/g over the 64-day period. The total microbial biomass (aerobic and facultative anaerobic heterotrophs) present in the N_2-purged respirometer experiments was 5.3×10^6 CFU/g after 3 to 8 days and 24×10^6 after 30 to 40 days, but the number of strict anaerobes present was around eight to forty times less, and so the incubations were not strictly anaerobic.

The results show that extensive degradation of triphenyl phosphate occurred in the study. Initially, most of the triphenyl phosphate added to the system adsorbed onto the sediment phase but by the end of the experiment the amount of extractable radioactivity associated with the sediment phase had decreased substantially. Detailed analysis of the sediment extracts indicated that the major portion of the extractable radioactivity was as unchanged triphenyl phosphate, with low levels of degradation products, including diphenyl phosphate, also present.

Muir and Grift (1983) investigated the degradation of ^{14}C-labelled triphenyl phosphate in river sediment incubated under aerobic and anaerobic (nitrogen atmosphere) conditions for up to 64 days. The tests were carried out using 50 g (wet weight) of sediment in 250 ml of dechlorinated water. Few other experimental details of this test are available. The results are summarised in Table 3.4. The reaction was followed by determining the amount of ^{14}C that could be extracted from the sediment (by refluxing with aqueous methanol) and the amount that could not be extracted. The extractable ^{14}C was found to be mainly in the form of unchanged triphenyl phosphate and diphenyl phosphate. The identity of the radioactivity in the unextracable fraction was not determined. Based on these results, around 92 per cent primary degradation of the triphenyl phosphate occurred in 64 days under aerobic conditions and around 83 per cent primary degradation occurred in 64 days under anaerobic conditions.

Table 3.4 Degradation of ^{14}C-labelled triphenyl phosphate in aerobic and anaerobic river sediments

Sediment type	Incubation time	Extractable radiolabel (%)			Unextractable radiolabel (%)
		As triphenyl phosphate	As metabolites	Total	
Aerobic	32 days	20.8	9.1	29.9	70.1
	64 days	8.1	9.5	17.6	82.4
Anaerobic	32 days	48.7	11.1	59.8	40.2
	64 days	17.4	5.3	22.7	77.3

Source: Muir and Grift (1983).
Notes: a) The main metabolite found was diphenyl phosphate.

Pickard *et al.* (1975) studied the biodegradation of a commercial triaryl phosphate (Fyrquel 220), along with triphenyl phosphate, trixylenyl phosphate and tri-*ortho*-cresyl phosphate by mixed bacterial populations. The actual composition of the commercial product was not given, but it was reported that the following mixture of phenolic compounds was used to manufacture the product: 2.6 per cent phenol, 0.5 per cent *ortho*-cresol, 13.6 per cent *meta*- and *para*-cresol, 0.6 per cent 2-ethylphenol, 22.3 per cent 2,4- and 2,5-xylenol, 49.2 per cent mixed xylenols, 8.6 per cent 3,4-xylenol, 1.3 per cent C_6-C_9 phenolics and 1.4 per cent trimethyl phenol. Based on this starting mixture, the commercial product would be a mixture of trixylenyl phosphate and tricresyl phosphates,

along with other aryl phosphates. The purity of the triphenyl phosphate, trixylenyl phosphate and tri-*ortho*-cresyl phosphate used was not reported.

The microbial cultures used in the study were obtained by enrichment (using 0.1 per cent solutions of Fyrquel 220) of a mud sample from a lake that had been used as a seaplane base and that was rich in oil-utilizing microorganisms. The enriched mixed microbial cultures were capable of growing on Fyrquel 220, tri-*ortho*-cresyl phosphate and trixylenyl phosphate as sole source of carbon, but grew only poorly when triphenyl phosphate was used as the sole carbon source.

Boethling and Cooper (1985) estimated that the removal of triphenyl phosphate during biological waste water treatment at a production plant in the United States was 99 per cent based on the average concentration in waste water (0.74 mg/l) and the average concentration in effluent from the treatment plant (0.007 mg/l). The removal was thought to be due to biodegradation, since air stripping was not thought to be an important removal mechanism, and sludge wastage was not practiced at the facility. However, it was also indicated that the results should be treated with caution, as the recoveries found for the effluent samples were generally much lower than found for the waste water samples (27 per cent overall versus 89 per cent overall). Thus, concentrations in the effluent may have been higher than indicated (and hence the removal lower than indicated).

Summary of degradation

Abiotic degradation

The available information indicates that triphenyl phosphate undergoes hydrolysis to form diphenyl phosphate which is more stable to hydrolysis than the parent compound. The rate of hydrolysis has been determined in several studies, and is found to increase with pH. The half-lives are generally under 3 days at pH 9 and above, 7 to 24 days at pH 8 and 19 days or longer at pH 7. There is also evidence that aryl phosphates will undergo hydrolysis under acidic conditions. Experiments were mostly carried out at room temperature (typically 20-30°C) and so the rate of hydrolysis in the environment may be slower than found in the experiments, due to typically lower temperatures. Nevertheless, the laboratory studies generally show rapid hydrolysis at very high pHs (typically pH 9 or greater) and rapid hydrolysis may also occur at low pH. Since the pH in the environment is usually outside these extremes, and since other biotic removal mechanisms are likely to be much more important than hydrolysis for triphenyl phosphate at these pHs, the rate of hydrolysis of triphenyl phosphate is assumed to be zero in this assessment. However, in some acidic or alkaline environments, hydrolysis could become significant and so the effect of inclusion of a more rapid hydrolysis rate (half-life 24 days) on the overall conclusions of the risk assessment is considered in Annex C. At least one study indicated that the initial product of hydrolysis, the diphenyl phosphate, was not susceptible to further hydrolysis.

Boethling and Cooper (1985) suggested that the hydrolysis rates of triaryl phosphates containing alkyl substituents on the aromatic ring should be lower than those for triphenyl phosphate, due to the electron-donating character of these groups.

Several studies have shown that triphenyl phosphate can undergo photolytic degradation using UV radiation. However, as the amount of UV radiation reaching the earth's surface is small, the significance of these studies to the behaviour in the environment is limited. For the purposes of the assessment, the rate of photolysis of triphenyl phosphate is assumed to be zero.

Atmospheric photooxidation of triphenyl phosphate is predicted to occur with a half-life of around 36 hours. This reaction is taken into account in this risk assessment.

In summary, the abiotic degradation rate constants and half-lives assumed in the assessment are as follows, and the importance of hydrolysis to the overall conclusions of the risk assessment is considered further in Annex C.

Hydrolysis	$khydr_{water}$ = 0 d^{-1}	half-life = infinite
Photolysis	$kphoto_{water}$ = 0 d^{-1}	half-life = infinite
Atmospheric photooxidation	k_{OH} = 1.08×10^{-11} cm^3/molecule s	half-life = 36 h

Biodegradation

The most likely pathway for biodegradation of aryl phosphates is the initial hydrolysis of the phosphate ester to form orthophosphate and corresponding phenolic compounds or alcohols, which themselves undergo further biodegradation (Saeger *et al.* 1979).

Many studies show that triphenyl phosphate is degraded rapidly in a range of test systems. In standard tests, triphenyl phosphate can be considered to be readily biodegradable, but it is not clear if the 10-day window is met.

The recommended biodegradation half-lives for surface water and soil from the TGD for triphenyl phosphate (assuming it is readily biodegradable but not meeting the 10-day window or readily biodegradable meeting the 10-day window) with a Kp_{soil} of 200 l/kg (see Section 3.1.2) are summarised below:

	Not meeting 10-day window	Meeting 10-day window
Surface water	half-life = 50 days	half-life = 15 days
Soil	half-life = 900 days	half-life = 300 days

The available screening studies with which to compare these data are limited, in that most of them have measured primary degradation rather than mineralisation. However, the available river die-away studies generally show rapid primary degradation (half-life less than seven days), and primary degradation half-lives of one to three days have been measured in sediment-water systems. Given that the intermediate product of primary degradation is likely to be phenol, which itself is likely to undergo rapid mineralisation, it can be concluded that the default half-life of 15 days for surface water is more appropriate for triphenyl phosphate than the longer default half-life of 50 days. Furthermore, some studies for soil have measured a mineralisation half-life of around 100 days for triphenyl phosphate at 20°C. This is again in line (allowing for the lower average ambient temperature found in the environment) with the lower default half-life of 300 days given above. Overall, the default mineralisation half-lives from the TGD assuming the ten day window is met appear to be most appropriate for triphenyl phosphate. These values are therefore used in the risk assessment.

For sediment, the TGD recommends that the default rate constant should be ten times lower than that for soil to reflect the fact that the deeper sediment layers are anaerobic (this calculation assumes that degradation under anaerobic conditions does not occur). However, the available information for triphenyl phosphate suggests that biodegradation occurs at a similar rate under anaerobic and aerobic conditions. Therefore, for this assessment, it is assumed that the degradation rate constant (and hence half-life) in sediment is the same as in soil.

In summary, the following biodegradation rate constants half-lives are used here:

Sewage treatment plant	$k = 1\ h^{-1}$	half-life = 0.7 hours
Surface water	$k = 4.7\times10^{-2}\ d^{-1}$	half-life = 15 days
Sediment	$k = 2.3\times10^{-3}\ d^{-1}$	half-life = 300 days
Soil	$k = 2.3\times10^{-3}\ d^{-1}$	half-life = 300 days

Although the phenolic part of the triaryl phosphate will undergo mineralisation, orthophosphate/phosphoric acid will also be produced as a result of the degradation. The fate, behaviour and effects of this substance are beyond the scope of this assessment.

3.1.2 Environmental partitioning

Adsorption

Anderson *et al.* (1993) investigated the adsorption of a ^{14}C-labelled triphenyl phosphate (radiochemical purity 98.4 per cent, mixed with non-labelled triphenyl phosphate (purity 99.46 per cent)) to three soils. The soils used were classed as a loamy sand (organic carbon content 2.2 per cent), a silty clay (organic carbon content 0.6 per cent) and a silt loam (organic carbon content 2.6 per cent). Four initial exposure concentrations, corresponding to five per cent, 25 per cent, 37 per cent and 50 per cent of the water solubility, were carried out for each soil using 0.01 M calcium chloride solutions and the equilibration was carried out at 20°C using a batch process. Few other details of the tests method were reported but they were carried out in accordance with a USEPA method (USEPA 1987). Equilibrium was reached after 48 hours and similar K_{oc} values were determined for all three soils in the range 2,514 to 3,561 l/kg. In some soils, partial degradation of the triphenyl phosphate was seen (probably as a result of hydrolysis) resulting in the formation of a small amount of diphenyl phosphate (maximum degradation was seen in the silty clay) and so the K_{oc} values determined are a function of the adsorption properties of both triphenyl phosphate and the small amount of degradation product.

Huckins *et al.* (1991) investigated the adsorption of triphenyl phosphate (purity 99 per cent) onto top soil (wind-blown loess, five per cent sand, 77 per cent silt, 18 per cent clay and 1.12 per cent organic carbon content). The experiments were carried out by adding 100 μg of the test substance as a solution in acetone to 250 ml bottles and evaporating the acetone with a stream of air. Air-dried top soil (100 mg) and 100 ml deionised water (pH 6.5) were then added to the bottles, which were capped and shaken for 24 hours at 25°C. After 24 hours, the water and solid phases were separated by filtration and the concentrations of triphenyl phosphate in the solid and water phases were determined. In addition, some experiments were carried out using centrifugation rather than filtration to separate the aqueous and solid phases. These were carried out in a similar way as described above, except that the initial amount of triphenyl phosphate added to the bottles was 3.0 or 30.2 μg. The Kp values determined were 112 ± 26.8 l/kg for the filtration experiments and 96 ± 41 l/kg for the centrifuge experiments. As the soil organic carbon content was 1.12 per cent, these values are equivalent to K_{oc} values of 8,570 to 10,000 l/kg.

Similar experiments with montmorillonite clay (organic carbon content 0.3 per cent) gave variable results but the mean Kp value determined was around three times higher than the value obtained with soil (Huckins *et al.* 1991). This suggested that adsorption of triphenyl phosphate to active sites on clay minerals may also be important.

Boethling and Cooper (1985) report a K_{oc} value of 3,100 l/kg for triphenyl phosphate. The value was estimated from a water solubility of 1.9 mg/l. The same paper also reported a

result from a study by Kenmotsu *et al.* (1980) which determined a sediment-water partition coefficient of 60 l/kg for triphenyl phosphate with marine sediment.

A K_{oc} value of 5,237 l/kg can be estimated for triphenyl phosphate from its structure using the Syracuse Research Corporation PCKOC version 1.63 software with a molecular connectivity index method.

Chapter 4 of the TGD recommends the following equation for estimating K_{oc} from log K_{ow} for phosphates:

$$\log K_{oc} = 0.49 \log K_{ow} + 1.17$$

Using this equation for triphenyl phosphate (log K_{ow} of 4.63) results in an estimated K_{oc} value of 2,746 l/kg. This value is in good agreement with those measured by Anderson *et al.* (1993) but is lower than those estimated from the data reported by Huckins *et al.* (1991).

As measured data are available, the K_{oc} value used in the assessment is based on these data rather than predicted values (although predicted values generally support the measured data, to within a factor of two or three). As noted above, the data generated by Anderson *et al.* (1993) may have been influenced by any ^{14}C-labelled degradation products present in the system and so a K_{oc} value of 10,000 l/kg (the upper end of the range from the Huckins *et al.* (1991) study is assumed for the risk assessment. The resulting partition coefficients for soils and sediment calculated using the methods given in the TGD are shown below.

K_{oc}	10,000 l/kg
Kp_{susp}	1,000 l/kg
$K_{susp\text{-}water}$	251 m^3/m^3
Kp_{sed}	500 l/kg
$K_{sed\text{-}water}$	251 m^3/m^3
Kp_{soil}	200 l/kg
$K_{soil\text{-}water}$	300 m^3/m^3

These values are used in the risk assessment, although there are uncertainties in the available data. If sorption to clay is important, as indicated by one of the studies, then basing the Kp values on the organic carbon content may not be the most appropriate approach[5].

Volatilisation

No studies on the volatilisation of triphenyl phosphate appear to be available. The Henry's law constant estimated for triphenyl phosphate is 0.41 Pa m^3/mol at 25°C. This indicates that volatilisation from water is likely to be limited.

Fugacity modelling

The potential environmental distribution of triphenyl phosphate has been studied using a generic level III fugacity model. The model used was a four-compartment model (EQC version 1.01, May 1997) that has been circulated for use within the OECD HPV programme. The model was run four times with a nominal release rate of 1,000 kg/hour initially entering the air, soil or water compartments in different proportions. The physico-chemical properties used and results of the exercise are shown in Table 3.5.

[5] For further discusson on sorption, see Annex D.

The results of the model show that only a small amount of the triphenyl phosphate released to the environment will be in the air compartment at steady state. When the substance is released to air it distributes mainly to the soil compartment, presumably by atmospheric deposition. When it is released to soil, the substance generally remains in the soil, with only a small fraction distributing to the water and sediment compartment. When released to water, the substance is likely to distribute equally to the water and sediment phase at steady state.

Table 3.5 Results of generic level III fugacity model for triphenyl phosphate

Input data	**Value**
Vapour pressure	1.2×10^{-3} Pa at 20°C
Water solubility	1.9 mg/l
Henry's law constant	0.21 Pa m^3/mol at 20°C
Log K_{ow}	4.63
Atmospheric half-life	36 hours
Half-life in water	15 days
Half-life in soil and sediment	300 days

Emission rate	**Model results at steady state**				
	Amount in air	**Amount in soil**	**Amount in water**	**Amount in sediment**	**Overall residence time/ persistence**
1,000 kg/hour to air 1,000 kg/hour to soil 1,000 kg/hour to water	0.26%	94.2%	2.9%	2.7%	163 days
1,000 kg/hour to air 0 kg/hour to soil 0 kg/hour to water	3.8%	94.5%	0.92%	0.86%	34.2 days
0 kg/hour to air 1,000 kg/hour to soil 0 kg/hour to water	4.3×10^{-4}%	100%	0.023%	0.021%	429 days
0 kg/hour to air 0 kg/hour to soil 1,000 kg/hour to water	0.032%	0.81%	51.3%	47.8%	26.6 days

The behaviour of triphenyl phosphate during waste water treatment was estimated using the EUSES 2.0 model. Using a degradation rate constant of 1 h^{-1} (see Section 3.1.1), a K_{oc} of 10,000 l/kg (see above) and a vapour pressure of 2.4×10^{-3} Pa at 25°C (see Section 1.3.5), the following behaviour is predicted:

Degraded	50.6%
Adsorbed to sludge	41.0%
Volatilised to air	0.09%
To effluent	8.27%

These values are used in the predicted environmental concentration (PEC) calculations.

3.1.3 Bioaccumulation and metabolism

Measured data

Uptake from water

The bioconcentration and metabolism of triphenyl phosphate (purity not given) were studied in goldfish (*Carassius auratus*) and killifish (*Oryzias latipes*) (Sasaki *et al.* 1981). The fish weighed around 0.8-2.8 g (goldfish) or 0.1-0.2 g (killifish) and were acclimated to laboratory conditions for at least ten days prior to the start of the test. A static exposure test system was used. This consisted of 2 litre beakers containing a solution of the triphenyl phosphate (0.25 mg/l) and either 10 to 20 killifish or 3 to 5 goldfish. The solutions were maintained at around 25°C without aeration for up to 96 hours. The fish were not fed during the test and the concentration of triphenyl phosphate was determined in the fish and water at various times during the test. Control experiments (without fish) showed that triphenyl phosphate was stable in the test water over the 96-hour exposure period under the conditions used in this test.

The results for killifish indicated that the concentration of triphenyl phosphate in the water phase decreased rapidly (half-life of around five hours) indicating that the substance was being taken up by the fish. The triphenyl phosphate initially in the exposure vessel was found to have been completely metabolised by the fish within 72 hours and was not detected in the water or fish after this time. The bioconcentration factors measured were 330 l/kg after around eight hours, 500 l/kg after around 24 hours, 480 l/kg after around 48 hours and 250 l/kg after around 72 hours. The uptake of triphenyl phosphate in gold fish was also found to be rapid over the first five hours exposure, but then the uptake slowed (half-life was estimated to be above 100 hours). The bioconcentration factors determined were 150 l/kg after around 24 hours, 130 l/kg after around 48 hours and 110 after around 72 hours. As the exposure concentrations in these studies were not maintained, the reported bioconcentration factor (BCF) values cannot be taken to represent steady-state values.

The accumulation of triphenyl phosphate in killifish (*Oryzias latipes*) was also studied using a flow-through system by Sasaki *et al.* (1982). In the experiment, groups of 70 to 100 fish were exposed to a triphenyl phosphate concentration of 0.009 to 0.01 mg/l at 25°C for up to 18 days at 25°C (a small amount of acetone co-solvent was also present in the system). Concentrations of the test substance were found to be constant throughout the test period. The BCF determined at the end of the 18-day exposure period was 84 l/kg based on parent compound analysis. However, the uptake curve given in the paper indicates that the concentration in fish was still increasing at this point and so the value cannot be considered a steady-state value. At the end of the exposure period, the fish were placed in clean water and a depuration half-life for triphenyl phosphate of 1.2 hours was determined. Experiments carried out under static conditions gave a BCF of 157-390 l/kg. A further test was carried out exposing the fish to a mixture of around 0.1 mg/l tributyl phosphate, around 0.09 mg/l of tris(1,3-dichloroisopropyl) phosphate and around 0.02-0.03 mg/l of triphenyl phosphate for 32 to 35 days. The BCF determined for triphenyl phosphate over this longer exposure period was 189-193 l/kg. Again, the uptake curve given in the paper indicates that steady state may not have been reached in this study for triphenyl phosphate.

Muir *et al.* (1980) investigated the uptake, accumulation and tissue distribution of ^{14}C-labelled triphenyl phosphate by rainbow trout (*Oncorhynchus mykiss*). The substance used had a radiochemical purity of 99 per cent and was diluted with unlabelled triphenyl phosphate (99.5 per cent pure).

In the uptake and accumulation part of the test 20 fingerling rainbow trout (0.2-0.6 g) were placed in four litres of test water (river water, dechlorinated tap water or dilutions (1:3 and 2:3) of river water with dechlorinated tap water) at 10°C and acclimated for 16 hours. Following the acclimation period, the test substance was added to the water to give a concentration of 50 μg/l (two replicates were used for each water type) and the test solutions were continuously stirred throughout the exposure period. At various times during the test (30 minutess, 1, 2, 4, 12 and 24 hours) three fish were removed from each replicate and analysed for the presence of radiolabel. In addition, the stability of the test substance in river water over the 24-hour exposure period was determined by parent compound analysis (gas chromatography with a nitrogen-phosphorus detector). Studies to investigate the tissue distribution of the radiolabel were carried out in a similar way except that the fish used were 100-200 g and these were exposed to 50 μg/l of the test substance for 24 hours in 20 litres of dechlorinated tap water. Finally, depuration of radiolabel from rainbow trout was studied by exposing fry (0.1-0.3 g) to a test substance concentration of 10 μg/l in four litres of dechlorinated tap water for six hours and then transferring the fish to a 40-litre tank with a constant flow of dechlorinated tap water. Samples of fish (five per time point) were analysed at various times during the depuration period (24, 48, 144, 216, 384 and 744 hours).

The test substance was found to be stable in river water over the 24-hour exposure period in the study. The rate of uptake by fingerling rainbow trout was found to be different in river water compared with tap water. Initial rates of uptake were found to be 1.89 μg/g hour in river water, 2.6 μg/g hour in the 2:1 dilution of river water, 2.18 μg/g hour in the 1:2 dilution of river water and 2.32 μg/g hour in dechlorinated tap water. It was thought that some, but not all, of the difference between the initial rates of uptake could be explained by adsorption of the test substance onto suspended matter (the concentration of suspended matter was highest in river water and lowest in dechlorinated tap water) and that other water quality parameters (such as dissolved organic matter) might also be important in determining the bioavailability of the test substance. The tissue distribution studies indicated that the highest rate of uptake of radiolabel was associated with the liver (initial uptake rate 2.75 μg/g hour), followed by kidney (2.01 μg/g hour), caeca (0.62 μg/g hour), intestine (0.53 μg/g hour), blood (0.56 μg/g hour) and muscle (0.45 μg/g hour). The depuration part of the experiment showed that the radiolabel was eliminated from the fish at a rapid rate until about day nine (when only 1 to 2 per cent of the radiolabel remained in the fish), where the rate of depuration decreased markedly. Based on the initial rate of uptake in dechlorinated tap water (rate constant 46.36 $hour^{-1}$) and the rate of depuration (rate constant 0.0179 $hour^{-1}$ up to day nine or 0.00245 $hour^{-1}$ from day nine onwards) a bioconcentration factor of 2,590 l/kg or 18,900 l/kg can be determined. However, these values are based on ^{14}C-measurements and so include contributions from any metabolites formed (the paper indicates that gas chromatographic analysis of whole fish exposed for 24 hours showed that less than 50 per cent of the radiolabel present was triphenyl phosphate). Further, the uptake and depuration rate constants appear to have been obtained with different fish populations (uptake rate constants were obtained with fingerling rainbow trout (0.2-0.6g) whereas depuration rate constants were obtained with rainbow trout fry (0.1-0.3 g)) using different exposure concentrations, which adds some uncertainty to the bioconcentration factors obtained in this study.

Muir *et al.* (1983a) investigated the bioconcentration of ^{14}C-labelled triphenyl phosphate (radiochemical purity above 98 per cent, mixed with unlabelled triphenyl phosphate) by both rainbow trout (*Oncorhynchus mykiss*) and fathead minnows (*Pimephales promelas*). The fish (mean weight 0.75 g for rainbow trout (loading density 3.0 g/l) and 2.5 g for fathead minnows (loading density 7.2 g/l)) were exposed in glass aquaria containing 10 litres of dechlorinated, carbon-filtered tap water at 10°C. The fish were acclimated to the aquaria for 16 hours prior to the addition of the test substance at either 5 μg/l or 50 μg/l (the test substance was added as a solution in acetone (0.05-0.4 ml added to each aquaria)). Fish (three at each sampling point) were removed from the aquaria at various

times (1, 3, 6, 12 and 24 hours) during the exposure period for analysis of the concentration of radiolabel. At the end of the 24-hour exposure period, the remaining fish were removed and placed in a 40-litre tank with a continuous flow of water and depurated for up to 432 hours (fish were sampled after 12, 24, 48, 96, 144, 240 and 432 hours of depuration). In addition, the concentration of radiolabel in the water phase was determined during the exposure part of the experiment. In some cases, concentrations of the test substance were also determined by parent compound analysis (gas chromatography using an nitrogen-phosphorus detector).

During the exposure part of the test, the concentration of the test substance in water decreased with time as a result of uptake by the fish. This decrease was more marked for fathead minnows than for rainbow trout due to the higher loading used for fathead minnow. For trout, steady-state concentration was not reached within the 24-hour exposure period but the concentrations in fathead minnows generally reached a maximum steady-state value within 12 hours of exposure. Depuration of the test substance was found to be biphasic, especially with rainbow trout, with a more rapid depuration seen over the first six days on transfer to clean water. The kinetic data and bioconcentration factors determined from the data are summarised in Table 3.6. The authors also estimated BCF values of 324 ± 99 l/kg and 420 ± 25 l/kg for rainbow trout and fathead minnows respectively based on the estimated amount of untransformed triphenyl phosphate present in the fish.

Table 3.6 Uptake and elimination of ^{14}C-labelled triphenyl phosphate in rainbow trout and fathead minnow

Species	Exposure concentration (μg/l)		Uptake rate constant (initial rate method) (hour^{-1})[a]	Depuration rate constant (hour^{-1})[a]		Bioconcentration factor		
	0 hours	24 hours		0-144 hour	0-432 hour			
Rainbow trout	5.6	3.1	22.7±13.8	0.0174 ±0.0074	0.0116 ±0.0041	1,368±329[b]	573±97[c]	931±122[d]
	50.4	23.8	20.7±12.7	0.0163 ±0.0071	0.0144 ±0.0050			
Fathead minnow	3.6	0.8	16.5±10.3	0.0121 ±0.0068	0.0076 ±0.0029	1,743±282[b]	561±115[c]	218±55[d]
	34.9	12.5	14.5±8.5	0.0140 ±0.0054	0.0107 ±0.0037			

Source: Muir *et al.* (1983a).

Notes:
a) Uptake and depuration data calculated based on ^{14}C measurements.
b) Bioconcentration factor determined using initial rate method for uptake rate constant.
c) Bioconcentration factor determined using the method of Zitko (1980) to take account of the decrease in exposure concentration with time.
d) Bioconcentration factor determined using the BIOFAC computer program. The values calculated by this method are not considered reliable as the method requires the exposure concentration to remain constant during the test.

A further study of the bioconcentration of triphenyl phosphate in fathead minnow (*Pimephales promelas*) was carried out by Muir *et al.* (1982). In this study, fish were exposed to an initial ^{14}C-labelled triphenyl phosphate concentration of 60 μg/l in a small, shallow, artificial pond (2.5 × 4 m^2 and 0.5 m deep). The pond water had a pH of 8.6 and a total suspended solids concentration of 11 mg/l. The bottom sediments were silty clays and had a pH of 6.8 and six per cent organic matter content. The pond was stocked with 200 fish for two weeks prior to addition of the test substance (in small volume of ethanol). A similar pond acted as control. Fish were sampled at regular intervals up to 15 weeks after addition of the test substance and both the fish and water phases were analysed for the presence of radiolabel. The results for the first ten days are summarised in Table 3.7. The concentration of triphenyl phosphate in the water was found to decrease rapidly with a half-life of under 24 hours (as a result of degradation and/or adsorption onto sediment

and/or uptake into biota). The maximum concentration of triphenyl phosphate in fish was reached after 12 and 24 hours exposure and the BCF was in the range 68-160 l/kg (the highest value was obtained at ten hours post-treatment). The same study also evaluated the concentrations of radiolabel in duckweed (*Lemna minor*) and cattails (*Typha* sp.) present in the pond. The maximum BCF for duckweed was found to be 43 l/kg, and the level of radioactivity in the plant was found to be similar at one hour and ten days after exposure. The BCF for cattails was determined to be less than 1 l/kg ten days after the start of exposure. The fact that the exposure concentration was not maintained during this experiment (the half-life for triphenyl phosphate in the water phase was estimated to be around 15 hours in this study) limits its usefulness in deriving steady-state BCF values.

Table 3.7 Distribution of radioactivity with time in an artificial pond initially exposed to 60 μg/l of ^{14}C-labelled triphenyl phosphate

Time hours	Distribution of radioactivity (as percentage of initial amount added; values in [ ] refer to the actual concentration present)					
	Water	Sediment (0-3 cm depth)	Duckweed	Cattails	Fish	Total
1-4	[63.5 μg/l]					
10	74% [36.8 μg/l]	29% [138 μg/kg dry wt.]	1.4% [2,143 μg/kg wet wt.]	[4 μg/kg wet wt.]	2.7% [8,070 μg/kg wet wt.]	107.1%
24	52% [26.0 μg/l]	34% [162 μg/kg dry wt.]	1.4% [2,031 μg/kg wet wt.]	[5 μg/kg wet wt.]	3.4% [10,250 μg/kg wet wt.]	90.8%
48	34% [16.8 μg/l]	43% [211 μg/kg dry wt.]	1.2% [1,775 μg/kg wet wt.]	[12 μg/kg wet wt.]	1.3% [4,004 μg/kg wet wt.]	79.5%
72	28% [14.4 μg/l]	33% [147 μg/kg dry wt.]	0.9% [1,370 μg/kg wet wt.]	[9 μg/kg wet wt.]	0.9% [2,750 μg/kg wet wt.]	62.8%
120	23% [12.2 μg/l]	40% [181 μg/kg dry wt.]	0.5% [766 μg/kg wet wt.]	[42 μg/kg wet wt.]	0.6% [1,740 μg/kg wet wt.]	64%
240	13% [6.3 μg/l]	36% [149 μg/kg dry wt.]	0.5% [735 μg/kg wet wt.]	[43 μg/kg wet wt.]	0.5% [1,530 μg/kg wet wt.]	50%

Source: Muir *et al.* (1982).

Muir and Grift (1981) investigated the uptake and accumulation of diphenyl phosphate (a possible degradation/hydrolysis product of triphenyl phosphate and other alkyl/aryl diphenyl phosphates) by rainbow trout (*Oncorhynchus mykiss*) fry. The substance used in the test was ^{14}C-labelled. The method was similar to that used previously for triphenyl phosphate (Muir *et al.* 1980, see above). In the uptake part of this study, fry (around 0.1-0.2 g) were exposed to a diphenyl phosphate concentration of 100 μg/l in river (pH 8.1, suspended solids concentration 30 mg/l) or dechlorinated tap water for up to 4.5 hours (previous experiments had shown that the highest concentrations of diphenyl phosphate occurred in fish during the first 30 minutes of exposure). In the depuration part of the study, fish were exposed to 100 μg/l of the test substance in dechlorinated tap water for 30 minutes and then transferred to clean water where the depuration was studied over 90 minutes.

In the uptake part of the experiment, the initial rate of uptake (over the first 20 minutes of the study) was found to be 52 μg/kg hour in dechlorinated tap water and 15 μg/kg hour in river water. Body burdens found in fish during both the uptake and depuration parts of the experiment are shown in Table 3.8. The concentration of radiolabel increased during the first 20-30 minutes of exposure and then decreased, indicating that diphenyl phosphate was rapidly eliminated from the system by metabolism. The depuration data also indicated that the substance was rapidly eliminated by the fish. Based on these data, the initial rate of uptake and the rate of depuration in tap water were determined to be 0.52 ± 0.30 $hour^{-1}$ and 1.62 ± 0.67 $hour^{-1}$ respectively, giving a BCF of around 0.29 for diphenyl phosphate. The log K_{ow} for diphenyl phosphate was determined to be around 1.29 using a thin layer chromatographic method based on that used by Renberg *et al.* (1980).

Table 3.8 Uptake and depuration of ^{14}C-diphenyl phosphate by rainbow trout

Time (minutes)	Concentration of ^{14}C in whole fish (μg/kg)	
	Dechlorinated tap water	River water
Uptake phase of experiment		
10	3.2 ± 2.7	2.6 ± 2.9
20	15.2 ± 3.9	4.9 ± 2.2
30	10.7 ± 2.5	7.1 ± 0.9
60	1.9 ± 0.7	2.0 ± 1.7
270	0.5 ± 4.7	1.6 ± 9.0
Depuration phase of experiment (following exposure to 100 μg/l for 30 minutes)		
15	4.4 ± 2.4	
45	2.7 ± 2.6	
90	1.9 ± 2.0	

Source: Muir and Grift (1981).

The accumulation of a commercial hydraulic fluid (Pydraul 50E) in lake trout (*Salvelinus namaycush*) was studied by Mayer *et al.* (1993) as part of a toxicity test (further details of the toxicity study are given in Annex A). The substance tested consisted of 36 per cent triphenyl phosphate, 40 per cent nonylphenyl diphenyl phosphate, 22 per cent cumylphenyl diphenyl phosphate and two per cent other components. The test was carried out for 120 days using a flow-through system. The test was started by placing eyed eggs (eight replicates of 25 each) into chambers (120-litre chambers, water depth 15 cm, water replacement rate of 250 ml every three minutes) with total concentrations of phosphate esters of 2.6 μg/l, 5.3 μg/l or 16.0 μg/l (mean measured concentrations as the sum of the three main components of the commercial product over nine sampling periods) plus a control group. At various times during the study (day 15, 30, 60, 90 and 120), fish were collected from each exposure and control group and analysed for the presence of each component of the commercial product. The results are shown in Table 3.9. Based on the measured concentrations of triphenyl phosphate in water and fish, a BCF of around 886 l/kg can be derived from the data. Toxic effects were seen, including mortality from day 60 onwards, at the highest concentration tested and effects on growth from day 60 onwards at the middle and highest concentration tested. Effects on growth in particular may have skewed the results from this study for triphenyl phosphate, as the BCF appears to increase with increasing exposure concentration.

A similar study was carried out by Mayer *et al.* (1981) using rainbow trout fry (*Oncorhynchus mykiss*). The substances tested in this study included Pydraul 50E (36 per cent triphenyl phosphate, 40 per cent nonylphenyl diphenyl phosphate and 22 per cent cumylphenyl diphenyl phosphate) Pydraul 115E (seven per cent triphenyl phosphate, 29 per cent nonylphenyl diphenyl phosphate and 62 per cent cumylphenyl diphenyl

phosphate), and also pure triphenyl phosphate, nonylphenyl diphenyl phosphate and cumylphenyl diphenyl phosphate. The test was carried out at 12°C for 90 days using a flow-through system. Concentrations of the individual alky/aryl phosphates in the water phase were determined at two-weekly intervals during the study, and whole-body concentrations of each component were determined after 90 days exposure. The results for triphenyl phosphate are summarised in Table 3.10, along with estimated BCF values. The mean BCF estimated from this data is 271 ± 80 l/kg. The equivalent BCFs for pure nonylphenyl diphenyl phosphate and cumylphenyl diphenyl phosphate are estimated to be 691 ± 223 l/kg and 2,807 ± 988 l/kg respectively from the data reported in the paper.

Table 3.9 Bioconcentration of components of a commercial hydraulic fluid by lake trout

Exposure period (days)	Measured concentration in water (µg/l)			Measure concentration in whole fish (mg/kg)			Bioconcentration factor (l/kg)		
	TPP	NPDPP	CPDPP	TPP	NPDPP	CPDPP	TPP	NPDPP	CPDPP
15	0.81	1.2	0.54	0.23	0.58	0.35	284	483	648
	2.0	2.2	1.1	2.0	4.4	7.7	1,000	2,000	7,000
	6.3	6.0	3.6	10	7.0	25	1,587	1,167	6,944
30	0.81	1.2	0.54	0.73	0.84	0.76	901	700	1,407
	2.0	2.2	1.1	1.4	6.9	13	700	3,136	11,818
	6.3	6.0	3.6	4.7	12	46	1,905	2,000	12,778
60	0.81	1.2	0.54	0.11	0.76	0.51	136	425	944
	2.0	2.2	1.1	1.3	6.9	10	650	3,136	9,091
	6.3	6.0	3.6	4.0	17	50	635	2,833	13,889
90	0.81	1.2	0.54						
	2.0	2.2	1.1	1.6	4.7	8.5	800	2,136	7,727
	6.3	6.0	3.6	6.8	26	55	1,079	4,333	15,278
120	0.81	1.2	0.54	0.16	0.55	0.48	198	458	889
	2.0	2.2	1.1	2.5	5.3	12	1,250	2,409	10,909
	6.3	6.0	3.6	a	a	a			
						Mean	886±521	1,940±1,238	7,640±5,269

Source: Mayer *et al.* (1993).

Notes: a) 100 per cent mortality occurred.
TPP = Triphenyl phosphate.
NPDPP = Nonylphenyl diphenyl phosphate.
CPDPP = Cumylphenyl diphenyl phosphate.

Table 3.10 Accumulation of triphenyl phosphate in rainbow trout over 90 days

Measured concentration in water (mg/l)	Measured concentration in whole fish (mg/kg)	BCF (l/kg)
Control	Not detected	
0.00022	0.080	364
0.00038	0.11	289
0.00044	0.13	295
0.00064	0.13	203
0.00091	0.12	132
0.0012	0.41	342
0.0014	0.38	271
	Mean	271 ± 80

Source: Mayer *et al.* (1981).

The bioconcentration of a commercial triaryl phosphate product in bleak (*Alburnus alburnus*) was investigated by Bengtsson *et al.* (1986). The substance tested contained triphenyl phosphate, cresyl diphenyl phosphate (two main components), tricresyl phosphate (three main components) and trixylenyl phosphate (three main components) – a quantitative composition was not included. Tests were carried out using a flow-though system with natural brackish water (7‰ salinity) at 10°C. The fish used in the test had an average weight of five grams (53 fish were used in 60 litres of water) and were fed twice daily (once daily at weekends) with a 0.25 g portion of commercial food. The pH of the water was 7.6-7.9 and the dissolved oxygen concentration remained above 90 per cent of saturation throughout the study. The experiment consisted of a 14-day uptake period where the fish were exposed to a nominal concentration of 50 μg/l of the triaryl phosphate product, followed by a 14-day depuration period in clean-flowing water. Samples of both fish (three fish per sample time except at day 14 and 28 where five groups of three fish were sampled) and water were analysed for concentrations of the main components (as determined by gas chromatographic analysis) of the triaryl phosphate product on days 0, 1, 2, 4, 7, 14, 17, 18, 21 and 28 of the experiment. No mortality or abnormal behaviour was seen in the fish during the experiment. Steady state was found to have been reached within the 14-day exposure period (steady state was actual attained within two days) for triphenyl phosphate, the cresyl diphenyl phosphate components and two of the tricresyl phosphate components of the mixture. Steady-state BCFs were determined to be 400 l/kg, 100-220 l/kg and 800 l/kg for these components respectively. For the other components, steady state was approached, but had not been reached by the end of the 14-day uptake period and the non-steady state BCF values estimated at 14 days were 400 l/kg for the remaining tricresyl phosphate component and 1,300-1,900 l/kg for the three trixylenyl phosphate components. All components were found to be rapidly eliminated from the fish, with a depuration half-life of four days or less. The triphenyl phosphate, cresyl diphenyl phosphate and tricresyl phosphate components were almost completely eliminated from the fish within 14 days but the trixylenyl phosphate components were still evident in the fish after 14 days.

Uptake and accumulation of the triphenyl phosphate component of several commercial phosphate ester products by fathead minnows (*Pimephales promelas*) were studied as part of a 90-day partial life-cycle toxicity study. The substances tested included Fyrquel GT (containing 15-20 per cent triphenyl phosphate), Kronitex 200 (containing 4 to 6 per cent triphenyl phosphate), Phosflex 31 P (containing 28-30 per cent triphenyl phosphate), Pydraul 50E (containing 36 per cent triphenyl phosphate) and Santicizer 148 (containing six per cent triphenyl phosphate). Fish were exposed to five concentrations of the commercial products for up to 90 days in a flow-through system. At 30, 60 and 90 days of exposure, a composite sample of ten fish was removed from each treatment group and

analysed for the concentration of triphenyl phosphate and other phosphate esters present in the products. Concentrations of the triphenyl phosphate component in the water were also determined at two-weekly intervals. The results are summarised in

The uptake of ^{14}C-labelled triphenyl phosphate (radiopurity above 98 per cent) by fourth instar larvae of the midge *Chironomus tentans* was investigated using various water-sediment systems (Muir *et al.* 1983b). The sediments investigated were a pond sediment (25 per cent silt and 75 per cent clay, 3.7 per cent organic carbon content, 60 per cent water content and pH 7.6), a river sediment (seven per cent sand, 45 per cent silt and 48 per cent clay, 2.3 per cent organic carbon content, 50 per cent water content and pH 7.7) and sand (100 per cent sand, 0.1 per cent organic carbon content, 25 per cent water content and pH 7.0). The test system consisted of 50 grams of wet sediment spiked with 50 or 500 μg/kg of the test substance (the substance was added as a solution in acetone) and 250 ml of dechlorinated tap water (giving a sediment:water ratio of 1:5). The spiked sediment-water system was allowed to equilibrate prior to addition of the larvae. Larvae were either suspended in the water phase above the sediment in cages (20 larvae per replicate) or added directly to the water-sediment system (40 larvae per replicate). Four replicates were carried out for each treatment. The concentration of radiolabel in the larvae (five organisms sampled on each occasion per replicate) and water phase was determined at various times during the 96-hour exposure period. Following this period, the remaining organisms were transferred to vessels containing clean silica sand and dechlorinated tap water and depuration over 48 hours was monitored.

Table 3.11. The mean BCFs determined at 90 days for triphenyl phosphate were 1,958 ± 383 l/kg in the experiments with Fyrquel GT, 1,206 ± 355 l/kg in the experiments with Kronitex 200, 1,758 ± 1,506 l/kg in the experiment with Phosflex 31P, 1,534 ± 268 l/kg in the experiments with Pydraul 50E and 1,007 ± 224 l/kg in the experiments with Santicizer 148. When placed in clean water, depuration of the triphenyl phosphate component from the fish was found to be rapid, with half-lives of under seven days. Toxic effects were seen in many of these experiments, particularly on growth, and so this adds some uncertainty to the BCF estimates.

A BCF of 271 l/kg was determined for rainbow trout at 12°C using a static system (Sitthichaikasem 1978, cited in WHO 1991). The value was based on parent compound analysis of the fish tissues. However, the original source of this data appears to have carried out bioconcentration studies on tri-*p*-cresyl phosphate rather than triphenyl phosphate and so the basis behind this value is unclear.

Lombardo and Egry (1979) determined a BCF of 190-280 l/kg for triphenyl phosphate in edible tissue of rainbow trout (*Oncorhynchus mykiss*). The fish were exposed to concentrations of 2.3, 8.8 or 12.8 μg/l of a commercial product that contained around 35 per cent triphenyl phosphate (along with nonylphenyl diphenyl phosphate and cumylphenyl diphenyl phosphate) for 90 days. Few other details of the study are available.

The uptake of ^{14}C-labelled triphenyl phosphate (radiopurity above 98 per cent) by fourth instar larvae of the midge *Chironomus tentans* was investigated using various water-sediment systems (Muir *et al.* 1983b). The sediments investigated were a pond sediment (25 per cent silt and 75 per cent clay, 3.7 per cent organic carbon content, 60 per cent water content and pH 7.6), a river sediment (seven per cent sand, 45 per cent silt and 48 per cent clay, 2.3 per cent organic carbon content, 50 per cent water content and pH 7.7) and sand (100 per cent sand, 0.1 per cent organic carbon content, 25 per cent water content and pH 7.0). The test system consisted of 50 grams of wet sediment spiked with 50 or 500 μg/kg of the test substance (the substance was added as a solution in acetone) and 250 ml of dechlorinated tap water (giving a sediment:water ratio of 1:5). The spiked sediment-water system was allowed to equilibrate prior to addition of the larvae. Larvae were either suspended in the water phase above the sediment in cages (20 larvae per replicate) or added directly to the water-sediment system (40 larvae per replicate). Four replicates were carried out for each treatment. The concentration of radiolabel in the

larvae (five organisms sampled on each occasion per replicate) and water phase was determined at various times during the 96-hour exposure period. Following this period, the remaining organisms were transferred to vessels containing clean silica sand and dechlorinated tap water and depuration over 48 hours was monitored.

Table 3.11 Bioconcentration of triphenyl phosphate present in several commercial aryl and aryl/alkyl phosphates in fathead minnow

Mean measured concentration in water (mg/l)		Mean measured concentration of triphenyl phosphate in fish (mg/kg wet wt.)			BCF at 90 days (l/kg)
		30 days	60 days	90 days	
Fyrquel GT	0.005	1	4	8	1,600
	0.010	1	4	16	1,600
	0.022	1	22	42	1,909
	0.042	18	60	93	2,214
	0.111	54	246	274	2,468
	control	1	<0.2	1	
Kronitex 200	0.002	1	2	3	1,500
	0.003	2	2	3	1,000
	0.005	3	4	5	1,000
	0.009	5	14	15	1,667
	0.036	15	16	31	861
	control	<1	<1	<1	
Phosflex 31P	0.005	2	3	4	800
	0.008	4	5	8	1,000
	0.013	6	20	16	1,231
	0.014	12	28	56	4,000
	control	<0.8	<0.2	<0.2	
Pydraul 50E	0.008	10	8	10	1,250
	0.014	20	16	22	1,571
	0.029	40	32	47	1,621
	0.061	86	90	80	1,311
	0.176	316	339	338	1,920
	control	<0.2	0.3	<0.2	
Santicizer 148	0.001	0.8	0.5	0.7	700
	0.002	1	1	2	1,000
	0.003	3	1	3	1,000
	0.006	6	3	6	1,000
	0.021	16	20	28	1,333
	control	0.2	<0.2	1	

Source: Cleveland *et al.* (1986).

Concentration of triphenyl phosphate in the overlying water was found to reach a steady-state concentration with that in the sediment within the two-day equilibration period, and remained reasonably constant until the end of the study (the overlying water concentration was 2.1, 0.5 and 2.0 μg/l for the pond, river and sand sediments spiked at 50 μg/kg and 13.6, 10.2 and 19.2 μg/l for the three sediments spiked at 500 μg/kg, respectively). At steady state, the majority of the triphenyl phosphate added to the system was associated with the sediment phase (85 per cent of the total added in the pond sediment, 89 per cent of the total in the river sediment and 79 per cent of the total in the sand). Separate experiments showed that degradation of triphenyl phosphate in the test system was negligible over the equilibration period and first 24 hours of exposure.

The concentration of radioactivity in the larvae exposed solely via the water phase was found to reach steady state within 24 hours of exposure. The half-life for elimination was in the range 18 to 63 hours. Based on the measured concentrations of radioactivity in *C. tentans* (exposed via sediment-water and via the water phase alone) and water, the BCF

after 24 hours was found to be in the range 6 to 173 l/kg at the high-exposure concentration and 12 to 208 l/kg at the low-exposure concentration. The higher BCFs were generally obtained in the experiments with sand. The paper estimated that triphenyl phosphate was roughly equally taken up from the water and sediment phase in organisms exposed via the sediment.

Uptake from food

A long-term dietary accumulation study with a commercial triaryl phosphate product was carried out with minnows (*Phoxinus phoxinus*) (Bengtsson *et al.* 1986). The substance tested contained triphenyl phosphate, cresyl diphenyl phosphate (two main components), tricresyl phosphate (three main components) and trixylenyl phosphate (three main components). The test was carried out using a flow-through system with six groups of 30 fish (average body weight 1.9 g), each in 50 litres of natural brackish water (7‰ salinity). The test was started in January and was carried out for 163 days (although the resulting concentrations in fish were determined after four months only) and water temperature followed natural seasonal fluctuations (3.6-4.5°C from January to April, 7°C by the end of April, 13°C by the end of May and 12 to 15°C from June until the end of the experiment). The dissolved oxygen concentration was always above 90 per cent of saturation and the pH of the water was 7.7 to 8.0. Five concentrations of the test substance were used (100, 300, 1,000, 3,000 and 10,000 mg/kg food) and a control using uncontaminated food was run. The food used was a commercial fish food that was spiked by adding the triaryl phosphate as a solution in acetone and evaporating off the solvent. The fish were given two 0.25 g portions of food per day. The total amount of food given to the fish by four months of the experiment was two grams per fish. No mortalities or abnormal behaviour were seen in any of the fish, and all food given to the fish was eaten. Concentrations of the various components in the fish after four months exposure are shown in Table 3.12. Overall, only around 0.017-0.14 per cent of the total amount of test substance fed to fish was found to be present in the fish at the end of the study. The bioaccumulation factors, based on the estimated concentrations in fish and in food, are all very much less than one.

Table 3.12 Concentrations in minnows after four months exposure to contaminated food

Food concentration	Total concentration in fish (mg/kg fresh weight)			
	Triphenyl phosphate	Cresyl diphenyl phosphate (sum of 2 components)	Tricresyl phosphate (sum of 3 components)	Trixylenyl phosphate (sum of 3 components)
Control	0.005	Not detected	0.005	Not detected
100 mg/kg	0.030	0.023	0.053	0.094
300 mg/kg	0.020	0.016	0.085	0.47
1,000 mg/kg	0.225	0.019	0.225	0.896
3,000 mg/kg	0.12	0.016	0.88	2.01
10,000 mg/kg	0.73	0.043	1.39	2.05

Calculated data

For the terrestrial food chain, the TGD requires a BCF for earthworms. No experimental data are available for this endpoint and so an earthworm BCF value is estimated using the following equation given in the TGD:

$$BCF_{earthworm} = 0.84 + 0.012\ K_{ow}/RHO_{earthworm}$$

Where $RHO_{earthworm}$ = density of the earthworm = 1 kg/l

K_{ow} = octanol-water partition coefficient

Using a log K_{ow} of 4.63 and methods outlined in the TGD, the $BCF_{earthworm}$ is estimated as 513 l/kg. This value is used in the assessment, though its reliability is unknown.

Summary of accumulation

Available fish BCFs are summarised in The log K_{ow} of triphenyl phosphate is 4.63. Using the methods recommended in the TGD, a BCF for fish of 1,720 l/kg can be estimated. This value is higher than those determined in the more reliable studies based on parent compound analysis, but is similar to those determined using ^{14}C-measurements.

A BCF value of 420 l/kg is used in this risk assessment for triphenyl phosphate.

In addition to a BCF, the revised TGD also requires a biomagnification factor (BMF) to be taken into account. For triphenyl phosphate, the default BMF would be 1 based on the BCF values determined above. This is consistent with the available feeding study data that showed that bioaccumulation from food was low.

Using a log K_{ow} of 4.63 and the methods recommended in the TGD, the $BCF_{earthworm}$ is estimated to be 513 l/kg.

Table 3.13. Numerous studies have shown bioconcentration of triphenyl phosphate in fish. Some studies, however, are limited in their usefulness for risk assessment, as the concentration was not maintained adequately during the study (and so the resulting BCF does not represent a steady-state value) or the result was based on ^{14}C-measurements (and so may lead to an overestimate of the actual bioconcentration factor for triphenyl phosphate if extensive metabolism occurred in the fish), or effects on growth were seen (the actual effect of this on the BCF is unclear but adds some uncertainty to interpretation of the data).

The log K_{ow} of triphenyl phosphate is 4.63. Using the methods recommended in the TGD, a BCF for fish of 1,720 l/kg can be estimated. This value is higher than those determined in the more reliable studies based on parent compound analysis, but is similar to those determined using ^{14}C-measurements.

A BCF value of 420 l/kg is used in this risk assessment for triphenyl phosphate.

In addition to a BCF, the revised TGD also requires a biomagnification factor (BMF) to be taken into account. For triphenyl phosphate, the default BMF would be 1 based on the BCF values determined above. This is consistent with the available feeding study data that showed that bioaccumulation from food was low.

Using a log K_{ow} of 4.63 and the methods recommended in the TGD, the $BCF_{earthworm}$ is estimated to be 513 l/kg.

Table 3.13 shows that when more reliable data based on parent compound analysis are considered, the BCF is generally around 271 to 420 l/kg. Higher values (up to 1,743 l/kg) have been determined based on ^{14}C measurements but this study found the BCF based on parent compound to be around 420 l/kg. The difference between the values obtained on ^{14}C measurements and parent compound measurements reflects the relatively rapid metabolism of triphenyl phosphate in fish. Metabolites of triphenyl phosphate have generally been shown to be rapidly excreted from fish.

The log K_{ow} of triphenyl phosphate is 4.63. Using the methods recommended in the TGD, a BCF for fish of 1,720 l/kg can be estimated. This value is higher than those determined in the more reliable studies based on parent compound analysis, but is similar to those determined using ^{14}C-measurements.

A BCF value of 420 l/kg is used in this risk assessment for triphenyl phosphate.

In addition to a BCF, the revised TGD also requires a biomagnification factor (BMF) to be taken into account. For triphenyl phosphate, the default BMF would be 1 based on the BCF values determined above. This is consistent with the available feeding study data that showed that bioaccumulation from food was low.

Using a log K_{ow} of 4.63 and the methods recommended in the TGD, the $BCF_{earthworm}$ is estimated to be 513 l/kg.

Table 3.13 Summary of bioconcentration factors for triphenyl phosphate

BCF (l/kg)	Species	Comment	Rel.	Ref.
400	*Alburnus alburnus*	14-day flow-through study. The substance tested was a mixture of triaryl phosphate. BCF based on parent compound measurements.	2	Bengtsson *et al.* 1986
110-150	*Carassius auratus*	96-hour study. Exposure concentration not maintained. Based on parent compound measurements.	3	Sasaki *et al.* 1981
250-500	*Oryzias latipes*	96-hour study. Exposure concentration not maintained. Based on parent compound measurements.	3	Sasaki *et al.* 1981
84		18-day flow-through study. Based on parent compound measurements. Concentration in fish was still increasing at the end of the study.	3	Sasaki *et al.* 1982
189-193		32-35 day flow-through study. Based on parent compound measurements. Exposed in combination with tris(1,3-dichloroispropyl) phosphate. Concentration in fish was still increasing at the end of the study.	3	Sasaki *et al*. 1982
157-390		Static study. Based on parent compound measurements.	4	Sasaki *et al.* 1982
2,590-18,900	*Oncorhyn-chus mykiss*	24-hour study. Based on ^{14}C measurements. BCF estimated using the initial rate method, but the rate of uptake and rate of depuration were determined on different populations using different concentrations and so are uncertain.	3	Muir *et al.* 1980
573-1,368		24-hour study. Based on ^{14}C measurements. The concentration in water was found to decrease with time and so the BCF was estimated using the initial rate method and a method that takes this decrease into account. BCF based on parent compound was estimated to be 324 l/kg.	2	Muir *et al.* 1983a
132-364 (mean 271)		120-day flow-through study. The substance was tested as a mixture of triaryl phosphates and as a pure substance. The BCF was based on parent compound measurements. Toxic effects were seen at the higher concentrations tested (these concentrations resulted in the higher BCFs).	2	Mayer *et al.* 1981
270		The substance tested may have been tri-p-cresyl phosphate.	4	WHO 1991
190-280		90-day study. Value is for the edible tissues.	4	Lombardo and Egry
561-1,743	*Pimephales promelas*	24-hour study. Based on ^{14}C measurements. The concentration in water was found to decrease with time and so the BCF estimated using the initial rate method and a method that takes this decrease into account. The BCF based on parent compound was estimated to be 420 l/kg.	2	Muir *et al.* 1983a
68-160		24-hour study. Based on ^{14}C measurements using an artificial pond. The concentration in water was not maintained during the study.	3	Muir *et al.* 1982
Mean values 1,007-1,958		90-day flow-through study. The substance tested was a mixture of triaryl phosphate. The BCF based on parent compound measurements. Toxic effects (including effects on growth) were seen in many of these studies.	3	Cleveland *et al.* 1986.
136-1,905 (mean 886)	*Salvelinus namay-cush*	120-day flow-through study. The substance tested was a mixture of triaryl phosphate. The BCF based on parent compound measurements. Toxic effects were seen at the higher concentrations tested (these concentrations resulted in the higher BCFs).	3	Mayer *et al.* 1993.

Notes: Rel. = Reliability: 1) Valid without restrictions. 2) Valid with restrictions. 3) Not valid. 4) Insufficient information.

3.2 Environmental releases

3.2.1 General discussion

Releases from the production and use of triphenyl phosphate were estimated using a number of sources such as default methods from the TGD and the Emission Scenario Document (ESD) on plastics additives (OECD 2004). In the absence of specific information on the substance, the ESD is considered to be a reasonable basis for emission estimation; TGD default values are intended for use as realistic worst case values in the absence of other data. Hence, estimates from these sources will have some degree of uncertainty. The actual calculations are considered confidential as they are based on confidential production and use figures.

The producers of triphenyl phosphate provided information on the amounts used by representative large customers, and this was used in the local estimates of emissions from use. Some additional information on waste treatment and cleaning at a small number of user sites was also provided; this information did not contradict the assumptions made on the basis of the ESD.

A study carried out for the United States Environmental Protection Agency (USEPA) in 1979 estimated that, at the time, 1 to 3 per cent of all phosphate esters would be lost to the environment during manufacturing, transportation and recycling, and that around 75 per cent of the annual use in hydraulic fluids would be lost to the environment by leakage (Midwest Research Institute 1979, cited in Mayer *et al.* 1981).

As noted in Section 1.2.3, triphenyl phosphate is a significant impurity in several other substances reviewed in this series of assessments. The contribution of these substances to the overall emissions of triphenyl phosphate has not been assessed in detail, but is considered briefly in Section 5.

3.2.2 Releases from production

Releases from production sites were estimated from specific information provided by the producing companies. The results are included in Table 3.15.

3.2.3 Releases from use (processing)

Emissions from the use in various polymers and resins were estimated using the methods outlined in the Emission Scenario Document on plastics additives (OECD 2004). The ESD provides methods for estimating the releases from three stages:

- handling of raw materials;
- compounding – the blending into the polymer of additives;
- conversion – the forming of the polymer into finished articles.

The first two stages are assumed to always take place together. There are companies which compound the plastics and then sell them on to converters, so separate calculations are carried out for the two as well as for the case where compounding and conversion take place together. Emission factors in the ESD are derived from information on a model substance, di(2-ethylhexyl)phthalate (DEHP), and are modified according to the relative properties of this substance and the substance of interest. The main property affecting the emissions is the vapour pressure of the substance. Triphenyl phosphate has a higher vapour pressure than DEHP, and is classed as of ‘high volatility’ according to the

criteria in the ESD[6]. The ESD also uses the particle size of the substance in estimating possible releases from raw materials handling. Triphenyl phosphate is supplied as flakes or pellets (Section 1.3.1) and these forms are considered to be above the threshold criteria value of 40 µm.

The emission factors derived using the ESD methods are:

- Compounding (including raw materials handling): 0.025 per cent to air, 0.245 per cent to waste water.
- Conversion: 0.025 per cent to air, 0.025 per cent to waste water.

These are applied to the production of all four types of plastic.

The ESD does not assume direct release to water through use of water in the processes. Instead it is assumed that releases occur through volatilisation, with some of this deposited on floors or surfaces and the remainder released to external air. Deposited material is assumed to be removed through washing with water, hence the releases to waste water.

3.2.4 Releases over lifetime of products

Triphenyl phosphate is used in products which are expected to have extended service lives (more than one year). These are therefore potentially important sources of emission. Possible losses from products through leaching and volatilisation are considered in this section. Limited information on the release of triphenyl phosphate is available, and was used as the basis for estimates. This information was used in the methods outlined in the Emission Scenario Document (OECD 2004) and also takes into account the approaches used in the risk assessment of other substances (for example, the risk assessment on medium-chain chlorinated paraffins carried out under the Existing Substances Regulation (ECB 2005)). The approach taken also considers the release of polymer particulates (waste remaining in the environment) over the lifetime of products and at disposal as appropriate; this is based on the treatment of this area in other risk assessments such as that on medium-chain chlorinated paraffins.

Leaching loss

Ahrens *et al*. (1978) investigated the extraction of triphenyl phosphate from a vinyl automotive upholstery fabric. The fabric had a calendered finish and a cotton or cotton/polyester bonded backing material. The material (4 × 9 inch area (approximately 232 cm^2)) was shaken with 500 ml of well water for 24 hours and then the water was extracted with benzene and the extracts analysed for organic compounds. Triphenyl phosphate was identified as the major substance present in the water extracts. No information was given in the paper on the concentration of triphenyl phosphate in either the starting vinyl fabric sample or the subsequent water extracts. Another test exposed six goldfish (*Carassius auratus*) to the vinyl fabric (7 × 15 inch area (approximately 677 cm^2)) in 20 litres of water for 24 hours. In this test all the goldfish died within the 24-hour period, and death was preceded by hyperactivity, disoriented swimming, sluggishness, aimless drifting and maintenance of a vertical position in the water. Subsequently the fabric sample was removed from the tank, rinsed and placed in clean water containing further goldfish. These fish again displayed the above toxic symptoms but no deaths were observed within the 24-hour exposure period. The authors also exposed goldfish directly to triphenyl phosphate in well water and found that the same toxic symptoms were shown

[6] 'High volatility' is used in comparison to DEHP which is of 'medium volatility'. All phosphates assessed in this series have vapour pressures considered low for organic substances.

in fish exposed to 1, 3 or 5 mg/l of triphenyl phosphate (but not in control fish), and that all fish were dead within eight hours at 1 mg/l, five hours at 3 mg/l and one hour at 5 mg/l. These data imply that the concentrations of triphenyl phosphate in water in the extraction experiment with fish were approaching 1 mg/l.

The use of triphenyl phosphate in vinyl fabrics does not form part of the current use pattern of the substance (see Section 2). The types of product made using triphenyl phosphate include computer casings, printed circuit boards and photo film, none of which are likely to come into contact with water during their normal use. As a result, the possibility of losses through leaching during the service life of these products is considered to be negligible. The exception to this is use in thermoset plastics, for which a loss to water of 0.5 per cent over the service life of the articles is assumed.

Volatile loss

The stability of, and volatile loss from, several commercial aryl and alkyl/aryl phosphate products has been studied using a combination of differential scanning calorimetry (DSC) and thermogravimetric analysis (TGA) under a nitrogen atmosphere (Shankwalkar and Cruz 1994) and an oxygen atmosphere (Shankwalkar and Placek 1992). The results of the studies are summarised in

Table 3.14. The results under a nitrogen atmosphere show that the triaryl phosphates start to decompose at temperatures of around 310-350°C, whereas alkyl diphenyl phosphates start to decompose at a temperature of around 260°C. Decomposition temperatures under an oxygen atmosphere are significantly lower. For all the substances tested, significant weight loss occurred at temperatures below that at which decomposition begins, indicating a loss of the substance by volatilisation at elevated temperatures.

Although triphenyl phosphate itself was not studied under a nitrogen atmosphere, several of the triaryl phosphate products tested would have contained significant amounts of triphenyl phosphate (see Section 1.2.3) and so it can be inferred that the weight loss likely from triphenyl phosphate at elevated temperature would be similar to that seen from other triaryl phosphates under these conditions.

Brauman (1977) carried out a series of weight loss/pyrolysis experiments with triphenyl phosphate (Phosflex TPP) in various polymers (including low density polyethylene, polypropylene, polystyrene, various polyesters, polyethylene terephthalate and flexible dense polyurethane) at 20 per cent loadings. The studies found that at temperatures up to around 350°C in nitrogen or air atmospheres, the triphenyl phosphate was found to volatilise rapidly from the polymer without degrading. At temperatures above around 350°C, volatilisation of the undegraded triphenyl phosphate was still apparent, but there was also evidence that triphenyl phosphate degraded within the polymer matrix to form, amongst other products, orthophosphoric acid.

Table 3.14 Thermal degradation temperature and weight loss of aryl and alkyl/aryl phosphates

Phosphate ester	Experiments under an oxygen atmosphere				Experiments under a nitrogen atmosphere		
	Start of thermal degradation	1% weight loss	5% weight loss	10% weight loss	Start of thermal degradation	5% weight loss	10% weight loss
Triphenyl phosphate	>400°C	188°C	236°C	252°C			
Tricresyl phosphate	215°C	184°C	255°C	252°C	333°C	272°C	306°C
Trixylenyl phosphate	210°C	224°C	268 °C	286°C	311°C	276°C	302°C
Isopropylphenyl diphenyl phosphate[a]	210-215°C	200-218°C	239-265 °C	263-288°C	311-314°C	264-282°C	293-307°C
Tertbutylphenyl diphenyl phosphate[a]	295-305°C	213-234°C	262-277°C	280-295°C	338-347°C	274-278°C	305-306°C
2-Ethylhexyl diphenyl phosphate	200°C	90°C	220°C	229°C	257°C	226°C	231°C
Isodecyl diphenyl phosphate	165°C	93°C	213°C	235°C	264°C	233°C	246°C

Notes: a) Data for three (nitrogen atmosphere) or four (oxygen atmosphere) different grades.

The weight loss on heating a 10 mg sample of a commercial triphenyl phosphate (Reofos TPP) at a rate of 10°C per minute under nitrogen atmosphere was determined as five per cent at 208°C, ten per cent at 225°C and 50 per cent at 272°C by thermogravimetric analysis (Great Lakes Chemical Corporation 2002).

Carlsson *et al.* (2000) investigated the concentrations of triphenyl phosphate in air in an office where a new computer was running. Sampling was carried out using personal exposure samplers and each sample (including particulates) was collected over a 700-minute period (total volume of air sampled was 2.1 m^3). A short-term pilot study was carried out using an office with an area of 12.4 m^2 and a height of 2.7 m (total volume = 33.48 m^3). The air exchange rate in the room was 3.5 air changes per hour (117.2 m^3 per hour). The room contained office furniture and two old (more than three years old) computers, a laser printer and a telephone. The concentration of triphenyl phosphate in the air of the office prior to installation of the new PC was 0.7 ng/m^3 (mean of three samples, CV = 22 per cent). The new computer introduced to the office had a triphenyl phosphate content of eight per cent by weight in the monitor housing and was operated continually for eight days. Air was collected from two samplers in the middle of the room (both one metre away from the computer and at a height of one metre). The concentration of triphenyl phosphate in air increased up to 82 ng/m^3 (mean of four samples, CV = eight per cent) after one day of operation, but had fallen to 39 ng/m^3 (mean of two samples) by day eight of the experiment.

Following on from the pilot study, a long-term study was carried out using a smaller office (area 9.6 m^2 and height 2.7 m, giving a total volume of 25.9 m^3) with an air exchange rate of 92 m^3 per hour (roughly 3.5 air changes per hour). The room contained office furniture but all electronic equipment was removed from the room eight weeks prior to the start of the experiment. The background concentration of triphenyl phosphate in the room was determined from samples collected on four consecutive days (two samplers each day at the same locations as those used for subsequent sampling with the computer present) prior to the addition of a new computer. The mean concentration found was 0.7 ng/m^3

(mean of eight samples, CV = 32 per cent) with above 99 per cent associated with the particulate phase. The new computer had a triphenyl phosphate content of ten per cent by weight in the monitor housing. Once installed in the office, levels of triphenyl phosphate were determined (sampled at 0.5 m for the computer monitor in the breathing zone of an imaginary operator sitting in front of the computer) each day for two days. After this period, the computer was switched on and kept in continuous operation for 183 days and samples were collected at various time points. During this time, the top of the monitor reached a temperature of 50°C. During the initial two days, when the computer was present in the room but not switched on, the concentration of triphenyl phosphate in air was similar to the background concentration found before the computer was installed. On the first day of operation of the computer, the concentration of triphenyl phosphate in air had increased to 94 ng/m^3 (mean of two samples). The concentration of triphenyl phosphate in air then gradually decreased with increasing time of operation of the computer, decreasing to around 42 per cent (roughly 39 ng/m^3) of the initial concentration by day eight and to a steady concentration of around 8.6 ng/m^3 (mean of two samples) by day 183. The authors indicated 183 days of continuous operation is equivalent to around two years under normal operating conditions.

Using the known room volumes and air exchange rate, it is possible to estimate the daily steady-state emission of triphenyl phosphate from the computer. The total volume of the room is 25.9 m^3 and hence a steady-state concentration of 8.6 ng/m^3 is equivalent to 223 ng in the room. Since the air exchange rate was around 3.5 changes per hour, the emission of triphenyl phosphate into the room must have been 3.5 × 223 = 781 ng/hour. This represents the steady-state emission of triphenyl phosphate from the working computer. The actual mass of triphenyl phosphate in the computer monitor is not given, but it was reported to be present at ten per cent by weight in the monitor housing. Assuming the monitor housing weighed around 1 to 2 kg, the amount of triphenyl phosphate initially present in the monitor would be around 0.1 to 0.2 kg. Thus, based on the emission rate of 781 ng/hour from the computer, the emission factor (based on the mass of triphenyl phosphate present) is estimated to be 0.39 to 7.8 μg/kg per hour of operation.

The authors also indicated that 183 days continuous operation would be equivalent to around two years under normal operating conditions. This leads to a figure of around 2,196 hours of operation per year under normal conditions. Applying this to the above emission rate, the yearly emission rate for triphenyl phosphate would be 0.86 to 17.1 mg/kg per year. Assuming a lifetime of 5 to 7 years for the equipment, this would give a lifetime emission factor of 4.3 to 119 mg/kg, or 4.3×10^{-4} to 0.012 per cent over the lifetime. The value of 0.012 per cent over the lifetime was used to estimate volatile losses from the use of triphenyl phosphate in thermoplastics. The substance is also used in a related area in printed circuit boards. These generally experience a higher operating temperature than the monitor housing, and so the emission factor is increased by a factor of five to 0.06 per cent over the lifetime of these products (this is an assumption, as there are no data on releases from printed circuit boards).

Photo film can be kept or stored for long time periods. Under storage conditions, emissions to the environment from the film will be limited. Emissions are most likely in the early part of the service life, when the film is initially used and then shown (movie film, slides) or used for prints. After this initial use, the film material will be accessed infrequently. An estimate of the average loss over the lifetime of the material is required. For the purpose of this assessment, an average of one-day emission per year over a lifetime of 100 years is assumed, using the thin film emission factors included in the plastics additives ESD (OECD 2004). The estimated loss factor to air from service life is 2.7 per cent.

Waste in the environment

This section considers the loss of substance in particles of plastic material from articles in use. The approach is the same as that used in the risk assessment for medium-chain chlorinated paraffins. Loss of particles of plastics from the products in use is not considered to be likely in view of the nature of uses. There is the potential for some loss on disposal, and a value of two per cent is used for thermoplastics, thermosets and printed circuit boards in line with other assessments. Losses on disposal of photo films are considered to be negligible. In the calculations, the substance in these particles is assumed to be available in the environment; this is likely to be an overestimate, but there are no actual data to indicate how much may be available.

Other sources of release

A small quantity of triphenyl phosphate is not allocated to one of the three use areas. It is assumed that this amount is in fact used in these areas, but passes through a longer supply chain and hence its use is not known to the major producers and suppliers who provided the information. To deal with this, an overall emission factor was derived from estimated releases from the quantity allocated to specific uses. This factor was applied to unallocated tonnage, and the release divided between the different compartments in the same ratio as for the allocated tonnage. These releases appear in the summary table (Table 3.15) under miscellaneous uses.

Triphenyl phosphate is a component of several other triaryl phosphates for which risk assessments have been carried out as part of this project (see Section 1.2.3). The triphenyl phosphate present in these products was not included in the calculations of environmental concentrations in this assessment, as the amount present in the other substances can vary depending on the substance and purpose for which it is used. This should not affect local estimates of releases, as they relate to the use of triphenyl phosphate. However, these will add to overall emissions and so regional environmental concentrations may be underestimated. This is considered in the risk characterisation.

There are no known natural sources of triphenyl phosphate (WHO 1991).

3.2.5 Summary of environmental releases

Estimated environmental releases of triphenyl phosphate are given in Table 3.15.

Table 3.15 Summary of estimated environmental release of triphenyl phosphate

Life cycle stage		Local (kg/day)		Regional (kg/year)			Continental (kg/year)		
		Air	Water	Air	Water[a]	Soil	Air	Water[a]	Soil
Production		0 0	5.9 <2		180 to surface water[b]			<172 to surface water[b]	
Printed circuit boards	Raw materials handling and compounding	0.125	1.225						
	Conversion	0.125	0.125						
	Raw materials handling, compounding and conversion	0.25	1.35	c	c				
	In service losses			0.6			5.4		
	Waste in the environment			0.02	4.96 to surface water[d]	14.9	0.18	44.7 to surface water[d]	135
Thermo-plastics/ styrenics	Raw materials handling and compounding	0.36	3.55						
	Conversion	0.36	0.36						
	Raw materials handling, compounding and conversion	0.73	3.91	c	c		c	c	
	In service losses			12.6			114		
	Waste in the environment			2.1	522 to surface water[d]	1,573	18.9	4,699	14,155

Table 3.15 continued.

Life cycle stage		Local (kg/day)		Regional (kg/year)			Continental (kg/year)		
		Air	**Water**	**Air**	**Water[a]**	**Soil**	**Air**	**Water[a]**	**Soil**
Thermosets and epoxy resins	Raw materials handling and compounding	0.125	1.225						
	Conversion	0.125	0.125						
	Raw materials handling, compounding and conversion	0.25	1.35	c	c		c	c	
	In service losses			17.5	35		158	315	
	Waste in the environment			0.14	34.5 to surface water[d]	104	1.25	310 to surface water[d]	935
Photographic film	Raw materials handling and compounding	0.126	1.226						
	Conversion	0.126	0.126						
	Raw materials handling, compounding and conversion	0.25	1.35	c	c		c	c	
	In service losses			576			5,103		
Miscellaneous uses				1.99	1.2 plus 1.7 to surface water	5.0	17.9	10.7 plus 15.1 to surface water	45.3
Total				887	2,314	1,697	5,804	7,661	15,257

Notes: a) Regional and continental emissions to water are split 80:20 between waste water treatment and direct discharge to surface water, except where noted.
b) Emissions calculated from site-specific data, after waste water treatment (sludges from production sites are incinerated, calculating the values after treatment allows this to be reflected in the emission estimates).
c) Values for individual steps are confidential, but are included in the total figure.
d) Releases from waste in the environment are assumed to go directly to surface water.

3.3 Environmental concentrations

3.3.1 Aquatic environment (surface water, sediment and wastewater treatment plant)

Calculation of PECs

PECs for surface water and sediment were estimated with the EUSES 2.0.3 program using the data summarised in the previous sections as input. Concentrations predicted for water and sediment are shown in Table 3.16.

Table 3.16 Summary of predicted local concentrations for the aquatic compartment

Scenario		PEC_{local}			
		Microorganisms in sewage treatment plant (mg/l)	**Surface water - emission episode (μg/l)**	**Surface water - annual average (μg/l)**	**Sediment (mg/kg wet wt.)**
Production of triphenyl phosphate[a]		0.01 and 1.0	0.3 and 0.03	0.29 and 0.03	0.07 and 6.29×10^{-3}
Printed circuit boards	Compounding	0.05	5.0	0.64	1.09
	Conversion	5.17×10^{-3}	0.52	0.08	0.11
	Combined compounding and conversion	0.06	5.51	0.7	1.2
Thermo-plastics/ styrenics	Compounding	0.15	14.5	11.9	3.16
	Conversion	0.01	1.48	1.22	0.32
	Combined compounding and conversion	0.16	15.9	13.1	3.48
Thermosets and epoxy resins	Compounding	0.05	5.0	0.02	1.09
	Conversion	5.17×10^{-3}	0.52	0.52	0.11
	Combined compounding and conversion	0.06	5.51	4.53	1.2
Photo-graphic film	Compounding	0.05	4.98	4.1	1.09
	Conversion	5.21×10^{-3}	0.52	0.43	0.11
	Combined compounding and conversion	0.06	5.51	4.53	1.2

Notes: a) PECs for production sites are calculated using known water flows at the sites.

Predicted regional concentrations are 0.01 μg/l for surface water and 2.43×10^{-3} mg/kg wet weight for sediment.

Predicted concentrations were also calculated for the marine environment using the EUSES program. These are included in Table 3.17. Note that production is not included in this table; production sites do not discharge to the marine environment.

Table 3.17 Summary of predicted local concentrations for the marine compartment

Scenario		PEC_{local}		
		Marine water - emission episode (μg/l)	**Marine water - annual average (μg/l)**	**Marine sediment (mg/kg wet wt.)**
Printed circuit boards	Compounding	6.04	0.76	1.32
	Conversion	0.62	0.08	0.14
	Combined compounding and conversion	6.65	0.84	1.45
Thermo-plastics/ Styrenics	Compounding	17.5	14.4	3.82
	Conversion	1.77	1.46	0.39
	Combined compounding and conversion	19.3	15.8	4.2
Thermosets and epoxy resins	Compounding	6.04	0.02	1.32
	Conversion	0.62	0.62	0.14
	Combined compounding and conversion	6.65	5.47	1.45
Photo-graphic film	Compounding	6.01	4.94	1.31
	Conversion	0.62	0.51	0.14
	Combined compounding and conversion	6.65	5.47	1.45

Measured levels in water and sediment

Water

Triphenyl phosphate was monitored in England and Wales over the period November 2007 – April 2008 as part of the Environment Agency's Targeted Risk Based Monitoring (TRBM) initiative. Six samples were collected at approximately weekly intervals from 15 paired WWTP effluent/receiving water sites across all eight Environment Agency Regions. The site selection criteria are not specified – it is likely that most are reasonably large WWTP with mixed industrial/household influent.

The limit of detection was 0.05 µg/l. The few positive detections of the substance are listed in Table 3.19.

Table 3.18 Positive detections of triphenyl phosphate associated with WWTP in England and Wales during 2007/8

Sampling location	EA Region	Date of sampling	Measured concentration, µg/l
Pyford/Curborough Brook	Midlands	19/2/08	0.0529
Huddersfield WWTP	North east	14/2/08	0.122
Davyhulme WWTP	North west	1/2/08	0.078
Manchester Ship Canal	North west	21/1/08	0.0781
		5/2/08	0.434
Eastleigh Chickenhall WWTP	Southern	6/3/08	0.0527
Great Stour	Southern	30/1/08	0.0639

Triphenyl phosphate was detected in two out of six samples from the Manchester Ship Canal (up to 0.434 µg/l). The canal is associated with the Davyhulme WWTP, which serves an industrial complex that includes a production site. However, the detection of the substance in the receiving water at times when it was not found in the effluent suggests that there could be other sources locally as well.

It has also been detected on isolated occasions in WWTP and/or receiving waters in three other regions at concentrations generally close to the detection limit. Again, the data tend to suggest the presence of sources upstream from the WWTPs. These sources may be difficult to trace since triphenyl phosphate is a component of several other substances.

Other countries

Measured levels of triphenyl phosphate in water from other countries are summarised in Table 3.19. The concentration of triphenyl phosphate in water from the Meuse at Eysden has been routinely monitored since 1998 (RIZA 2002). Samples were collected daily over a 24-hour period and the samples were filtered prior to extraction of the triphenyl phosphate. The limit of detection of the method used was 0.03 µg/l (when calculating the mean concentration in samples, a not-detected value was taken to be zero). In 1998, nineteen samples were found to contain triphenyl phosphate at concentrations above the detection limit. The concentrations found were in the range 0.03 to 0.15 µg/l, but the overall mean level for 1998 was below 0.03 µg/l. For 1999, 57 samples were found to contain triphenyl phosphate above the detection limit, with the range of concentrations spanning 0.04 to 0.50 µg/l and the overall mean below 0.03 µg/l. For 2000, triphenyl phosphate was found to be present in 45 samples at 0.03-0.22 µg/l, with the mean again below 0.03 µg/l. For 2001, triphenyl phosphate was found to be present in 41 samples at 0.03-0.21 µg/l with a mean below 0.03 µg/l. Finally, for 2002, triphenyl phosphate was found in five out of 103 samples so far analysed at a concentration of 0.04 to 0.22 µg/l.

Boutrup *et al.* (1998) determined the levels of triphenyl phosphate in freshwater river samples collected from the Country of Aarhus, Denmark in 1997-1998. The detection limit of the method used was around 0.2 µg/l and the substance was not detected in the four samples investigated from two rivers (Giber Å River and Møddebro Bœk Stream). The water was sampled over a 24-hour period on each occasion.

Lenhart and Lemm (1993) determined the levels of triphenyl phosphate in various water courses in Germany. The levels found were up to 0.28 µg/l at 20 locations along the river Ruhr (the highest concentrations were from a heavily industrialised area), a mean of 0.4 µg/l in tributaries of the Ruhr and up to 3.4 µg/l in the Emscher canal.

Triphenyl phosphate was reported at a concentration of 3 µg/l in effluent from a waste water treatment plant treating both domestic and industrial waste water in Sweden (Paxeus 1996, cited in OECD 2002).

Boethling and Cooper (1985) reported that triphenyl phosphate was not detected (detection limit 10 μg/l) in water samples collected near to an aryl phosphate production site and a large user of hydraulic fluids in the United States.

Levels of triphenyl phosphate in streams from 30 states across the United States were determined by Kolpin *et al.* (2002). In all, 85 stream samples (each sample was a composite collected from about four to six vertical profiles from the stream) from 1999-2000 were analysed and triphenyl phosphate was found to be present in 14 per cent of samples (detection limit was around 0.1 μg/l) at a maximum concentration of 0.22 μg/l. The samples appear to have been filtered (0.7 μm) prior to analysis and so the results probably reflect the concentration of triphenyl phosphate in the dissolved phase.

Sheldon and Hites (1978) determined the levels of triphenyl phosphate in water from an industrialised area of the Delaware River. Samples were collected in August 1976 (11 grab samples) and early March 1977 (five grab samples). The samples were not filtered prior to extraction and so the levels reported will reflect the amount of substance present in both the dissolved and particulate phase. Triphenyl phosphate was found in two out of five samples taken in March 1977 at a concentration of 0.1-0.3 μg/l, and in all 11 samples collected in August 1976 at a concentration of 0.1-0.4 μg/l. The main aim of the study was to identify the substances present rather than to accurately quantify the concentration present and so the concentrations given are only approximate.

A second study by Sheldon and Hites (1979) investigated the level of triphenyl phosphate in the Delaware River (including several water input and extraction sources) in the Philadelphia area. The study centred on the effluent from a chemical plant that discharged, along with the effluent from several other industrial sites, to an industrial sewage treatment plant and finally to the Delaware River and a drinking water treatment plant that extracted its water from the Delaware River. Samples were taken in August 1988 and were timed to follow the same “slug” of water through the water treatment system under investigation. Triphenyl phosphate was not detected in the effluent from the chemical plant but was present in the influent to the industrial waste water treatment plant (16 μg/l), effluent from the same plant (2 μg/l), Delaware River (trace-0.3 μg/l), inflow to drinking water treatment plant (0.2 μg/l) and treated drinking water (0.03 μg/l). Again, the concentrations found are only approximate.

Mayer *et al.* (1981) determined the levels of triphenyl phosphate in water samples from thirteen locations in Midwestern United States and San Francisco Bay. Samples were from industrial and non-industrial locations and were collected during November 1977 to May 1978. Triphenyl phosphate was detected at levels above 0.1 μg/l in 32 out of the 63 samples analysed, with the majority of levels being below one μg/l. The geometric mean of the levels found (using half of the detection limit (that is, 0.05 μg/l) for samples below the limit of detection) was 0.12 μg/l.

WHO (1991) report that triphenyl phosphate was detected at a concentration of 0.01-0.12 μg/l in drinking water from the United States.

A survey of the levels of triaryl phosphates in Canadian drinking water was carried out by Williams and LeBel (1981). Drinking water samples (sampled over a 24-hour period) were collected from 29 municipalities during August-September and again during November-December. In general, higher levels were found in drinking water associated with river sources rather than lake sources and groundwater sources. Overall, triphenyl phosphate was detected (detection limit around 0.05 ng/l) in around 35 of the 58 samples taken. The mean levels found were 1.7 ng/l in river water-derived samples, 0.6 ng/l in lake water-derived samples and not detected in well or brook-water derived samples. The highest level found was 8.6 ng/l.

Levels of triphenyl phosphate in drinking water samples from twelve Great Lakes municipalities were determined by Williams *et al.* (1982). Samples were collected over a

24-hour period during January 1980 and again in July/August 1980. Triphenyl phosphate was detected in 23 of 24 samples taken, at a concentration of 0.2-4.8 ng/l.

Lebel *et al.* (1981) reported that triphenyl phosphate was present at a concentration of 0.3-2.6 ng/l in twelve samples of drinking water collected in 1978 from six water treatment plants in Eastern Ontario, Canada.

Triphenyl phosphate in untreated waste water from Japan was reportedly reduced from an influent concentration of 0.054-2.12 μg/l to an effluent concentration of 0.005-0.082 μg/l during waste water treatment (Fukushima and Kawai 1986, cited in WHO 1991).

Boethling and Cooper (1985) reported that the concentration of triphenyl phosphate in influent and effluent at an industrial biological waste water treatment plant at an aryl phosphate manufacturing plant in the US was 740 μg/l and 7 μg/l respectively in the late 1970s. The same authors also found triphenyl phosphate at 0.3-1.2 μg/l in four samples of river water collected 13 km downstream from the outfall of the plant.

Boethling and Cooper (1985) reported the results of a later (early 1980s) survey of the levels of triphenyl phosphate in the United States. Triphenyl phosphate was found at 0.7 μg/l in one out of four samples from Saginaw River (industrialised area) but was not detected (detection limit 0.1 μg/l) in four samples from Baltimore Harbour (industrialised area), three samples from Detroit River (industrialised area), four samples from Delaware River (industrialised area near to aryl phosphate manufacturer), seven samples from Kanawha River (industrialised area near to aryl phosphate manufacturer) and four samples from Eastern Lake Superior (remote area).

A concentration of triphenyl phosphate in drinking water of 0.12 μg/l was reported by Hansen *et al.* (2000). Muir (1984) report that triphenyl phosphate was found in two out of ten city water (presumably drinking water) samples from the United States at concentrations of below 0.01-0.12 μg/l.

Galassi and Benfenati (2000) found triphenyl phosphate at a concentration of 15.2 μg/l in leachate from an industrial landfill. The leachate sample was filtered (0.45 μm) prior to analysis and so the concentration represents that in the dissolved phase.

Triphenyl phosphate was found in waste water samples (influent of the Barcelona primary treatment plant) but not seawater (collected 1.5 km offshore of the Besos river estuary) collected in 1987 (Valls *et al.* 1990). The detection limit of the method used was around 0.6 ng/l for waste water and 0.05 ng/l for seawater.

A survey of the levels of triphenyl phosphate in influent, effluent and sewage treatment sludge from municipal waste water treatment plants in Denmark was carried out by Miljøstyrelsen (2002a). For each sewage treatment plant, around four individual samples of influent or effluent were collected and the mean concentration of triphenyl phosphate determined, while the overall mean and 5th and 95th percentiles were calculated for the total population of waste water treatment plants using the mean value for each individual plant (in calculating the overall statistics, if concentrations of more than half the samples were above the limit of detection, the not-detected results were assumed to be half the detection limit; where more than half of all samples were below the detection limit, the overall mean was calculated only from results above the detection limit). The detection limit for triphenyl phosphate in both influent and effluent samples was 0.02 μg/l, and 50 μg/kg dry weight in the sewage sludge samples. In all, 45 influent and 45 effluent samples were analysed from twelve waste water treatment plants. In the influent samples, triphenyl phosphate was found at a concentration above the detection limit in 43 samples with the mean, 5th percentile and 95th percentile concentrations found to be 0.18, 0.03 and 0.29 μg/l respectively. In effluent samples, triphenyl phosphate was found at concentrations above the detection limit in 27 samples with the mean, 5th percentile and 95th percentile concentration of 0.04, 0.01 and 0.09 μg/l respectively. For the sewage sludge, a total of 15

samples were analysed, and triphenyl phosphate was found at concentrations above the detection limit in 12 of these. The mean, 5th percentile and 95th percentile concentrations found were 169, 86 and 335 μg/kg dry weight respectively.

In addition to the above, Miljøstyrelsen (2002a) also reported the concentration of triphenyl phosphate in effluent from an industrial plant producing wire and cable. In all, four samples of effluent were analysed and triphenyl phosphate was found to be present in all four samples. The mean and maximum concentration found was 0.05 and 0.08 μg/l respectively.

Triphenyl phosphate was found in snow taken from a reference site and from close to a road intersection in Sweden. Snow at two metres from the intersection had 70 ng/kg of triphenyl phosphate; levels at 100 and 250 m from the intersection and at the reference site were 5-10 ng/kg. The substance was also detected in snow at an airport in Sweden. In a sample from the aircraft parking areas, the level was about 960 ng/kg; in a sample from the runway, the level was about 750 ng/kg (Naturvårdsverket 2006).

Triphenyl phosphate was reported at a concentration of 0.05-0.7 μg/l in river water from Osaka, Japan (Kawai *et al.* 1978, cited in WHO 1991).

Ishikawa *et al.* (1985b) found triphenyl phosphate at a concentration of 0.013 to 0.031 μg/l in five out of sixteen river water samples from Kitakyushu City, Japan. Triphenyl phosphate was not detected in nine samples of seawater from the area. The detection limit of the method used was 0.010 μg/l. Similarly, Ishikawa *et al.* (1985c) found triphenyl phosphate in four out of fourteen river water samples at 0.013-0.031 μg/l from the same area (these may be the same samples reported in Ishikawa *et al.* 1985b).

Ishikawa *et al.* (1985c) found triphenyl phosphate in two out of six influent samples from five sewage treatment plants in Japan at a concentration of 90-100 ng/l. The substance was not found in effluent samples from the plants (detection limit 30 ng/l). The substance was also not detected in eight samples of domestic effluent (detection limit 30 ng/l) or the influent and effluent of a night soil treatment facility (the original paper is in Japanese and so it is not clear exactly what this refers to).

Ishikawa *et al.* (1985c) also analysed factory effluents for the presence of triphenyl phosphate. The detection limit of the analytical method was 30 ng/l, and triphenyl phosphate was not detected in effluents from three food factories, nine chemical factories, two steel factories, three metal factories, but was found at 200 ng/l in the effluent of one out of eight factories classified as other industries.

Triphenyl phosphate was not detected (detection limit 0.02 μg/l) in either river water or seawater from Tokyo (Wakabayashi 1980, cited in WHO 1991). Triphenyl phosphate was found in seawater at a concentration of 0.003 μg/l in a study by Sugiyama and Tanaka (1982, cited in WHO 1991).

Takahashi and Morita (1988) measured concentrations of triphenyl phosphate in raw water (river water) and finished drinking water from Japan. The detection limit of the method was 7 ppt (7 ng/l) and triphenyl phosphate was detected only occasionally in raw water samples (up to 25 ng/l) and finished water samples (up to 33 ng/l).

Further surveys of the levels of triphenyl phosphate in surface waters from all over Japan were carried out by Environment Agency Japan (1996). The substance was not detected in 100 samples analysed in 1975 (detection limit in the range 0.02-0.2 μg/l).

Fukushima *et al.* (1992) summarised the results of a long-term monitoring programme for triphenyl phosphate and tricresyl phosphate in the Yodo River basin, Lake Biwa, Yodo River and rivers in Osaka City, Japan. The programme started in 1976 and water samples were collected seasonally or semi-annually, with more recent samples from the Yamato River and Osaka bay collected during 1989 and 1990. Occurrences of both triphenyl

phosphate and tricresyl phosphate were generally limited to highly polluted areas and were more often associated with suspended sediments and sediments than the water phase. The concentrations found were generally below 0.5 μg/l.

Table 3.19 Summary of measured levels in water

Location	Comment	Measured level	Reference
Influent to waste water treatment plant	Primary treatment plant, Barcelona, 1987. Detection limit ~ 0.6 ng/l.	Detected.	Valls *et al.* 1990
Municipal waste water treatment plants, Denmark	Influent from 12 plants. Detection limit 0.02 μg/l.	Detected in 43 out of 45 samples - 0.18 μg/l mean.	Miljøstyrelsen 2002a
	Effluent from 12 plants. Detection limit 0.02 μg/l.	Detected in 27 out of 45 samples - 0.04 μg/l mean.	
Effluent from a wire and cable production plant, Denmark		Detected in all four samples – 0.05 μg/l mean 0.08 μg/l max.	Miljøstyrelsen 2002a
Waste water treatment plant effluent, Sweden		3 μg/l	Paxeus 1996, cited in OECD 2002
Giber Å river, Aarhus, Denmark.	Two 24-hour samples 1997-1998. Detection limit 0.2 μg/l.	Not detected.	Boutrup *et al.* 1998

Table 3.19 continued.

Location	Comment	Measured level	Reference
Møddebro bœk stream, Aarhus, Denmark.	Two 24-hour samples 1997-1998. Detection limit 0.2 μg/l.	Not detected.	Boutrup *et al.* 1998
Emscher canal, Germany		Up to 3.4 μg/l	Lenhart and Lemm 1993
River Ruhr, Germany	20 sites along length of river	Up to 0.28 μg/l	Lenhart and Lemm 1993
	Tributaries	Mean 0.4 μg/l	
Meuse at Eysden	Routine daily monitoring 1998. Detection limit 0.03 μg/l. Routine daily monitoring 1999. Detection limit 0.03 μg/l. Routine daily monitoring 2000. Detection limit 0.03 μg/l. Routine daily monitoring 2001. Detection limit 0.03 μg/l. Routine daily monitoring 2002. Detection limit 0.03 μg/l.	Detected in 19 samples at 0.03-0.15 μg/l. Yearly mean below 0.03 μg/l. Detected in 57 samples at 0.04-0.50 μg/l. Yearly mean below 0.03 μg/l. Detected in 45 samples at 0.03-0.22 μg/l. Yearly mean below 0.03 μg/l. Detected in 41 samples at 0.03-0.21 μg/l. Yearly mean below 0.03 μg/l. Detected in 5 out of 103 samples at 0.04-0.22 μg/l.	RIZA 2002
Landfill leachate	Industrial landfill.	15.2 μg/l	Galassi and Benfenati 2000
Aryl phosphate manufacturing plant, United States	Influent to waste water treatment plant.	740 μg/l	Boethling and Cooper 1985
	Effluent from waste water treatment plant.	7 μg/l	Boethling and Cooper 1985
	River water 13 km downstream of plant.	0.3-1.2 μg/l	Boethling and Cooper 1985
Night soil treatment facility, Japan	Detection limit 0.03 μg/l.	Influent – not detected. Effluent – not detected.	Ishikawa *et al.* 1985c
Factory effluent, Japan	Detection limit 0.03 μg/l. Samples included effluents from three food factories, nine chemical factories, two steel works, three metal factories and eight other industry. The substance was found only in the effluent from one other industry sample.	Detected in one out of 25 samples at 0.2 μg/l.	Ishikawa *et al.* 1985c
Waste water treatment plant, Japan		Influent 0.054-2.12 μg/l. Effluent 0.005-0.082 μg/l.	Fukushima and Kawai 1986, cited in WHO 1991

Table 3.19 continued.

Location	Comment	Measured level	Reference
Aryl phosphate manufacturing plant and large user of hydraulic fluids, United States	Detection limit 10 μg/l.	Not detected	Boethling and Cooper 1985
Delaware River, United States	Industrial area. Detected in 11/11 samples in August 1976.	0.1-0.4 μg/l	Sheldon and Hites 1978
	Industrial area. Detected in 2/5 samples in March 1977.	0.1-0.3 μg/l	Sheldon and Hites 1978
Water sources around Philadelphia, United States	Influent to industrial waste water treatment plant, August 1997.	16 μg/l	Sheldon and Hites 1979
	Effluent from industrial waste water treatment plant, August 1997.	2 μg/l	Sheldon and Hites 1979
	Delaware River, August 1997.	Trace-0.3 μg/l	Sheldon and Hites 1979
	Inflow to drinking water treatment plant, August 1997.	0.2 μg/l	Sheldon and Hites 1979
	Treated drinking water, August 1997	0.03 μg/l	Sheldon and Hites 1979
Industrial and non-industrial areas of United States	Samples collected during November 1977 and May 1978. Detection limit 0.1 μg/l.	Detected in 32 out of 63 samples at 0.1-7.9 μg/l. Geometric mean level 0.12 μg/l.	Mayer *et al*. 1981
Industrial areas of United States.	Water from Saginaw River, Baltimore Harbour, Detroit River, Delaware River, and Kanawha River. Detection limit 0.1 μg/l.	Detected in one out of 22 samples at 0.7 μg/l.	Boethling and Cooper 1985
Streams, United states	85 samples from 30 states, 1999-2000. Detected in 14 per cent (detection limit 0.1 μg/l).	0.04 μg/l median 0.22 μg/l max.	Kolpin *et al.* 2002
River water, Osaka, Japan	Detected in 11 out of 13 samples.	0.05-0.7 μg/l	Kawai *et al.* 1978, cited in WHO 1991
River water, Kitakyushu City, Japan	Detection limit 0.010 μg/l. Detected in five out of 16 samples.	0.013-0.031 μg/l	Ishikawa *et al.* 1985b
Seawater, Kitakyushu City, Japan	Detection limit 0.010 μg/l. Not detected in nine samples.	Not detected	Ishikawa *et al.* 1985b
River water, Tokyo, Japan	Detection limit 0.02 μg/l. Not detected in any of the 12 samples.	Not detected	Wakabayashi 1980, cited in WHO 1991

Table 3.19 continued.

Location	Comment	Measured level	Reference
Yodo River Basin, Lake Biwa, Yodo River and rivers in Osaka City, Japan	Long-term monitoring program.	Generally below 0.5 μg/l	Fukushima *et al.* 1992
Surface water, Japan	Detection limit 0.02-0.2 μg/l. Not detected in 100 samples collected in 1975.	Not detected	Environment Agency Japan 1996
Seawater, Tokyo, Japan	Detection limit 0.02 μg/. Not detected in 3/3 samples.	Not detected	Wakabayashi 1980, cited in WHO 1991
Seawater, Tokyo, Japan		0.003 μg/l	Sugiyama and Tanaka 1982, cited in WHO 1991
Seawater	1.5 km offshore of Besos river estuary, 1987. Detection limit ~ 0.05 ng/l.	Not detected	Valls *et al*. 1990
Eastern Lake Superior	Remote area. Detection limit 0.1 μg/l.	Not detected in four samples.	Boethling and Cooper 1985
Drinking water, Louisiana, United States	Detected in one out of three plants sampled.	0.12 μg/l	Boethling and Cooper 1985
River water and drinking water, Japan	Detection limit 0.007 μg/l. Detected only occasionally.	Raw water (river water) – up to 0.025 μg/l. Drinking water – up to 0.033 μg/l.	Takahashi and Morita 1988
Drinking water, Canada.	Samples from 29 municipalities. Detected in 35 out of 58 samples taken.	Not detected-0.0086 μg/l.	Williams and LeBel 1981
Drinking water, Canada.	Samples from 12 municipalities, Great Lakes area. Detected in 23/24 samples taken.	Not detected-0.0048 μg/l.	Williams *et al*. 1982
Drinking water, Canada.	Samples from six water treatment plants in Eastern Ontario. Detected in all 12 samples.	0.0003-0.0026 μg/l	Lebel *et al.* 1981
Drinking water		0.12 μg/l	Hansen *et al.* 2000
Drinking water, United States		0.01-0.12 μg/l	WHO 1991
City water (drinking water), United States	Detection limit 0.01 μg/l.	Detected in two out of 10 samples at below 0.01-0.12 μg/l.	Muir (1984)

Sediment

Levels of triphenyl phosphate in sediment are summarised in Table 3.20.

Triphenyl phosphate was not present in sediment samples (collected from the Besos river estuary or 4 km offshore the river mouth) collected in 1987 (Valls *et al.* 1990).

Mayer *et al.* (1981) determined the levels of triphenyl phosphate in sediment samples from thirteen locations in Midwestern United States and San Francisco Bay. Samples were from industrial and non-industrial locations and were collected during November 1977 to May 1978. Triphenyl phosphate was detected at levels above 0.01 mg/kg in 13 of the 40 samples analysed at a concentration of 0.01 to 4.0 mg/kg. The geometric mean of the levels found (using half of the detection limit (0.005 mg/kg) for samples below the limit of detection) was 0.11 mg/kg. It is not clear if these results refer to wet or dry sediment weights. The highest levels (1-4 mg/kg) were reported in the Saginaw River sampled 1.6 to 3.2 km downstream from several plants manufacturing automobile parts (WHO 1991).

Triphenyl phosphate was found in ten out of fifteen river sediment samples from Tokyo at a concentration of 0.7-3.3 μg/kg and in two out of three marine sediment samples from Tokyo at a concentration of 0.2-0.3 μg/kg (Wakabayashi 1980, cited WHO 1991).

Ishikawa *et al.* (1985b) found that triphenyl phosphate was not detected in six marine sediment samples from the Kitakyushu City area of Japan. The detection limit of the method used was 5 μg/kg.

Further surveys of the levels of triphenyl phosphate in sediments from all over Japan were carried out by Environment Agency Japan (1996). The substance was not detected in 100 samples analysed in 1975 (detection limit in the range 2 to 50 µg/kg dry weight).

Boethling and Cooper (1985) report the results of monitoring studies carried out in the late 1970s near an aryl phosphate production site in the United States. The substances included in the studies were triphenyl phosphate, tricresyl phosphate, isopropylphenyl diphenyl phosphate and aryl phosphates with molecular weights above 410 (which included trixylenyl phosphate and di-(isopropylphenyl) phenyl phosphate). The concentration of total aryl phosphates found in the sediment was 229 mg/kg at the outfall and 4.4 mg/kg at a location eight miles downstream from the outfall. A further 12 sediment samples were analysed and were found to contain total aryl phosphate concentrations of 0.07 to 1,032 mg/kg. As a result of these findings, a more comprehensive survey was undertaken. This found total aryl phosphate concentrations of 7 to 6,320 mg/kg at locations less than 100 yards downstream of the plant. Levels further downstream were much lower, but concentrations above 1 mg/kg were found in some samples ten miles downstream. The report indicates that the actual concentration present in the sediments could have been much, as the analytical recovery from spiked sediment was around six per cent. The mixed aryl phosphates with molecular weights above 452 were thought to be present at the highest concentrations and triphenyl phosphate was thought to be present at the lowest concentrations in these samples.

Boethling and Cooper (1985) reported the results of a later (early 1980s) survey of the levels of triphenyl phosphate in sediments from the United States. Triphenyl phosphate was not found (detection limit (0.03-0.2 mg/kg) in four samples from Saginaw River (industrialised area), three samples from Baltimore Harbour (industrialised area), two samples from Detroit River (industrialised area), two samples from Delaware River (industrialised area near aryl phosphate manufacturer), six samples from Kanawha River (industrialised area near aryl phosphate manufacturer) and two samples from Eastern Lake Superior (remote area).

Boutrup *et al.* (1998) determined the levels of triphenyl phosphate in freshwater and marine water sediments collected from the County of Aarhus, Denmark in 1997-1998. The

detection limit of the method was around 10 μg/kg dry weight and triphenyl phosphate was found in two out of five freshwater lake sediments at 10-11 μg/kg dry weight, one out of five freshwater river sediments at 8.9 μg/kg dry weight and was not detected in six marine sediments.

A further study of the levels of triphenyl phosphate in marine sediments from around Denmark was carried out by Glob (1998). The detection limit of the method was 10 μg/kg dry weight and triphenyl phosphate was not detected in 15 samples from the County of Southern Jutland, was detected in one out of 15 samples from the County of Vejle at 40 μg/kg dry weight and was detected in two out of 38 samples from the County of Fyn at 10-27 μg/kg dry weight.

Miljøstyrelsen (2002b) reported the results of further surveys on levels of triphenyl phosphate in marine sediments from around Denmark. These surveys found that triphenyl phosphate was not detected (detection limit 35 μg/kg dry weight) in twelve harbour sediments collected in 1999 or five harbour sediments collected in 2000 or in more open marine areas (Nibe Bredning, Kattegat, Øresund, Smålandsfarvandet and Mecklenburg Bugt). In addition, another survey found no triphenyl phosphate in harbour sediments (detection limit was again 35 μg/kg dry weight).

Comparison of measured levels with predicted levels

Monitoring data generally show elevated concentrations of triphenyl phosphate (up to a few μg/l in surface water and up to a few mg/kg in sediment) close to sources of release. Concentrations of this order of magnitude are consistent with the PECs calculated in this report. Many of the monitoring data showing elevated levels are from the United States in the late 1970s and so may not reflect the current situation in the EU. Nevertheless, more recent data from the EU shows that triphenyl phosphate is present in surface water and sediment, albeit at lower levels than calculated for production and use sites, although measurements cannot be related to activities.

Data on influent and effluent concentrations allow estimates to be made of the degree of removal in treatment plants, and these can be compared with the estimated value from SimpleTreat of 92 per cent. Removal rates estimated from measurements are:

- above 99 per cent (Boethling and Cooper 1985);
- 78 per cent (Miljøstyrelsen 2002a);
- 61 per cent to more than 90 per cent (Fukushima and Kawai 1986);
- 87.5 per cent (Sheldon and Hites 1978).

These values are not very different from the calculated value. In addition, the measured concentrations in production site effluent (Boethling and Cooper 1985) are of a similar order to those calculated.

As the calculated PECs appear to be reasonably consistent with the available monitoring data, these are used in the risk characterisation.

Table 3.20 Summary of levels of triphenyl phosphate in sediment

Location	Comment	Measured level	Reference
Freshwater sediment, Aarhus, Denmark	Collected in 1997-1998. Detection limit 0.010 mg/kg dry wt.	Detected in two of five lake samples at 0.010 to 0.011 mg/kg dry wt. Detected in one out of five river samples at 0.0089 mg/kg dry wt.	Boutrup *et al.* 1998
Besos River estuary and 4 km offshore	Samples collected in 1987.	Not detected.	Valls *et al.* 1990
Marine sediment, Aarhus, Denmark	Collected in 1997-1998. Detection limit 0.010 mg/kg dry wt.	Not detected in six samples.	Boutrup *et al.* 1998
Marine sediment, Southern Jutland, Denmark	Detection limit 0.010 mg/kg dry wt.	Not detected in 15 samples.	Glob 1998
Marine sediment, Vejle, Denmark	Detection limit 0.010 mg/kg dry wt.	Detected in one out of 15 samples at 0.040 mg/kg dry wt.	Glob 1998
Marine sediment, Fyn, Denmark	Detection limit 0.010 mg/kg dry wt.	Detected in two out of 38 samples at 0.010-0.027 mg/kg dry wt.	Glob 1998
Marine sediment, Denmark	Samples were taken in harbours and more open marine areas in 1999 or 2000. Detection limit 0.035 mg/kg dry wt.	Not detected.	Miljøstyrelsen 2002b
Marine samples from Tokyo, Japan		Detected in two out of three samples at 0.0002-0.0003 mg/kg.	Wakabayashi 1980, cited in WHO 1991
Marine samples, Kitakyushu City, Japan	Detection limit 0.005 mg/kg.	Not detected in six samples.	Ishikawa *et al.* 1985b
Industrial areas, United States	Samples collected in early 1980s from Saginaw River (4 samples), Baltimore Harbour (3 samples), Detroit River (2 samples), Delaware River (2 samples) and Kanawha River (6 samples). Detection limit 0.03-0.2 mg/kg.	Not detected in 17 samples.	Boethling and Cooper 1985
Midwestern United States and San Francisco Bay	Samples from industrial and non-industrial locations in 1977-1978. Detection limit 0.01 mg/kg.	Detected in 13 out of 40 samples at 0.01-4 mg/kg (geometric mean 0.11 mg/kg).	Mayer *et al.* 1981
Near to aryl phosphate production site, United States	Samples collected in the late 1970s. Results given as total triaryl phosphate.	Up to 6,320 mg/kg as total triaryl phosphate.	Boethling and Cooper 1985
Eastern Lake Superior, United States.	Remote area. Samples collected in early 1980s.	Not detected in two samples.	Boethling and Cooper 1985
Japan	Samples from all over Japan, 1975. Detection limit 0.002-0.050 mg/kg.	Not detected in 100 samples.	Environment Agency Japan 1996
River samples from Tokyo, Japan		Detected in 10 out of 15 samples at 0.0007-0.0033 mg/kg.	Wakabayashi 1980, cited in WHO 1991

3.3.2 Terrestrial compartment

Calculation of PECs

PECs for the soil compartment were estimated using EUSES 2.0.3 and are summarised in Table 3.21.

The estimated regional concentrations for the soil compartment are summarised below.

$PEC_{regional}$ = 7.71×10^{-5} mg/kg wet weight for agricultural soil
= 4.36×10^{-4} μg/l for pore water of agricultural soil
= 1.90×10^{-5} mg/kg wet weight for natural soil
= 5.07×10^{-3} mg/kg wet weight for industrial soil

Table 3.21 Summary of predicted local concentrations for the terrestrial compartment

Scenario		PEC_{local}			
		Annual average conc. in air (mg/m^3)	**Agricultural soil – 30 day average (mg/kg wet wt.)**	**Agricultural soil – 180 day average (mg/kg wet wt.)**	**Groundwater under agricultural soil (μg/l)**
Production of triphenyl phosphate		1.45×10^{-6} and 4.44×10^{-8}	negligible[a]	negligible[a]	negligible[a]
Printed circuit boards	Compounding	4.42×10^{-6}	1.58	1.34	7.57
	Conversion	4.42×10^{-6}	0.16	0.14	0.77
	Combined compounding and conversion	8.8×10^{-6}	1.74	1.47	8.35
Thermo-plastics/ styrenics	Compounding	8.23×10^{-5}	4.58	3.87	21.9
	Conversion	8.23×10^{-5}	0.47	0.39	2.23
	Combined compounding and conversion	1.67×10^{-4}	5.04	4.27	24.2
Thermosets and epoxy resins	Compounding	1.4×10^{-7}	1.58	1.34	7.57
	Conversion	3.48×10^{-5}	0.16	0.14	0.77
	Combined compounding and conversion	5.72×10^{-5}	1.74	1.47	8.35
Photo-graphic film	Compounding	2.88×10^{-5}	1.57	1.33	7.54
	Conversion	2.88×10^{-5}	0.16	0.14	0.78
	Combined compounding and conversion	5.72×10^{-5}	1.74	1.47	8.35

Notes: a) Sewage sludge from the production sites is not applied to land.

Measured levels

WHO (1991) detected triphenyl phosphate in agricultural soil collected from vineyards in Scotland, but no actual levels were reported. No other EU data are available.

Boethling and Cooper (1985) reported that triphenyl phosphate was not detected (detection limit 0.1 mg/kg) in soil samples collected near an aryl phosphate production site and a large user of hydraulic fluids in the United States.

Boethling and Cooper (1985) report the results of monitoring studies carried out in the late 1970s near an aryl phosphate production site in the United States. The substances included in the studies were triphenyl phosphate, tricresyl phosphate, isopropylphenyl diphenyl phosphate and aryl phosphates with molecular weights above 410 (which included trixylenyl phosphate and di-(isopropylphenyl) phenyl phosphate). The concentration of total aryl phosphates found in a soil sample collected from the plant yard was 26,550 mg/kg. This sample was collected in an area subject to frequent spills. The total aryl phosphate concentration found in river bank soil (collected from an area known to have received discarded soil from the plant yard) was 37 mg/kg.

Comparison of measured levels with predicted levels

Monitoring data indicate that triphenyl phosphate may be present in soil near sources of release; however, the relatively small database available precludes a meaningful comparison of monitoring data with calculated PECs for this compartment. Therefore, calculated PECs are used in the risk characterisation as a worst case.

3.3.3 Air compartment

Concentrations of triphenyl phosphate in air were estimated using EUSES 2.0.3. The PECs calculated are summarised in Table 3.21.

The predicted regional concentration in air is 4.44×10^{-8} mg/m^3.

Measured levels

Sjödin *et al.* (2001) investigated the levels of triphenyl phosphate in indoor and outdoor air at various locations in Sweden. Indoor air samples were taken from an electronics equipment recycling plant (samples taken on two working days at three locations in the dismantling hall and one close to the shredder), a printed circuit board manufacturing plant (samples taken on one working day at three locations within the plant), a computer repair facility (samples taken in one day at one location), from a computer teaching hall with 20 computers (samples taken one day at one location) and two offices equipped with two or three computers (samples taken in one day in two different offices). The outdoor air sample was taken from a suburban area close to Stockholm. Samples were collected using personal sampling equipment and were collected over a 500-minute period at a flow rate of 3.0 l/minute (corresponding to a total air sample of 1.5 m^3) or a 400-minute period at a flow rate of 9.0 l/minute (corresponding to a total air sample of 3.6 m^3). Duplicate samplers were used at each location investigated. Triphenyl phosphate was found in air samples from the electronics equipment recycling facility at 12-40 ng/m^3 (mean 19 ng/m^3) in the dismantling hall and 120-180 ng/m^3 in the shredder room. The triphenyl phosphate was found to be associated mainly with the particulate phase. No data were reported for levels of triphenyl phosphate in air from the other locations sampled.

Triphenyl phosphate was found in all dust samples taken from a range of indoor environments in Sweden (Naturvårdsverket 2006). These were homes, day care centres, radio shop, hotel, university lobby, library, aircraft, hospital wards, textile shop, prison office, public dance hall and cinema. Levels ranged from a few mg/kg to around 90 mg/kg.

Hansen *et al.* (2000) carried out a study to determine the concentration of triphenyl phosphate in air and dust in six schools and two kindergartens where building materials containing organophosphate chemicals (in particular tris(2-chloroethyl) phosphate) were thought to be used. The sampling was carried out in accordance with VDI 4300 B1.8 (dust) and VDI 4300 B1.1 (indoor air). The level of triphenyl phosphate in dust was below 1 to 220 mg/kg. The concentrations in indoor air were below 0.01 $\mu g/m^3$. The concentration of triphenyl phosphate in outdoor and indoor air from other buildings where contamination was not suspected was also found to be below 0.01 $\mu g/m^3$. The paper also reported concentrations of triphenyl phosphate in indoor air from other studies to be in the range 0.01-0.03 $\mu g/m^3$.

The levels of triphenyl phosphate in indoor air at three schools, a day care centre and an office building were investigated by Carlsson *et al.* (1997). In this study, four air samples were collected from one room in each building. The samples were collected overnight when the ventilation equipment in the rooms was switched off. Triphenyl phosphate was found to be present in all samples collected. The mean concentrations present were trace (below 0.5 ng/m^3) to 0.8 ng/m^3 in the school buildings, trace (below 0.5 ng/m^3) in the day care centre and 0.7 ng/m^3 in the office building. In addition to these indoor samples, the outdoor air was sampled at the same time outside the office building and outside one of the school buildings. The concentration present was found to be below the detection limit of the method used (1 ng/m^3). The triphenyl phosphate in air was thought to be associated mainly with the particulate phase.

Bergman (1997) analysed air from an office (7.5 m^2) containing a personal computer for the presence of nine organophosphate esters including triphenyl phosphate. Triphenyl phosphate was found to be present in the samples (thought to be mainly associated with the particulate matter) but no levels were reported. The source of the triphenyl phosphate was thought to be the computer as no triphenyl phosphate could be detected in air particulate samples in the absence of the computer and the air levels were higher in the presence of a new computer compared to those found in the presence of an older computer.

Yasuda (1980, cited in WHO 1991) determined the levels of triphenyl phosphate in air at various locations in Japan. The concentrations found were 0.5-1.4 ng/m^3 in samples from Seto Inland Sea (detected in three out of four samples collected) and 0.9-14.1 ng/m^3 in urban air of Matsuyama. It was not detected in air collected in agricultural areas (Dogo Plain and Ozu Basin).

Otake *et al.* (2001) determined the concentrations of triphenyl phosphate in indoor air in six houses from Tokyo. Each house was sampled over a three-day period and the concentrations found ranged from below 1.2×10^{-3} $\mu g/m^3$ in four houses to 0.01 $\mu g/m^3$ in two houses.

Boethling and Cooper (1985) reported that triphenyl phosphate was not detected (detection limit 2 $\mu g/m^3$) in air samples collected near an aryl phosphate production site and a large user of hydraulic fluids in the United States.

WHO (1991) report results from the United States National Institute for Occupational Safety and Health (NIOSH) indicating that triphenyl phosphate was present in all six samples of work place air at an automobile manufacturing plant in the United States where hydraulic fluids were used. The concentration found was 0.008 to 0.057 mg/m^3.

An old study by Sutton *et al.* (1960) found that triphenyl phosphate was present in 78 samples of work place air from a triphenyl phosphate manufacturing plant in the United States at a concentration of 0.5 to 29.6 mg/m^3.

Comparison of measured levels with predicted levels

Monitoring data indicate that concentrations of triphenyl phosphate in air in the environment are generally low. Elevated levels have been found in workplace air, and triphenyl phosphate has also been detected in indoor air; however, it is not possible to compare these data directly with the scenarios considered in this assessment.

Predicted concentrations are considered in the risk characterisation.

3.3.4 Non-compartment specific exposure relevant for the food chain

Predicted concentrations in biota and food

Predicted concentrations of triphenyl phosphate in fish and earthworms are shown in Table 3.22 and predicted concentrations in prey species for marine food chains are also included. Predicted concentrations in human food are shown in Table 3.23.

Table 3.22 Summary of predicted local concentrations for secondary poisoning

Scenario		Predicted concentration			
		Fish (mg/kg)	Earthworms (mg/kg)	Marine fish (mg/kg)	Marine top predators (mg/kg)
Production of triphenyl phosphate		0.06 and 7.6×10^{-3}	negligible[a]	n/a	n/a
Printed circuit boards	Compounding	0.14	1.81	0.16	0.03
	Conversion	0.02	0.19	0.02	3.63×10^{-3}
	Combined compounding and conversion	0.15	2.0	0.18	0.04
Thermo-plastics/ Styrenics	Compounding	2.5	5.25	3.02	0.60
	Conversion	0.26	0.53	0.31	0.06
	Combined compounding and conversion	2.76	5.78	3.32	0.67
Thermo-sets and epoxy resins	Compounding	7.4×10^{-3}	1.81	3.84×10^{-3}	1.07×10^{-3}
	Conversion	0.11	0.19	0.13	0.03
	Combined compounding and conversion	0.95	2.0	1.15	0.23
Photo-graphic film	Compounding	0.86	1.8	1.04	0.21
	Conversion	0.09	0.19	0.11	0.02
	Combined compounding and conversion	0.95	2.0	1.15	0.23

Notes: a) Sewage sludge from the production sites is not applied to land.
n/a – not applicable (no discharges from production to marine waters).

Measured levels in biota and food

Mayer *et al.* (1981) determined the levels of triphenyl phosphate in fish samples from thirteen locations in Midwestern United States and San Francisco Bay. Samples were

from collected from industrial and non-industrial locations during November 1977 to May 1978. The fish sampled included skipjack herring (*Alosa chrysochloris*), alewife (*Alosa pseudoharengus*), gizzard shad (*Dorosoma cepediamum*), coho salmon (*Oncorhynchus kisutch*), brown trout (*Salmo trutta*), lake trout (*Salvelinus namaycush*), rainbow smelt (*Osmerus mordax*), northern pike (*Esox lucius*), carp (*Cyprinus carpio*), common shiner (*Notropis cornutus*), spottail shiner (*Notropis hudsonius*), sand shiner (*Notropis stramineus*), quillback (*Carpiodes cyprinus*), black buffalo (*Ictiobus niger*), rehorse (*Moxostoma* sp.), blue catfish (*Ictalurus furcatus*), yellow bullhead (*Ictalurus natalis*), channel catfish (*Ictalurus punctatus*), flathead catfish (*Pylodictis olivaris*), brook silverside (*Labidesthes sicculus*), white bass (*Morone chrysops*), bluegill (*Lepomis macrochirus*), yellow perch (*Perca flavescens*), sauger (*Stizostedion canadense*) and freshwater drum (*Aplodinotus grunniens*). Triphenyl phosphate was detected in 16 of the 82 samples at a concentration of 0.1 to 0.6 mg/kg.

The concentration of triphenyl phosphate in dolphin (*Tursiops truncates*) blubber was determined (Kuehl and Haebler 1995, cited in OECD 2002). Samples were collected in 1990 from the Gulf of Mexico as a result of an unusual mortality event. Concentrations of triphenyl phosphate were 17 to 3,790 μg/kg lipid (mean 863 μg/kg lipid) in sucklings, 19 to 244 μg/kg lipid (mean 68 μg/kg lipid) in immature dolphins, 19 to 42 μg/kg lipid (mean 30 μg/kg lipid) in adult females and 15 to 142 μg/kg (mean 25-56 μg/kg lipid) in adult males (males were collected from several different locations). The conclusion in the OECD assessment was that although triphenyl phosphate is not persistent in the environment, an accumulation via the food chain should be assumed.

Gilbert *et al.* (1986) surveyed total trialkyl and triaryl phosphates, including triphenyl phosphate, in composite total diet samples (of 15 commodity food types) representing an average adult diet for eight regions of the UK. The mean total dietary intake of total organic phosphates was estimated to be 0.072 to 0.105 mg/day. In general, the highest concentrations of total phosphate esters (total triaryl and trialkyl) were in offal and nuts (these food groups have only a low relative importance in diet). Triphenyl phosphate was found to occur mainly in poultry, meat products and, to a lesser extent, vegetables.

Total diet studies carried out in the United States between April 1982 and April 1984 indicated that the mean total daily intake of triphenyl phosphate was 0.3 ng/kg body weight in infants, 4.4 ng/kg body weight for toddlers, 1.2-1.6 ng/kg body weight for 14 to 16 year olds and 0.5-1.6 ng/kg body weight for adults (Gunderson 1988).

Triphenyl phosphate was reported to be present at a concentration of 2-6 μg/kg in samples of fish and shellfish from Seto Inland Sea, Japan (Kenmotsu *et al*. 1981, cited in WHO 1991). The substance was detected in 12 out of 41 samples analysed.

Further surveys of triphenyl phosphate in fish from all over Japan were carried out by Environment Agency Japan (1996). The substance was not detected in any of the 100 samples analysed in 1975 (detection limit was in the range five to 50 μg/kg wet weight).

Lombardo and Egry (1979) found triphenyl phosphate at a concentration of 60 to 150 μg/kg in the edible portions of fish (two carp and one goldfish) from an area downstream of a user of hydraulic fluids in the United States.

Comparison of measured levels with predicted levels

Relatively few data are available on actual levels of triphenyl phosphate in biota. A US study from the late 1970s found triphenyl phosphate in fish near sources of release at up to 0.6 mg/kg. This concentration is comparable with many of the concentrations predicted for local sources. In addition, triphenyl phosphate has been reported in some marine mammals and has also been found in various food items, particularly meat products. Predicted concentrations are considered in this risk characterisation.

Table 3.23 Summary of predicted local concentrations in food for human consumption

Scenario		Concentration							
		Fish (mg/kg)	Root crops (mg/kg)	Leaf crops (mg/kg)	Drinking water (mg/l)	Meat (mg/kg)	Milk (mg/kg)	Air (mg/m^3)	Total daily human intake (mg/kg bw/day)
Production of triphenyl phosphate		0.12 0.01	4.68×10^{-5} 3.86×10^{-5}	2.37×10^{-3} 7.24×10^{-5}	1.43×10^{-4} 1.28×10^{-5}	1.8×10^{-4} 6.01×10^{-6}	5.69×10^{-5} 1.9×10^{-6}	1.41×10^{-6}	2.43×10^{-4} 1.96×10^{-5}
Printed circuit boards	Compounding Conversion Combined compounding and conversion	0.27 0.03 0.30	2.71 0.28 2.99	8.32×10^{-3} 7.33×10^{-3} 0.02	7.57×10^{-3} 7.73×10^{-4} 8.35×10^{-3}	1.27×10^{-3} 5.99×10^{-4} 1.86×10^{-3}	4.01×10^{-4} 1.89×10^{-4} 5.88×10^{-4}	4.38×10^{-6} 4.38×10^{-6} 8.76×10^{-6}	0.02 1.72×10^{-3} 0.02
Thermo-plastics/ styrenics	Compounding Conversion Combined compounding and conversion	5.0 0.51 5.51	7.87 0.8 8.67	0.14 0.14 0.28	0.02 2.23×10^{-3} 0.02	0.01 9.95×10^{-3} 0.02	3.76×10^{-3} 3.15×10^{-3} 6.99×10^{-3}	8.23×10^{-5} 8.23×10^{-5} 1.67×10^{-4}	0.05 7.68×10^{-3} 0.06
Thermosets and epoxy resins	Compounding Conversion Combined compounding and conversion	0.01 0.22 1.9	2.71 0.28 2.99	1.33×10^{-3} 0.06 0.09	7.57×10^{-3} 7.73×10^{-4} 8.35×10^{-3}	7.6×10^{-4} 4.19×10^{-3} 7.58×10^{-3}	2.4×10^{-4} 1.33×10^{-3} 2.4×10^{-3}	9.52×10^{-8} 3.48×10^{-5} 5.71×10^{-5}	0.02 2.92×10^{-3} 0.02
Photographic film	Compounding Conversion Combined compounding and conversion	1.72 0.18 1.9	2.7 0.28 2.99	0.05 0.05 0.09	7.54×10^{-3} 7.79×10^{-4} 8.35×10^{-3}	4.15×10^{-3} 3.49×10^{-3} 7.58×10^{-3}	1.31×10^{-3} 1.1×10^{-3} 2.4×10^{-3}	2.88×10^{-5} 2.88×10^{-5} 5.71×10^{-5}	0.02 2.69×10^{-3} 0.02
Regional sources		4.52×10^{-3}	1.56×10^{-4}	7.24×10^{-5}	5.38×10^{-6}	5.61×10^{-6}	1.77×10^{-6}	4.44×10^{-8}	9.73×10^{-6}

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4 Effects assessment: Hazard identification and dose (concentration) – response (effect) assessment

4.1 Aquatic compartment

The following sections review the available toxicity data for triphenyl phosphate with aquatic organisms. Where possible, a validity marking is given for each study (this appears in the summary tables within each section). The following validity markings have been used:

1. **Valid without restriction.** The test is carried out to internationally recognised protocols (or equivalent protocols) and all or most of the important experimental details are available.
2. **Use with care.** The test is carried out to internationally recognised protocols (or equivalent protocols) but some important experimental details are missing, or the method used, or endpoint studied, in the test means that interpretation of the results is not straightforward.
3. **Not valid.** There is a clear deficiency in the test that means the results cannot be considered valid.
4. **Not assignable.** Insufficient detail is available on the method used to allow a decision to be made on the validity of the study.

In terms of the risk assessment, toxicity data assigned a validity marking of one or two are considered of acceptable quality when deriving the PNEC.

A small number of the tests are unpublished studies carried out by industry. It has not been possible to validate all of these tests within the scope of this report and these are assigned a validity marking of four unless it is clear that some aspects of the test invalidate the results (for these a validity marking of three is given). The studies given a validity marking of four have been considered alongside studies assigned a validity marking of one and two when deriving the PNEC.

One important property when considering the aquatic toxicity data is water solubility. The water solubility of triphenyl phosphate is 1.9 mg/l. Several studies have been carried out at concentrations greater than this water solubility and, although this in itself does not necessarily invalidate the test (for example, cosolvents or solubility aids could have been used to aid dispersion of the substance in the test media), this introduces some uncertainty in the concentration to which the organisms were actually exposed in the test. In cases where it is clear that undissolved test substance was present in the test media, the tests have been marked as not valid.

4.1.1 Toxicity to fish

Short-term studies

Freshwater fish

The short-term toxicity of triphenyl phosphate to freshwater fish is given in Table 4.1.

The acute toxicity of triphenyl phosphate (purity not given) was studied with killifish (*Oryzias latipes*) and goldfish (*Carassius auratus*) (Sasaki *et al.* 1981). The experiment was carried out using a static method at 25°C without aeration. The 96-hour LC_{50}s determined were 1.2 mg/l for killifish and 0.7 mg/l for goldfish. Deformation of the spine (a characteristic manifestation of toxicity for some organophosphorus compounds) was seen in fish exposed to concentrations close to the LC_{50}.

Palawski *et al.* (1983) investigated the effects of acute exposure to triphenyl phosphate (purity 99 per cent) on the survival and condition of rainbow trout (*Oncorhynchus mykiss*). In the test, 100 fish per treatment (fry at 12 days past the swim-up stage) were exposed to the test substance in 30-litre tanks for 96 hours using a static exposure system. The concentrations tested were 0.21, 0.24 and 0.29 mg/l and were selected based on the known 96-hour EC_{31}, 96-hour EC_{50} and 96-hour EC_{69} values for the substance determined in previous studies (the effects investigated previously with rainbow trout included mortality, immobility and loss of equilibrium of fish). In addition, standard acute toxicity tests using ten fish per treatment in three-litre jars were carried out simultaneously to determine the 96-hour EC_{50} and 96-hour LC_{50} with the particular strain of fish used in this experiment. The 96-hour LC_{50} determined in the test was 0.36 mg/l and the 96-hour EC_{50} was determined to be 0.30 mg/l.

After the 96-hour exposure period, up to 40 surviving fish per treatment were removed from the exposure tanks and were maintained in clean, flowing water for 30 days. During this observation period, the fish were fed three times per day with salmon starter and brine shrimp nauplii. At day seven of the observation period, the number of fish per treatment was reduced to 30 to prevent overcrowding. After 30 days, growth of the fish (weight and total length) and presence of physical abnormalities were determined. No significant increase in mortality was seen in the 30-day observation period and fish that were immobilised during the 96-hour exposure period were found to regain their locomotor activity in the first two days of the observation period. Around 12 per cent of the survivors exposed to 0.29 mg/l of the test substance showed spinal curvatures, possibly as a result of vertebral damage in the caudal area. No statistically significant effects were seen on growth of the fish during the observation period.

Sinks and Schultz (2001) and Admans *et al.*, (2001) reported a 96-hour LC_{50} of 0.88 mg/l for triphenyl phosphate with the fathead minnow (*Pimephales promelas*). The test used a flow-through system and the test was carried out as part of the United States Environmental Protection Agency's fathead minnow acute mortality database.

The acute toxicity of triphenyl phosphate (no information on purity) to bluegill sunfish (*Lepomis macrochirus*) was determined by Dawson *et al.* (1977). The test system used was a static system and the report indicates that gentle aeration was applied whenever the dissolved oxygen was depleted during the test (no information is given as to whether or not this occurred with the test with triphenyl phosphate). The 96-hour LC_{50} determined was 290 mg/l.

Table 4.1 Short-term toxicity of triphenyl phosphate to freshwater fish

Species	Test guide-line	Number of animals per treat-ment	Age/ size	Co-solvent	Concs. tested	N or M	Test conditions						End-point	Control resp.	Effect conc.	Ref.	Val.
							Media	Temp.	Hard.	pH	Static/ flow	D.O.					
Carassius auratus		7-9 in 7 litres of water	0.8-2.8 g	No		N	Tap water	25°C			Static		Mortality		96h-LC_{50} = 0.70 mg/l	Sasaki *et al.* 1981	2
Ictalurus punctatus	USEPA 1975		0.23 g					22°C	38	7.5	Static		Mortality		96h-LC_{50} = 0.42 mg/l	Mayer and Ellersieck 1986	2
Lepomis macrochirus		Loading <1 g/l.		Direct addition of the test sub-stance	125, 180, 320, 420 and 560 mg/l plus control.	N	Well water	23°C	55	7.6-7.9	Static		Mortality	1.3% Mortality overall	96h-LC_{50} = 290 mg/l	Dawson *et al.* 1977	3
	USEPA 1975	10 in 15 litres of water	0.5-1.0 g	Acetone, <100 µg/l.	0.5, 1.0, 2.5, 5.0 and 10 mg/l plus control	N	Well water	22°C	73	7.6	Static		Mortality		96h-LC_{50} = 0.78 mg/l	Huckins *et al.* 1991	2
Leuciscus idus													Mortality		96h-LC_{50} >5 mg/l	Bayer 2002	4
Oncorhyn-chus mykiss	ASTM 1980	10 in 3 litres of water	0.11 g			N	Well water	12°C	272	7.5	Static		Mortality	0% Mortality	96h-LC_{50} = 0.36 mg/l	Palawski *et al.* 1983	2
													Immobil. mortality	0% Immobil. mortality	96h-EC_{50} = 0.30 mg/l	Palawski *et al.* 1983	2
	USEPA 1975					N					Static		Mortality		96h-LC_{50} = 0.40 mg/l	Mayer *et al.* 1981	2
	USEPA 1975		0.6 g					12°C	40	7.4	Static		Mortality		96h-LC_{50} = 0.37 mg/l	Mayer and Ellersieck 1986	2

Table 4.1 continued.

Species	Test guide-line	Number of animals / treat-ment	Age/ size	Co-solvent	Concs. tested	N or M	Test conditions						End-point	Control resp.	Effect conc.	Ref.	Val.
							Media	Temp.	Hard.	pH	Static/ flow	D.O.					
Oncorhyn-chus mykiss (continued)	OECD 203	10	0.94 g	Octanol, Tween 20 and ethylene -glycol methyl ether	Control and solvent control	N	Tap water	15°C	172	7.8-8.1	Static	5.4-6.7	Mortality		96h-LC_{50} = 0.85 mg/l	IUCLID 2000, OECD 2002	2
	USEPA 1975	10 per treat-ment in 15 litres	Fing-erling (45 day)	Acetone	0.180, 0.240, 0.320, 0.420, 0.56, 1.00 mg/l plus control and solvent control	N	Recon water	12°C	40-48	7.0-7.2	Static		Mortality		96h-LC_{50} = 0.32mg/l	Sitthich-aikasem 1978	2
													Hemorrh -agic areas		96h-EC_{50} = 0.30 mg/l	Sitthich-aikasem 1978	2
		30 per treat-ment in 22 litres	Fing-erling (45 day)	Acetone	0.055, 0.090, 0.125, 0.160, 0.240, 0.310 and 0.450 mg/l plus control and solvent control. Highest conc. measured and used to estimate lower ones.	M	Well water	12°C	295-305	7.4-7.5	Flow	7.3-8.5 mg/l	Mortality		96h-LC_{50} >0.45 mg/l	Sitthich-aikasem 1978	2
													Hemorrh -agic areas		96h-EC_{50} = 0.26 mg/l	Sitthich-aikasem 1978	2
	USEPA 1975	10 per treat-ment in 3 litres	Sac fry (10 day)	Acetone	0.180, 0.240, 0.320, 0.420, 0.560 and 1.00 mg/l plus control and solvent control	N	Recon water	12°C	40-48	7.0-7.2	Static		Mortality		96h-LC_{50} = 0.31 mg/l	Sitthich-aikasem 1978	2
													Hemorrh -agic areas		96h-EC_{50} = 0.31 mg/l	Sitthich-aikasem 1978	2

Table 4.1 continued.

Species	Test guide-line	Number of animals / treat-ment	Age/ size	Co-solvent	Concs. tested	N or M	Test conditions						End-point	Control resp.	Effect conc.	Ref.	Val.
							Media	Temp.	Hard.	pH	Static/ flow	D.O.					
Oncorhyn-chus mykiss (continued)	USEPA 1975	15 per treat-ment in 10 litres	Sac fry (10 day)(10 day)	Acetone	0.055, 0.090, 0.125, 0.160, 0.240, 0.310 and 0.450 mg/l plus control and solvent control. Highest conc. measured and used to estimate lower ones	M	Well water	12°C	295-305	7.4-7.5	Flow	7.3-8.5 mg/l	Mortality		96h-LC_{50} = >0.45 mg/l	Sitthich-aikasem 1978	2
													Hemorrh-agic areas		96h-EC_{50} = 0.24 mg/l	Sitthich-aikasem 1978	2
Oryzias latipes		7-9 in 1 litre of water	0.1-0.2 g	No		N	Tap water	25°C			Static		Mortality		96h-LC_{50} = 1.2 mg/l	Sasaki *et al.* 1981	2
Pimephales promelas	USEPA	25	30 day								Flow		Mortality		96h-LC_{50} = 0.88 mg/l	Sinks and Schultz 2001	2
																Admans *et al*. 2001	
	USEPA 1975					N					Static		Mortality		96h-LC_{50} = 0.66 mg/l	Mayer *et al.* 1981	2
	USEPA 1975		1 g					22°C	44	7.3	Static		Mortality		96h-LC_{50} = 1.0 mg/l	Mayer and Ellersieck 1986	2

Notes:
N = Nominal concentration.
M = Measured concentration.
Temp. = Temperature.
Hard. = Water hardness (given as mg $CaCO_3$/l).
D.O. = Dissolved oxygen (given as mg O_2/l or per cent saturation).
Val. = Validity rating (see Section 4.1): 1) Valid without restriction; 2) Use with care; 3) Not valid; 4) Not assignable.

Concentrations tested in this study are much greater than the water solubility of the substance, and the substance appears to have been added directly to the test vessel. It is therefore possible that the effects seen in this test were indirect effects caused by undissolved test substance rather than a direct toxic effect of the substance itself and so the test is considered invalid.

A further study on the acute toxicity of triphenyl phosphate (purity 99 per cent) to bluegill sunfish was carried out by Huckins *et al.* (1991). The 96-hour EC_{50} value determined in this study was 0.78 mg/l. The triphenyl phosphate was added to the test medium as a solution in acetone (acetone concentration below 100 μg/l). Experiments were carried out to investigate actual dissolution of the triphenyl phosphate in the test medium. This showed that the concentration in water was lower than expected over the initial 4-hour period of the experiment. This was thought to be a result of the low volume of solvent used, creating a small initial mixing zone for the triphenyl phosphate in the test medium. The local solubility of the triphenyl phosphate may therefore have been exceeded and a true solution obtained after the initial 4-hour period, as the triphenyl phosphate subsequently dispersed/dissolved throughout the medium. The addition of 1 g/l of soil or clay was found to reduce the nominal toxicity of the substance due to adsorption onto the solid phase.

Mayer *et al.* (1981) determined the acute toxicity of triphenyl phosphate (no information on purity) to rainbow trout (*Oncorhynchus mykiss*) and fathead minnows (*Pimephales promelas*) using a static test system. The 96-hour LC_{50}s determined were 0.4 mg/l for *O. mykiss* and 0.66 mg/l for *P. promelas*.

Bayer (2002) reported a 96-hour LC_{50} of above 5 mg/l for a commercial triphenyl phosphate (Disflamoll TP) in an unpublished study with golden orfe (*Leuciscus idus*).

Sitthichaikasem (1978) carried out acute toxicity tests using triphenyl phosphate with rainbow trout (*Oncorhynchus mykiss*) fingerlings and sac fry. Two series of experiments were carried out, the first using a static test system and the second using a flow-through test system. In the first series, the 96-hour LC_{50} values determined for triphenyl phosphate were 0.31 mg/l for sac fry and 0.32 mg/l for fingerlings. In the second series, the 96-hour LC_{50} was reported to be above 0.450 mg/l for both sac fry and fingerlings. The reason for the difference in toxicity between the two series was unclear, but could be due to the generally poorer water conditions (in terms of dissolved oxygen concentration and so on) inherent in a static test system compared a flow-through system. However, other parameters such as water type (soft reconstituted water was used in the static test and hard well water in the flow-through test) could also have resulted in some differences. The study also determined EC_{50} values based on the appearance of hemorrhagic areas around the gills and behavioural effects. In the first (static) series of experiments, the 96-hour EC_{50} values for these effects were 0.31 mg/l for sac fry and 0.30 mg/l for fingerlings. In the second (flow-through) series of experiments, the 96-hour EC_{50} values were similar at 0.24 mg/l for sac fry and 0.26 mg/l for fingerlings. Attempts were made to monitor the actual concentrations present in the flow-through test, but there were analytical difficulties in measuring the lower concentrations. The measured concentration in the highest exposure group was 0.45 mg/l compared with a nominal concentration of 0.42 mg/l, and so this measured concentration was used to estimate the actual lower exposure concentrations using the known dilutions used. Thus, the results of the flow-through test are based on estimated concentrations rather than true measured values.

The results of a further unpublished industry study of the toxicity of triphenyl phosphate to rainbow trout (*Oncorhynchus mykiss*) is given in IUCLID (2000) and OECD (2002). The test was carried out according to the OECD 203 guideline and the 96-hour LC_{50} was reported to be 0.85 mg/l.

Mayer and Ellersieck (1986) report 96-hour LC_{50} values for triphenyl phosphate of 0.37 mg/l with rainbow trout (*Oncorhynchus mykiss*), 0.42 mg/l with channel catfish (*Ictalurus*

punctatus) and 1.0 mg/l with fathead minnow (*Pimephales promelas*). These data are from unpublished studies in the United States and the authors of the report consider the data to be valid.

A fish 96-hour LC_{50} and a 14-day LC_{50} of 1.9 and 2.2 mg/l respectively can be estimated for triphenyl phosphate from the log K_{ow} value of 4.63 using the USEPA ECOSAR (version 0.99h) software.

Using the methods given in the TGD, a 96-hour LC_{50} of 0.94 mg/l can be estimated using the equation for polar narcosis (recommended for esters) and a log K_{ow} of 4.63. This is in reasonable agreement with the available data.

Solomon *et al.* (1999) investigated the effect of exposure to triphenyl phosphate on brain acetyl cholinesterase (AChE) activity in mosquito fish (*Gambusia affinis*). The triphenyl phosphate used in the test had a purity of above 99 per cent. In the test, fish (0.2-0.26 g; 20-30 fish per treatment) were exposed to the test substance at a concentration of 0.2 mg/l in 20 litres of aerated tap water at 23°C for 96 hours in a static system, followed by a 192-hour recovery period in clean water. The concentration tested was the highest concentration that did not cause fish mortality in the test system. AChE activity in the exposed fish was determined (by measuring the rate of hydrolysis of acetylthiocholine) at various times during the experiment (six fish were analysed at 24, 48 and 96 hours of exposure and 96 and 192 hours of recovery) and compared to control fish. AChE activity in the fish exposed to triphenyl phosphate was found to be significantly reduced ($p=0.05$) compared to controls. This inhibition was higher at 24 hours of exposure (34 per cent inhibition) than at 96 hours of exposure (11 per cent inhibition). At the end of the 192-hour recovery period, AChE activity in the exposed fish showed an increase of 21 per cent compared with the control value.

A similar study carried out by Solomon *et al.* (2000) found that a concentration of 0.2 mg/l of triphenyl phosphate resulted in a time-dependent decrease in hepatic glutathione S-transferase (GST) activity compared with control populations in both mosquito fish (*Gambusia affinis*) (22 per cent inhibition) and topmouth gudgeon (*Pseudorasobora parva*) (51 per cent inhibition) exposed over 96 hours.

Marine

The short-term toxicity of triphenyl phosphate to marine fish is given in Table 4.2.

The acute toxicity of triphenyl phosphate (no information on purity) to tidewater silverside (*Menidia beryllina*) was determined by Dawson *et al.* (1977). The test system used was a static system and continuous aeration was applied during the test. The 96-hour LC_{50} determined was 95 mg/l. Concentrations tested in this study are much greater than the water solubility of the substance, and the substance appears to have been added directly to the test vessel. It is therefore possible that the effects seen in this test were indirect effects caused by undissolved test substance rather than a direct toxic effect of the substance itself and so the test is considered invalid.

Mayer *et al.* (1981) determined the acute toxicity of triphenyl phosphate (no information on purity) to sheepshead minnows (*Cyprinodon variegatus*) using a static test system. The 96-hour LC_{50} was between 0.32 and 0.56 mg/l.

Table 4.2 Short-term toxicity of triphenyl phosphate to marine fish

Species	Test guide-line	Number of animals/ treatment	Age/ size	Co-solvent	Concs. tested	N or M	Test conditions						End-point	Control resp.	Effect conc.	Ref.	Val.
							Media	Temp.	Sal.	pH	Static/ flow	D.O.					
Cyprinodon variegates	USEPA 1975					N					Static		Mortality		96h-LC_{50} >0.32 <0.56 mg/l	Mayer *et al.* 1981	2
Menidia beryllina		Loading <1g/l.	40-100 mm	Direct addition of test sub-stance.	75, 100 180, 320 and 560 mg/l plus control.	N	Artificial seawater	20°C			Static		Mortality	3% Mortality overall.	96h-LC_{50} = 95 mg/l.	Dawson *et al.* 1977	3

Notes: N = Nominal concentration.
M = Measured concentration.
Temp. = Temperature.
Sal. = Water salinity (given as parts per thousand (‰)).
D.O. = Dissolved oxygen (given as mg O_2/l or per cent saturation).
Val. = Validity rating (see Section 4.1): 1) Valid without restriction; 2) Use with care; 3) Not valid; 4) Not assignable.

Long-term studies

The long-term toxicity of triphenyl phosphate to freshwater fish is shown in Table 4.3.

The long-term toxicity of triphenyl phosphate (no information on purity) to rainbow trout (*Oncorhynchus mykiss*) and fathead minnow (*Pimephales promelas*) was investigated by Mayer *et al.* (1981).

The study with *O. mykiss* was a 90-day partial lifecycle study investigating the growth, survival and bone development in sac fry (Mayer *et al.* 1981). The test was carried out using a flow-through system and, in total, seven concentrations of triphenyl phosphate were tested (dilution factor between concentrations was 0.75). A cosolvent was used in the experiment at concentrations up to 0.05 ml/l and a solvent control was also run. Water samples were analysed at two-weekly intervals and these showed that the actual exposure concentrations were around 62 per cent of the nominal value during the experiment and so the results were expressed in terms of the mean measured concentration. The mortality and behaviour of the fish were recorded daily during the test, and the growth (weight and length) was determined after 15, 30, 45, 60 and 90 days. At the end of the study, the collagen, calcium and phosphorus contents of backbones from ten fish in each exposure group were determined, and a pathological examination of the eyes (cataracts) was also carried out. No statistically significant (p=0.05) effects compared with the control population on survival, growth, cataracts and vertebral collagen were seen at any concentration tested (the highest concentration being 1.4 µg/l).

The test with *P. promelas* was a standard 30-day embryo-larval study using a flow-through system. In addition to survival, growth and hatchability, the effects of triphenyl phosphate exposure on eyes were also determined. The measured concentrations of triphenyl phosphate during the study averaged around 39 per cent of the nominal value and so the results were expressed in terms of the mean measured concentrations. No statistically significant (p=0.05) effects on egg hatchability, growth or eyes were found in any of the treatment groups when compared with the control group and so the no observed effect concentration (NOEC) for these endpoints was above 230 µg/l (the highest concentration tested). However, the survival of the fry was significantly reduced at 230 µg/l compared with the control group and the NOEC for survival was 87 µg/l.

The same results as above, expressed as maximal acceptable toxicant concentrations (MATCs) (this is the geometric mean of the no observed effect concentration (NOEC) and lowest observed effect concentration (LOEC)) are reported in Mayer *et al.* (1986).

Long-term toxicity tests with triphenyl phosphate using rainbow trout (*Oncorhynchus mykiss*) sac fry and fingerlings were carried out by Sitthichaikasem (1978), using a flow-through system over 30 days. The LC_{50} was found to decrease with increasing exposure time and the asymptotic LC_{50} for triphenyl phosphate was determined to be 0.26 mg/l for sac fry and 0.24 mg/l for fingerlings. The study also investigated the growth of the fish (mean length and mean weight). Growth of the sac fry was found to be significantly (p=0.05) reduced (by both length and weight) compared with the control population at all exposure concentrations (concentrations were 0.055, 0.090, 0.125, 0.160, 0.240 and 0.310 mg/l). Therefore, the NOEC for this endpoint was below 0.055 mg/l (an EC_{10} of 0.037 mg/l has since been calculated for this endpoint from the raw data (OECD 2002)). The growth of the fingerlings was found to be statistically significantly reduced compared to controls at 0.09 mg/l (based on length) or 0.125 mg/l (based on weight). The NOEC for growth of fingerlings was therefore 0.055 mg/l.

Table 4.3 Long-term toxicity of triphenyl phopshate to freshwater fish

Species	Test guide-line	Number of animals / treat-ment	Age/ size	Co-solvent	Concs. tested	N or M	Test conditions						End-point	Control resp.	Effect conc.	Ref.	Val.
							Media	T.	Hard.	pH	Static/ flow	D.O.					
Oncorhyn-chus mykiss			Egg fry	Yes at up to 0.05 ml/l	0.22, 0.38, 0.44, 0.64, 0.91, 1.2, 1.4 µg/l plus solvent control	M	Well water	12° C	272	7.2	Flow		Mortality		90d-NOEC ≥1.4 µg/l	Mayer *et al.* 1981	2
													Growth		90d-NOEC ≥1.4 µg/l		
													Cataracts		90d-NOEC ≥1.4 µg/l		
													Verte-bral collagen		90d-NOEC ≥1.4 µg/l		
	USEPA 1975	30	Sac fry (10 day old)	Acetone	0.055, 0.090, 0.125, 0.16, 0.24, 0.31 and 0.45 mg/l plus control and solvent control (highest was measured and used to estimate lower concentrations)	M	Well water	12° C	295-305 mg/l	7.4-7.5	Flow	7.3-8.5	Growth	Mean length 37.9 mm; mean weight = 0.525 g	30d-LOEC = 0.055 mg/l 30d-EC_{10} = 0.037 mg/l	Sitthich-aikasem 1978, OECD 2002	2
													Survival	83% survival	30d-LC_{50} = 0.26 mg/l		
			Finger-ling (45 day old)	Acetone	0.055, 0.090, 0.125, 0.16, 0.24, 0.31 and 0.45 mg/l plus control and solvent control (highest was measured and used to estimate lower concs.)	M	Well water	12° C	295-305 mg/l	7.4-7.5	Flow	7.3-8.5	Growth	Mean length 59.2 mm; mean weight 2.150 g	30d-NOEC = 0.055 mg/l	Sitthich-aikasem 1978	2
													Survival	83% survival	30d-LC_{50} = 0.24 mg/l		

Table 4.3 continued.

Species	Test guide-line	Number of animals / treat-ment	Age/ size	Co-solvent	Concs. tested	N or M	Test conditions						End-point	Control response	Effect conc.	Ref.	Val.
							Media	T.	Hard.	pH	Static/ flow	D.O.					
Oncorhyn-chus mykiss (continued)	Feeding study												Growth		16-32 week LOEC = 1,000 mg/kg food	Muir 1984	4
													Mortality		16-32 week LD_{50} = 10,000 mg/kg food		
Pimephales promelas			Emb-ryo-larval		2.8, 12, 36, 87 and 230 μg/l plus control	M					Flow		Mortality		30d-NOEC = 87 μg/l	Mayer *et al.* 1981	2
													Growth		30d-NOEC ≥230 μg/l		
													Hatch.		30d-NOEC ≥230 μg/l		
													Catar-acts		30d-NOEC ≥230 μg/l		

Notes: N = Nominal concentration.
M = Measured concentration.
T. = Temperature.
Hard. = Water hardness (given as mg $CaCO_3$/l).
D.O. = Dissolved oxygen (given as mg O_2/l or per cent saturation).
Val. = Validity rating (see Section 4.1): 1) Valid without restriction; 2) Use with care; 3) Not valid; 4) Not assignable.

Muir (1984) reports the results of unpublished feeding studies exposing rainbow trout (*Oncorhynchus mykiss*) to triphenyl phosphate via the diet for 16 to 32 weeks. The concentrations used were in the range of 1 to 10,000 mg/kg food and it was reported that fish showed reductions in growth rate at 1,000 mg/kg food and 50 per cent mortality at 10,000 mg/kg food.

A long-term no effect concentration of 0.126 mg/l is estimated using the USEPA ECOSAR program (v0.99h).

There are no long-term toxicity data available for triphenyl phosphate with marine fish.

4.1.2 Toxicity to aquatic invertebrates

Short-term studies

Freshwater

The short-term toxicity of triphenyl phosphate to freshwater aquatic invertebrates is summarised in Table 4.4.

Ziegenfuss *et al.* (1986) determined the acute toxicity of triphenyl phosphate (no information on purity) to both the daphnid *Daphnia magna* and the midge *Chironomus tentans.* The test method was based on ASTM (1980). The 48-hour EC_{50} values determined were 1.0 mg/l for *D. magna* and 1.6 mg/l for *C. tentans.*

A similar 48-hour EC_{50} of 1 mg/l for *Daphnia magna* was determined by Mayer *et al.* (1981) for triphenyl phosphate (no information on purity) using a static system.

A 48-hour EC_{50} value of 1.05 mg/l is estimated using the USEPA ECOSAR program (v0.99h).

The acute toxicity of triphenyl phosphate (purity 99 per cent) to scud (*Gammarus pseudolimnaeus*) and midge larvae (*Chironomus riparius*) was investigated by Huckins *et al.* (1991). For *G. pseudolimnaeus*, the 96-hour EC_{50} value was determined to be 0.25 mg/l and the 48-hour EC_{50} was determined to be 0.36 mg/l for *C. riparius*. The triphenyl phosphate was added to the test medium as a solution in acetone (acetone concentration below 100 μg/l). Experiments were carried out to investigate the actual dissolution of the triphenyl phosphate in the test medium. This showed that the concentration in water was lower than expected over the initial four-hour period of the experiment. This was thought to be as a result of the low volume of solvent used, creating a small initial mixing zone for the triphenyl phosphate in the test medium. The local solubility of the triphenyl phosphate was therefore likely to be exceeded and a true solution obtained after the initial four-hour period, as the triphenyl phosphate subsequently dispersed/dissolved throughout the medium. The addition of 1.0 g/l of montmorillonite clay to the test solution resulted in an approximate five-fold decrease in the nominal toxicity of triphenyl phosphate to the species tested.

The toxicity of triphenyl phosphate (purity 98 per cent) to the golden apple snail (*Pomacea canaliculata*) was investigated by Lo and Hsieh (2000). The organisms used in the test were 35 to 40 days old from a third generation of a laboratory-reared population (the original population was collected from the wild). The 72-hour LC_{50} for triphenyl phosphate was determined to be 38.2 μg/ml (38.2 mg/l). The concentrations used in this test (10-250 mg/l), and the derived LC_{50}, are all above the water solubility of triphenyl phosphate and so the results of this test are uncertain.

Table 4.4 Short-term toxicity of triphenyl phosphate to freshwater invertebrates

Species	Test guide-line	Number of animals/ treatment	Age/ size	Co-solvent	Concs. tested	N or M	Test conditions						End-point	Control resp.	Effect conc.	Ref.	Val.
							Media	Temp.	Hard.	pH	Static/ flow	D.O.					
Chironomus tentans	ASTM 1980		2nd instar				Well water				Static		Immobil. mortality		48h-EC_{50} = 1.6 mg/l	Ziegen-fuss *et al.* 1986	2
Chironomus riparius	USEPA 1975	10 per replicate in 150 ml water	4th instar	Acetone	0.06, 0.10, 0.18, 0.32, 0.56 and 1.0 mg/l plus control	N	Well water	22°C	73	7.6	Static		Immobil. mortality		48h-EC_{50} = 0.36 mg/l	Huckins *et al.* 1991	2
Daphnia magna	ASTM 1980		<24 h				Well water				Static		Immobil. mortality		48h-EC_{50} = 1.0 mg/l	Ziegen-fuss *et al.* 1986	2
	USEPA 1975					N							Immobil. mortality		48h-EC_{50} = 1.0 mg/l	Mayer *et al.* 1981	2
	USEPA 1975	Five per replicate, four replicates		Yes			Recon water	20°C		8.6	Static	7.03 - 7.19	Immobil. mortality		48h-EC_{50} = 1.35 mg/l	IUCLID 2001	1
Gammarus pseudolim-naeus	USEPA 1975	Two replicates per treatment in four litres of water	60-90 day old	Acetone	0.010, 0.018, 0.032, 0.056, 0.10, 0.18, 0.32 and 1.0 mg/l plus control	N	Well Water	17°C	73	7.6	Static		Immobil. mortality		96h-EC_{50} = 0.25 mg/l	Huckins *et al.* 1991	2
Pomacea canaliculata		30/replicate in 450 ml of water, three replicates/ treatment	35-40 day		Six concentrations between 10 and 250 mg/l		Well water	26°C		7.5	Static		Mortality		72h-LC_{50} = 38.2 mg/l	Lo and Hsieh 2000	3

Notes: N = Nominal concentration.
M = Measured concentration.
Temp. = Temperature.
Hard. = Water hardness (given as mg $CaCO_3$/l).
D.O. = Dissolved oxygen (given as mg O_2/l or per cent saturation).
Val. = Validity rating (see Section 4.1): 1) Valid without restriction; 2) Use with care; 3) Not valid; 4) Not assignable.

IUCLID (2000) report the results of an unpublished industry study into the toxicity of triphenyl phosphate to *Daphnia magna.* The 48-hour EC_{50} was found to be 1.35 mg/l.

Using the methods given in the TGD, a 48-hour EC_{50} of 1.4 mg/l can be estimated for *Daphnia magna* using the equation for polar narcosis (recommended for esters) and a log K_{ow} of 4.63. This is in reasonable agreement with the data available. The USEPA ECOSAR program (v0.99h) predicts a value of 1.05 mg/l for the same endpoint.

Marine

The short-term toxicity of triphenyl phosphate to marine invertebrates is summarised in Table 4.5.

Mayer *et al.* (1981) determined the toxicity of triphenyl phosphate (no information on purity) to mysid shrimp (*Mysidopsis bahia*) using a static test system. The 96-hour LC_{50} determined was between 0.18 mg/l and 0.32 mg/l.

Long-term studies

No long-term toxicity data appear to be available for triphenyl phosphate with freshwater or marine invertebrates.

4.1.3 Toxicity to algae

Freshwater

The toxicity of triphenyl phosphate to fresh water algae is summarised in Table 4.6.

Millington *et al.* (1988) investigated the toxicity of triphenyl phosphate to alga using various growth media. The tests were all carried out according to OECD Guideline 201 with *Selenastrum capricornutum*[7], *Scenedesmus subspicatus* and *Chlorella vulgaris*. The growth media used included the standard OECD media, Bold's basal media (which has a higher nitrogen and phosphorus content compared with other media) and a standard USEPA media. Acetone was used as a cosolvent in the test (concentration below 100 μl/l) and all tests were carried out in triplicate. The 72-hour LOECs (lowest concentration causing a significant (p=0.05) reduction in total biomass (area under the growth curve)) were 0.5 to 5 mg/l for *S. capricornutum* and *S. subspicatus* and 5 mg/l for *C. vulgaris.* The report indicates that similar results were obtained when the results were analysed in terms of growth rates.

Mayer *et al.* (1981) determined the toxicity of triphenyl phosphate (no information on purity) to *Selenastrum capricornutum* using a method recommended by the United States Environmental Protection Agency (USEPA 1971). The 96-hour EC_{50} was determined to be 2 mg/l.

[7] Now *Pseudokirchneriella subcapitata.*

Table 4.5 Short-term toxicity of triphenyl phosphate to marine invertebrates

Species	Test guide-line	Number of animals / treat-ment	Age/ size	Co-solvent	Concs. tested	N or M	Test conditions						End-point	Control resp.	Effect conc.	Ref.	Val.
							Media	Temp.	Sal.	pH	Static/ flow	D.O.					
Mysodopsis bahia	USEPA 1975					N					Static		Mortality		96h-LC_{50} = >0.18 <0.32 mg/l	Mayer *et al.* 1981	2
Crangon Crangon		20	0.57g 36mm	Acetone	0.2, 0.5, 1.0, 2.0 mg/l	M	Sea	14.5-15.7	35	7.8-8.1	Semi-static	7.0-7.6	Mortality		96h-LC_{50} =0.304 mg/l	IUCLID 2001	2

Notes: N = Nominal concentration.
M = Measured concentration.
Temp. = Temperature.
Sal. = Water salinity (given as parts per thousand (‰)).
D.O. = Dissolved oxygen (given as mg O_2/l or per cent saturation).
Val. = Validity rating (see Section 4.1): 1) Valid without restriction; 2) Use with care; 3) Not valid; 4) Not assignable.

Table 4.6 Toxicity of triphenyl phosphate to freshwater algae

Species	Test guide-line	Initial inoculum conc.	Cosolvent	Concs. tested	N or M	Test conditions				Endpoint	Control response	Effect conc.	Reference	Val.
						Media	Temp.	Hard.	pH					
Anabaena flosaquae			Acetone at ≤0.05%	0.1, 1.0 and 5.0 mg/l plus solvent control.	N	BG-11 medium	20°C			Nitrogenase activity (acetylene reduction technique)	0.62 nmole/hour/ 10^5 cells	4h-EC_{16} = 0.1 mg/l 4h-EC_{23} = 1.0 mg/l 4h-EC_{32} = 5.0 mg/l	Wong and Chau 1984	2
Ankistro-desmus falcatus		4.7×10^4 cells/ml	Acetone at ≤0.05%.	Solvent control and dark control ran.	N	CHU-10 medium	20°C			Primary production (uptake of ^{14}C)	Uptake in dark control was <5% of total seen in solvent control.	4h-IC_{50} = 0.26 mg/l	Wong and Chau 1984	2
		1.4×10^4 cells/ml	Acetone at ≤0.05%	0.05, 0.10, 0.50, 1, 3 and 5 mg/l plus solvent control.	N	CHU-10 medium	20°C			Growth rate and biomass.	Control response given graphically.	72h-NOEC = 0.10 mg/l 22d-NOEC = 0.10 mg/l	Wong and Chau 1984	2
Chlorella vulgaris	OECD 201	1×10^4 cells/ml	Acetone at ≤100µl/l	0.05, 0.1, 0.5, 1.0 and 5.0 mg/l plus control/solvent control.	N	Bold's basal	22°C			Biomass and growth rate	Control response given graphically (ca. 1×10^6 cells/ml at 72 hours)	72h-LOEC = 5 mg/l	Millington *et al.* 1988	2
						OECD	22°C			Biomass and growth rate	Control response given graphically (ca. 1×10^6 cells/ml at 72 hours)	72h-LOEC = 5 mg/l		
						USEPA	22°C			Biomass and growth rate	Control response given graphically (ca. $1\text{-}2\times10^5$ cells/ml at 72 hours)	72h-LOEC = 5 mg/l		

Table 4.6 continued.

Species	Test guide-line	Initial inoculum conc.	Cosolvent	Concs. tested	N or M	Test conditions				Endpoint	Control response	Effect conc.	Reference	Val.
						Media	Temp.	Hard.	pH					
Scenedesmus quadricauda		4.7×10^4 cells/ml	Acetone at ≤0.05%.	Solvent control and dark control ran.	N	CHU-10 medium	20°C			Primary production (uptake of ^{14}C)	Uptake in dark control was <5% of total seen in solvent control.	4h-IC_{50} = 0.50 mg/l	Wong and Chau 1984	2
Scenedesmus subspicatus	OECD 201	1×10^4 cells/ml	Acetone at ≤100 µl/l	0.05, 0.1, 0.5, 1.0 and 0.5 mg/l plus control/solvent control.	N	Bold's basal	22°C			Biomass and growth rate	Control response given graphically (ca. $7\text{-}8\times10^5$ cells/ml at 72 hours)	72h-LOEC = 0.5 mg/l	Millington *et al.* 1988	2
						OECD	22°C			Biomass and growth rate	Control response given graphically (ca. $3\text{-}4\times10^5$ cells/ml at 72 hours)	72h-LOEC = 5.0 mg/l		
						USEPA	22°C			Biomass and growth rate	Control response given graphically (ca. $3\text{-}4\times10^5$ cells/ml at 72 hours)	72h-LOEC = 1.0 mg/l		

Table 4.6 continued.

Species	Test guide-line	Initial inoculum conc.	Cosolvent	Concs. tested	N or M	Test conditions				Endpoint	Control response	Effect conc.	Reference	Val.
						Media	Temp.	Hard.	pH					
Selenastrum capricornutum [a]	OECD 201	1×10^4 cells/ml	Acetone at ≤100 µl/l	0.05, 0.1, 0.5, 1.0 and 0.5 mg/l plus control/ solvent control.	N	Bold's basal	22°C			Biomass and growth rate	Control response given graphically (ca. 8-9×10^5 cells/ml at 72 hours)	72h-LOEC = 0.5 mg/l	Millington *et al.* 1988	2
						OECD	22°C			Biomass and growth rate	Control response given graphically (ca. 8-9×10^5 cells/ml at 72 hours)	72h-LOEC = 5.0 mg/l		
						USEPA	22°C			Biomass and growth rate	Control response given graphically (ca. 3-4×10^5 cells/ml at 72 hours)	72h-LOEC = 1.0 mg/l		
	USEPA 1971											96h-EC_{50} = 2.0 mg/l	Mayer *et al.* 1981	2
Natural algal community			Acetone at ≤0.05%.	Solvent control and dark control also ran.	N	CHU-10 medium	20°C			Primary production (uptake of ^{14}C)	Uptake in dark control was <5% of total seen in solvent control.	4h-IC_{50} = 0.20 mg/l	Wong and Chau 1984	2

Notes: N = Nominal concentration.
M = Measured concentration.
Temp. = Temperature.
Hard. = Water hardness (given as mg $CaCO_3$/l).
Val. = Validity rating (see Section 4 1): 1) Valid without restriction; 2) Use with care; 3) Not valid; 4) Not assignable.
a = now *Pseudokirchneriella subcapitata*.

Wong and Chau (1984) carried out several studies to investigate the toxicity of triphenyl phosphate (no information on purity) to algae. The first series of experiments investigated the effects on the primary production (as measured by ^{14}C-uptake from ^{14}C-carbonate) in cultures of *Scenedesmus quadricauda* and *Ankistrodesmus falcatus* over a four-hour period. The tests were carried out by inoculating 13.9 ml of growth medium with 1 ml of algal cell culture (7×10^5 cells per ml giving an initial inoculum concentration of 4.7×10^4 cells per ml in the test solution; the algal cells were in the logarithmic growth phase) and incubating for 24 hours with triphenyl phosphate. The triphenyl phosphate was added as a solution in acetone (final acetone concentration in the test solution was below 0.05 per cent and an acetone control was also run at this concentration). After the initial 24-hour incubation, 0.1 ml of a sodium ^{14}C-carbonate solution was added and the solution was incubated for a further four hours. At the end of four hours, the amount of radioactivity taken up by the cells (corrected for the uptake in dark controls) was determined and the concentration causing a 50 per cent reduction in primary production (IC_{50}) was determined to be 0.26 mg/l for *A. falcatus* and 0.50 mg/l for *S. quadricauda*. A similar experiment using natural phytoplankton from Lake Ontario yielded a 4-hour IC_{50} of 0.2 mg/l.

Wong and Chau (1984) also determined the toxicity of triphenyl phosphate to *A. falcatus* in an algal growth study. Cells used in the test were in the logarithmic growth phase (initial inoculum concentration was 1.4×10^4 cells per ml) and effects on the total biomass and growth rate were determined at regular time intervals over 22 days. Triphenyl phosphate concentrations of 0.05 mg/l and 0.10 mg/l had no effect on either the total biomass or growth rate at any time point investigated (in fact, these levels appeared to stimulate algal growth). The total biomass and growth rate was found to be reduced compared with the control at 0.5 mg/l and concentrations of 1 mg/l and above completely inhibited algal growth. The NOEC from this study is thus 0.1 mg/l.

A further test by Wong and Chau (1984) investigated the effect of exposure to triphenyl phosphate on nitrogenase activity in *Anabaena flos-aquae.* The test was carried out by placing 9 ml of a cell culture (the report indicates that the number of cells in this culture was 1×10 cells per ml, but this may be a typing error in the manuscript) in 1 ml of growth medium containing the triphenyl phosphate. The vial was then sealed and ten per cent of the air volume was replaced by acetylene. Cultures were incubated for four hours at 20°C, following which nitrogenase activity was determined by measuring the amounts of acetylene and ethylene present in the gas phase (acetylene reduction technique). Each determination was carried out in triplicate. The amount of ethylene produced was 0.62 ± 0.01 nmol/hour/10^5 cells in the control cultures and 0.52 ± 0.4, 0.47 ± 0.01 and 0.42 ± 0.02 nmol/hour/10^5 cells in the 0.1, 1.0 and 5.0 mg/l triphenyl phosphate treatments. These values correspond to 84, 77 and 68 per cent respectively of the control value. Pre-incubation of the algal cultures with triphenyl phosphate for 24 hours prior to addition of the acetylene lead to essentially the same results.

The USEPA ECOSAR program (v0.99h) predicts a 96-hour EC_{50} of 0.17 mg/l, and a long-term no effect concentration of 0.13 mg/l, for green algae.

No toxicity data appear to be available for triphenyl phosphate with marine algae.

4.1.4 Microorganisms

The toxicity of triphenyl phosphate to the ciliated protozoan *Tetrahymena pyriformis* was studied in a 40-hour growth assay (Sinks and Schultz 2001). The purity of the triphenyl phosphate used was above 95 per cent and a stock solution of the substance was prepared in dimethyl sulphoxide. The test was carried out using a static exposure system where growth was measured spectrophotometrically at 540 nm. Six to eight concentrations were tested and each concentration was tested in duplicate. Two controls (one with ciliates and one without ciliates) were also run. The test was considered valid if

the control-absorbency value was between 0.60 and 0.90. The concentration of triphenyl phosphate found to cause 50 per cent growth inhibition in the test system was 5.05 mg/l.

Eto *et al.* (1975) investigated the effects of triphenyl phosphate (no information on purity) on spore germination in the fungus *Aspergillus niger.* Spore suspensions (around 2.5×10^5 cells per ml) in growth medium were exposed to triphenyl phosphate (possibly added in acetone solvent) and incubated at 30°C for 24 hours. The triphenyl phosphate was tested at a concentration of 5×10^{-5} M, 5×10^{-4} M and 5×10^{-3} M (16, 163 and 1,631 mg/l). None of the concentrations tested reduced spore germination compared with the control. In addition, the effect of oxygen uptake by resting suspensions of *A. niger* was determined by the Warburg technique over a 30-minute period. A triphenyl phosphate concentration of 163 mg/l resulted in a nine per cent inhibition of respiration compared with the control population.

OECD (2002) reports the results of unpublished industry tests using triphenyl phosphate with *Escherichia coli* and *Pseudomonas fluorescens*. The 24-hour EC_0s were reported to be 200 mg/l for each bacterium. No further details are available.

4.1.5 Toxicity to sediment organisms

There are no single species tests on sediment organisms available at the present time.

Fairchild *et al.* (1987) investigated the effects of both uncontaminated sediment and sediment contaminated with triphenyl phosphate (purity 98 per cent) on the structure and function of an experimental headwater stream ecosystem. The test was carried out using three experimental streams. Each stream was 50 m in length and consisted of three sections (10 m long, 1 m wide and 0.1 m deep) each separated by a pool (10 m long, 2 m wide and 0.3 m deep). The streams had a 15-cm depth of river gravel. Well water was delivered to the stream via a weir at a rate of 20 l/s and half of the delivered water volume (10 l/s) was recirculated to the inflow. The streams were shaded from May to October to simulate a deciduous forest canopy, and a total of 27.2 kg dry weight of leaves (*Acer saccharum*) were distributed in each stream at a rate of 100 g/m^2 per week during November (three weeks) and March (one week) to simulate the natural inputs of organic matter.

The sediment used in the study had an organic carbon content of 0.7 per cent and consisted of five per cent sand, 77 per cent silt and 18 per cent clay. Two of the streams were treated with either uncontaminated sediment or triphenyl phosphate-contaminated sediment from the end of March. The third stream acted as a control. Each treatment consisted of 250 kg of sediment applied at the head of the second and third sections of the river (a total of 500 kg per stream) once per week for six weeks. Sediments were added during the first half of a simulated four-hour flood event where the water flow was increased to 40 l/s. Subsequently the streams received water at a low-flow rate of 10 l/s. The concentration of triphenyl phosphate in the sediment was increased each week, starting at 50 mg/kg and doubling each week until a maximum of 1,600 mg/kg was reached after six weeks.

The half-life for triphenyl phosphate on the sediment was determined to be 1.7 days. Concentrations of triphenyl phosphate in the water phase reached up to 2,000 μg/l immediately after adding the highest sediment concentration. The concentration in the outflow of the system (measured 96 hours after the treatments) was generally in the range 0.4-2.4 μg/l, and triphenyl phosphate was still present in the outflow water three weeks after the last addition (but could not be detected twelve weeks after the last addition). The interstitial water concentrations in the sediment reached 11-33 μg/l by the last addition, and triphenyl phosphate was still present at 0.1-0.5 μg/l in samples twelve weeks after the last addition.

Neither the clean nor the contaminated sediment treatments were found to affect the pH (7.31-8.04), alkalinity (213-256 mg/l as $CaCO_3$) and conductivity (340-590 μmho cm^{-1}) of the water in the streams compared with the control stream. No treatment-related effects were seen on the dynamics of leaf decomposition, periphyton biomass (as determined by chlorophyll a content of algae), total number of benthic invertebrates, number of benthic invertebrate species, diversity of benthic community structure or pattern of insect emergence in the treated streams compared with the control stream. In addition, no mortalities were apparent in caged bluegill sunfish (*Lepomis macrochirus*) exposed for 96 hours each week. However, both the clean sediment and the triphenyl phosphate-contaminated sediment were found to alter the patterns of drift dynamics of benthic invertebrates, increase the nutrient retention of the stream, decrease the percentage similarity of benthic invertebrates and decrease the algal drift of the system compared with the control stream. The results of this test are difficult to interpret in terms of the effects caused by triphenyl phosphate, as similar effects were seen with the clean and contaminated sediment.

4.1.6 Predicted no effect concentration (PNEC) for the aquatic compartment

Surface water

Acute toxicity data are available for fish, invertebrates and algae. The lowest results from standard tests are a 96-hour LC_{50} of 0.31 mg/l for fish, a 96-hour EC_{50} of 0.25 mg/l for *Gammarus pseudolimnaeus* and 26-hour IC_{50}s based on ^{14}C uptake of 0.2-0.5 mg/l and greater for algae. The predicted acute toxicity values are of the same order.

Long-term data are available for fish and algae. The lowest value obtained is an EC_{10} of 0.037 mg/l for growth of rainbow trout sac fry over 30 days. In addition, a 30-day NOEC of 0.087 was obtained in a standard embryo-larval study with fat head minnow. For algae, a NOEC from an algal growth study can be considered as a long-term result (as it is from a multigenerational study). The lowest NOEC available for algae is around 0.1 mg/l after 72 hours exposure. No long-term data are available for invertebrates. The predicted values for algae and for fish are similar to the measured values.

Annex B considers the available long-term invertebrate data for triaryl phosphates as a whole. The following regression equation was obtained between the log (NOEC for invertebrates (mole/l)) and the log (EPI estimated K_{ow}) (note, the correlation was poor):

$$\log(\text{measured NOEC}) = -0.2279 \times \log(\text{EPI estimated } K_{ow}) - 5.9317.$$

Based on the analysis of data given in Annex B, the NOEC for triphenyl phosphate with invertebrates is estimated to be around 0.032 mg/l. This value is similar to that obtained with fish.

The predicted no effect concentration (PNEC) is derived based on the available long-term data. There are no clear differences in sensitivity between fish, invertebrates and algae in the limited acute toxicity data available. Two NOECs are available for fish and algae; hence, an assessment factor of 50 is applied to the EC_{10} of 0.037 mg/l for fish, giving a $PNEC_{water}$ of 0.74 μg/l.

An alternative approach to deriving the $PNEC_{water}$ for triphenyl phosphate is to make use of the estimated NOEC of 0.032 mg/l for invertebrates, treating this as a third NOEC value. Here a factor of 10 is used, leading to a $PNEC_{water}$ of 3.2 μg/l.

Both of these PNECs are considered in the risk characterisation.

Limited data are available on marine species, and they do not show any clear difference in sensitivity from the freshwater results. A PNEC of 0.074 µg/l can be calculated using the long-term freshwater data and an assessment factor of 500 according to the TGD.

Microorganisms

The available toxicity data for microorganisms include an IC_{50} of 5.05 mg/l for the ciliated protozoan *Tetrahymena pyriformis*, an EC_9 of 163 mg/l for respiration inhibition in the fungus *Aspergillus niger* and EC_0s of 200 mg/l for the bacterium *Escherichia coli* and *Pseudomonas fluorescens.*

Based on these results, the protozoan *Tetrahymena pyriformis* appears to be the most sensitive species tested. According to the TGD, an IC_{50} from this type of study can be used to derive a $PNEC_{microorganisms}$ for sewage treatment processes using an assessment factor of 10. Thus, the $PNEC_{microorganisms}$ is determined as 0.51 mg/l.

A NOEC of 100 mg/l could be inferred from the positive results in a MITI I test as described in Section 3.1.1. This would give a PNEC of 10 mg/l. This is considered in the risk characterisation section.

Sediment

No sediment toxicity data are available suitable for determining a PNEC for triphenyl phosphate. In the absence of data, the equilibrium partitioning method can be used to estimate the PNEC:

$$\mathrm{PNEC}_{sed} = \frac{\mathrm{K}_{\mathrm{susp-water}}}{\mathrm{RHO}_{\mathrm{susp}}} \times \mathrm{PNEC}_{water} \times 1000$$

where $K_{susp\text{-}water}$ = suspended sediment-water partition coefficient = 251 m^3/m^3 (see Section 3.1.2)

RHO_{susp} = bulk density of suspended sediment = 1,150 kg/m^3

Using a $PNEC_{water}$ of 0.74 µg/l, the $PNEC_{sed}$ is estimated to be 0.16 mg/kg wet weight. The PNEC for marine sediments is derived in the same way as the marine water PNEC, and is 0.016 mg/kg wet weight. An additional factor of ten is applied to the PEC/PNEC ratios.

4.2 Terrestrial compartment

4.2.1 Toxicity to insects

Eto *et al.* (1975) determined the toxicity of triphenyl phosphate to an organophosphate-resistant strain of green rice leafhoppers (*Nephotettix cincticeps*) and an organophosphate-resistant strain of smaller brown planthoppers (*Laodelphax striatellus*). The tests with *N. cincticeps* were carried out by topical application of triphenyl phosphate as a solution in acetone to four- to five-day-old adult females. The treated females were then kept on rice seedlings at 25°C and the mortality after 24 hours was determined. The tests with *L. striatellus* were carried out with fifth instar larvae using a contact method. The LD_{50} determined for triphenyl phosphate with *N. cincticeps* was 4,600 mg/kg and the LD_{50} determined for *L. striatellus* was 570 µg/tube. The results of this test are not suitable for derivation of a PNEC.

4.2.2 Predicted no effect concentration (PNEC) for the terrestrial compartment

No terrestrial toxicity data are available suitable for determining a PNEC for triphenyl phosphate. In the absence of data, the equilibrium partitioning method can be used to estimate the PNEC.

$$\mathrm{PNEC}_{soil} = \frac{\mathrm{K}_{\mathrm{soil-water}}}{\mathrm{RHO}_{\mathrm{soil}}} \times \mathrm{PNEC}_{water} \times 1000$$

where $K_{soil\text{-}water}$ = soil-water partition coefficient = 300 m^3/m^3 (see Section 3.1.2).
RHO_{soil} = bulk density of wet soil = 1,700 kg/m^3.

Using a $PNEC_{water}$ of 0.74 μg/l, the $PNEC_{soil}$ is estimated to be 0.13 mg/kg wet weight. An additional factor of ten is applied to PEC/PNEC ratios in view of the high sorption coefficient to organic carbon and consequent potential for uptake via the solid phase.

4.3 Atmosphere

No information is available on the toxicity of triphenyl phosphate to plants and other organisms exposed via air. The low vapour pressure of the substance means that volatilisation to the atmosphere is likely to be limited and the resulting concentrations are likely to be low. The possibility of triphenyl phosphate contributing to atmospheric effects such as global warming and acid rain is thus likely to be small. In addition, as the substance does not contain halogen atoms, it will not contribute to ozone depletion.

4.4 Mammalian toxicity

4.4.1 Toxicokinetics, metabolism and distribution

There are no valid *in vivo* data on the absorption, distribution and elimination of triphenol phosphate in mammals, including humans. There is only one valid *in vitro* study (Sasaki *et al*. 1984, cited in OECD 2002, 2005) in the literature regarding metabolism of triphenyl phosphate. In this, homogenates of the microsomal and soluble fractions of Wistar rat livers were incubated with triphenyl phosphate (0.0004M) with and without nicotinamide adenine dinucleotide phosphate (NADPH) and major metabolites were characterised using gas chromatography; diphenyl phosphate was the only major metabolite found. Metabolism by the microsomal fraction occurred in the absence of NADPH, and it was concluded that arylesterase in the microsomes contributes to triphenyl phosphate metabolism. The metabolic reactions were inhibited almost completely by SKF-525A and carbon monoxide in the absence of NADPH whereas potassium cyanate (KCN) , sodium azide (NaN_3), dipyridyl and ethylene diamine tetracetic acid (EDTA) showed little effect, suggesting that the mixed function oxidase system in the microsomes plays a central role in metabolism. Diphenyl phosphate was not further metabolized by the microsomes. Overall, it was concluded that triphenyl phosphate is metabolised by hydrolysis (in rat liver homogenate) to the major metabolite diphenyl phosphate.

4.4.2 Acute toxicity

Oral

Several acute oral lethality studies cover various species, but these are generally conducted pre-1980 and do not fully conform to current guidelines, such as OECD or Good Laboratory Practice (GLP) guidelines. The most informative of these studies are described below.

In a study conducted by the Food and Drug Research Laboratories (FMC Corp Ind Chem Div 1975, cited in USEPA 2005, OECD 2002, OECD 2005), Wistar rats (five per sex) were given a single dose of 20 g/kg bw as a 25 per cent aqueous solution of triphenyl phosphate (of unknown purity) by gavage. The only other details of this study given are a 24-hour fasting period prior to dosing and that observations were limited to mortality for 14 days post-dosing. Necropsy was conducted on all animals. There were no deaths and necropsy revealed sporadic visceral hemorrhages. The LD_{50} was therefore greater than 20 g/kg bodyweight.

Johannsen *et al*. (1977) conducted a study in Sprague-Dawley rats (male and female; number not stated). The animals were given doses of triphenyl phosphate (purity not reported) in corn oil, up to 15,800 mg/kg bodyweight (individual doses not given). In a 14-day post-exposure observation period, mortality was monitored. The mortality data were not reported, however the LD_{50} was stated as 10,800 mg/kg bodyweight.

Male and female (total five per dose) mice (strain not specified) were given triphenyl phosphate (unknown purity), as an emulsion in aqueous gum acacia, at a dose of 2,500 or 5,000 mg/kg bodyweight. Observations of mortality and overt signs of toxicity were recorded over a post-dosing period of eight days. There was slight stupor noted for an unspecified time following dosing but no deaths occurred at either dose. Therefore, the LD_{50} in this study was greater than 5,000 mg/kg bodyweight (Ciba-Geigy 1954, cited in USEPA 2005, OECD 2002, OECD 2005).

Five groups, each of two hens (Rhode Island Red and Light Sussex), were given a single dose of triphenyl phosphate (in arachis oil) by gavage at dose levels of 2, 3, 5, 8 or 12.5 g/kg bodyweight, and observed for signs of neurotoxicity for 21 days. A necropsy of each of the hens was conducted following the 21-day observation period. There were no signs of toxicity or adverse necropsy findings. The LD_{50} and no observed effect level (NOEL) were therefore greater than 12.5 g/kg bodyweight (Ciba-Geigy 1980, cited in OECD 2002, OECD 2005). Similar studies in hens are available, but neurotoxicity was not observed in any of these (OECD 2005).

A number of less well-documented studies in rats, mice, guinea pigs, hens and rabbits confirmed the low potential for acute oral toxicity for triphenyl phosphate and found the LD_{50} values to be greater than the maximum doses tested (OECD 2002).

Inhalation

No reliable, valid data are available.

Two studies predate current testing guidelines. In one of these, conducted by the Food and Drug Research Laboratories (FMC Corp Ind Chem Div. 1975, cited in USEPA 2005, OECD 2002, OECD 2005), Wistar rats (five per sex) were exposed to a triphenyl phosphate powder (200 mg/l for one hour). However, the test report did not mention analysis of the particle size, so it is not known whether the particle size was in the respirable range. There was no sign of toxicity and no deaths were reported over a 14-day

observation period. In the second study (Sutton *et al*. 1960), Carworth Farms CF 1 male mice were exposed to triphenyl phosphate vapour (363 mg/l for six hours, or 757 mg/l for two and four hours). No toxic effects were observed. However, this study is not considered valid because the exposure levels were too low, the observation period of 24 hours is considered inadequate, and it appears that the test conditions involved use of a closed chamber which does not accord with the current test guideline.

Dermal

In a study conducted by the Food and Drug Research Laboratories (FMC Corp Ind Chem Div. 1975, cited in USEPA 2005, OECD 2002, OECD 2005), two groups of five albino rabbits (sex not specified) were treated with a dose of 10,000 mg/kg bodyweight triphenyl phosphate on either intact or abraded skin, and observed for 14 days. No additional information on the purity of triphenyl phosphate or the vehicle used was reported. None of the animals died (no further details reported). Therefore, the LD_{50} was greater than 10,000 mg/kg bodyweight.

Johannsen *et al*. (1977) applied undiluted triphenyl phosphate occlusively to the intact dorsal skin of male and female New Zealand white rabbits (numbers not specified) for 24 hours. Residues were then washed off and the animals observed for 14 days. At the end of the observation period a necropsy was performed on all of the animals. The results of the necropsy were not reported; however, none of the animals died and the LD_{50} was therefore considered to be above the maximum stated dose (other doses not given) of 7,900 mg/kg bodyweight.

Other

Several valid studies in which rats, mice or guinea-pigs were dosed by intraperitoneal or subcutaneous injection have given LD_{50}s greater than 3,000 mg/kg bodyweight, confirming the low acute toxicity of triphenyl phosphate irrespective of route of administration (OECD 2002).

Summary of acute toxicity

No information is available from human studies.

No studies conducted to current test guidelines are available for acute oral toxicity. However, the many limited studies allow adoption of a weight of evidence approach. After oral administration to rats, mice, rabbits, guinea pigs and hens, LD_{50} values are all in a range from 3,000 to above 20,000 mg/kg bodyweight. This is far above the limit dose (2,000 mg/kg bodyweight) applied in modern studies, which indicates a low level of toxicity after oral administration (OECD 2005).

There are no valid data for acute inhalation exposure.

The toxicity of triphenyl phosphate after dermal application is very low, with an LD_{50} of above 7,900 mg/kg bodyweight in rabbits.

A number of additional studies using intraperitoneal or subcutaneous routes are available. Among those studies considered to be of adequate quality, findings confirm that triphenyl phosphate is of low toxicity (OECD 2005).

4.4.3 Irritation

Only experimental animal data are available.

Skin

The skin irritation potential of triphenyl phosphate was investigated in a good quality study conducted to OECD test guideline 404 and GLP (Bayer AG 1990, cited in OECD 2002, 2005). In this study, three rabbits (strain not stated) were treated for four hours by applying 500 mg of water moistened triphenyl phosphate (99.7 per cent triphenyl phosphate as Disflamoll TP) to their clipped dorsal skin (2 × 3 cm) under a semi-occlusive patch. Rabbits were then observed daily for 14 days for signs of irritation. Erythema and oedema were not observed and the irritation index was zero.

Two additional studies were judged to be adequate by the USEPA (2005) and OECD (2005). In the first of these studies conducted by the Food and Drug Research Laboratories (FMC Corp Ind Chem Div. 1975, cited in USEPA 2005, OECD 2005), 500 mg triphenyl phosphate was applied to the shaved backs of six albino rabbits (sex not specified). A semi-occlusive patch was used on abraded and intact skin for 24 hours. After 24 hours the dressing was removed and observations were recorded at 24 and 72 hours. Neither erythema nor oedema was observed in abraded or intact skin in any of the rabbits.

In the second study conducted by Ciba-Geigy Pharmaceuticals Division (1983a, cited in USEPA 2005, OECD 2005), 500 mg triphenyl phosphate was applied under two occlusive patches on to the backs of six (three per sex) New Zealand white rabbits. One skin site was intact and the other abraded. The dressings were removed after 24 hours and irritation scored at 24 and 72 hours. No signs of irritation (erythema and oedema) were reported for either time point.

Several other studies are available. However, they are not discussed here owing to their poor reporting or inadequate protocols. All gave negative results.

Eye

Bayer AG (1990, cited in OECD 2002, 2005) reported a study conducted according to OECD test guideline 405 and GLP. In this good quality study, three rabbits (strain and sex not stated) were treated for 24 hours by applying 70 mg triphenyl phosphate (99.7 per cent triphenyl phosphate as Disflamoll TP) into the conjunctival sac of one eye. The other eye was untreated, thus serving as a control. After 24 hours the eyes were rinsed and examined for signs of irritation. There were no effects on the iris or cornea of any of the animals. However, there were mild, reversible signs of irritation to the conjunctiva comprising: slight redness in all treated eyes one to 24 hours after instillation, slight swelling of the eye in one animal after one hour and discharge from the eye in all animals after one hour. The study authors concluded that the mild reactions observed immediately following instillation were mechanically induced, and that the test substance did not have a significant potential to cause eye irritation (OECD 2005).

Two additional studies have been summarised by OECD (2002, 2005) and USEPA (2005). These studies were conducted using protocols approximating to the then US test guidelines, and are considered to be of adequate quality to assess the potential of triphenyl phosphate to cause eye irritation.

In the first of these studies, the Food and Drug Research Laboratories (FMC Corp Ind Chem Div. 1975, cited in USEPA 2005, OECD 2005) instilled 100 mg triphenyl phosphate into one eye each of six albino rabbits (group 1) without rinsing. In three additional albino

rabbits, eyes were rinsed four seconds after instillation of 100 mg triphenyl phosphate (group 2). All eyes were examined at 24, 48 and 72 hours, and seven days after instillation of triphenyl phosphate. In group 1, conjunctival effects were observed in all six animals (grade 1 redness in all animals and discharge in four animals). These effects resolved over the course of the 72-hour observation period. In group 2, no ocular effects were observed in any of the three animals. Thus, triphenyl phosphate was found to be mildly and transiently irritating when not washed out.

In a study conducted by Ciba-Geigy (1983b, cited in USEPA 2005, OECD 2005) triphenyl phosphate was also found to be minimally irritating to the eye. A dose of 100 mg of triphenyl phosphate (purity not stated) was instilled into the conjunctival sac of the left eyes of six New Zealand white rabbits (three per sex) and the eyelids held closed for one second. The eyes of three rabbits were flushed with approximately 200 ml warm water 30 seconds after instillation. The eyes of the other animals were not washed. Eye examinations were conducted at 1, 24, 48 and 72 hours and six days after triphenyl phosphate instillation. Mild conjunctival effects (slight redness) were observed in all exposed eyes by 24 hours which cleared in all but one unwashed eye by 72 hours, and developed in that eye by six days. Slight corneal opacity and damage to the surface epithelium was seen in one unwashed eye at 24 hours; this was no longer present at 48 hours. Thus, a minimal irritating potential of triphenyl phosphate in New Zealand white rabbits was found (highest score 7; score 0 - 10 minimally irritant).

Summary of irritation

No information is available from human studies.

In one OECD TG 404 study and two other reasonable quality, valid studies there was no evidence to suggest that triphenyl phosphate has skin irritation potential.

There is evidence from a study conducted to OECD TG 405 and other adequate studies that triphenyl phosphate causes mild reversible irritation to the eye, primarily to the conjunctiva. However, this initial irritation was thought to be mechanically induced. Thus, it was concluded that the test substance did not have a significant potential to cause eye irritation.

4.4.4 Corrosivity

None of the studies available for skin and eye irritation suggest triphenyl phosphate to have corrosive properties.

4.4.5 Sensitisation

Skin

Several human cases of apparent allergic dermatitis as a result of triphenyl phosphate exposure have been reported. One example is a reported case of allergy to triphenyl phosphate from cellulose acetate eyeglass frames that contained triphenyl phosphate as an additive. However, according to the authors, the reaction observed could have been due to tricresyl phosphate that was also present (Carlsen *et al*. 1986, cited in WHO 1991). Similarly, a few individuals have been reported to show positive reactions in patch tests using cellulose acetate film containing both tricresyl phosphate and triphenyl phosphate. Among the 23,192 patients patch tested from 1950 to 1962, positive reactions to cellulose

acetate film containing 7 to 10 per cent triphenyl phosphate and three to four per cent phthalic esters occurred in 15 individuals (0.065 per cent). Sensitivity to cellulose acetate film was analysed in detail in only two of these cases; in both cases the sensitizer was identified as triphenyl phosphate. In the other cases, the causative agent was uncertain since it might have been triphenyl phosphate, tricresyl phosphate or a phthalic ester (Hjorth 1964, cited in OECD 2002, WHO 1991).

Patch testing of individuals with dermatological problems has generally not implicated triphenyl phosphate as a skin sensitiser. For example, Tarvainen (1995, cited in OECD 2002, USEPA 2005) tested 343 patients because of suspected sensitivities to components of plastics and glues, and found none reacted to triphenyl phosphate. The low incidence of sensitization was confirmed by another study (Kanerva *et al*. 1997, 1999, cited in OECD 2002) in which 174 patients were tested, with irritation reactions noted in one patient but no evidence of sensitisation observed.

Ten test and five control male Dunkin-Hartley guinea pigs were used in a good quality maximisation study conducted to OECD test guideline 406 'Skin sensitisation', and GLP (Sanders 2001). Two phases were involved: an induction of a response and a challenge of that response. In preparation for the induction phase, an area of hair was shaved from the shoulder region of each animal. A row of three injections was then made on each side of the midline, comprising: a) Freund's Complete Adjuvant (FCA) plus distilled water in a ratio of 1:1; b) five per cent w/w formulation of triphenyl phosphate in arachis oil BP; and c) five per cent formulation of triphenyl phosphate in a 1:1 preparation of FCA and distilled water. Approximately 24 and 48 hours after administration the degree of erythema at each injection site was assessed. On day seven, the same area of skin was again clipped and triphenyl phosphate (in arachis oil) applied topically under an occlusive dressing for 48 hours. The degree of oedema and erythema were assessed one and 24 hours after removal of the dressings. For the control groups the same procedure was employed, except for the omission of triphenyl phosphate from the injections, and vehicle alone (arachis oil without triphenyl phosphate) was used in the topical application. Shortly before day 21, an area of skin on the flanks of each animal was shaved to begin the challenge phase. The maximum non-irritant triphenyl phosphate concentration (in arachis oil) was then applied to the shaved areas under an occlusive dressing for 24 hours. The dressings were removed and residual test material washed away with distilled water. Approximately 24 and 48 hours after the challenge phase, the degree of oedema and erythema was assessed. Skin reactions at the challenge sites of test animals were considered to be due to sensitisation unless reactions of equal severity were observed in control animals. Reactions of equal severity in test and control animals were considered to indicate irritation. In addition, for the definition of challenge reactions to apply, the reaction should persist for 48 hours after exposure. Based on these defined study criteria, triphenyl phosphate produced a zero per cent sensitisation rate and consequently the authors concluded that triphenyl phosphate was not a skin sensitizer under the conditions of this test.

Summary of sensitisation

There are a few human case reports showing evidence of skin sensitisation. However, the incidence of skin sensitisation is low, and the causative agent is often uncertain.

Triphenyl phosphate was negative for skin sensitisation in a good quality guinea pig study conducted to OECD test guideline 406 and GLP.

No information on respiratory tract sensitisation for triphenyl phosphate is available.

4.4.6 Repeated-dose toxicity

Animal data

There are no data relating to repeated inhalation exposure to triphenyl phosphate.

No repeat dose toxicity studies conducted to OECD test guidelines are available. OECD (2002, 2005), WHO (1991), and USEPA (2005) cited a similar limited set of studies in their reports on triphenyl phosphate.

Sutton *et al*. (1960) conducted a study on three groups of five male Holtzman rats treated with triphenyl phosphate (as Practical Grade Eastman Organic Chemicals No. P1149) by dietary administration for 35 days; the study design was limited by the small number of animals in each dose group, testing of only male animals, apparent absence of analysis of the achieved concentrations in the test diets, lack of clinical chemistry and histopathology investigations and poor reporting without inclusion of sufficient detail. The initial nominal dietary concentrations employed were zero (control; received untreated commercial rat food), 0.5 and 5.0 per cent w/w (estimated dose: 350-3,500 mg/kg bw/day). However, the high dose of 5.0 per cent w/w had to be reduced to 0.1 per cent (approximately 70 mg/kg bw/day) after three days as animals given this level refused food and lost weight. Hence, for the majority of the study period, the highest dose employed was 0.5 per cent w/w. Clinical observations, bodyweights (three times per week), food consumption, and haematology ('periodic' examination of: haemoglobin content, cell volume, red cell count, total and differential white cell count) were monitored. At the end of the treatment period, three rats from each dose group were maintained untreated for a further 14-day recovery period. The other two rats were killed at the end of the 35-day dosing period. All animals were killed and subjected to gross necropsy, and liver and kidney weights were recorded. Treatment caused a slight depression of bodyweight gain in the 0.5 per cent group (weight gain at 35 days: control, 181g; 0.1 per cent, 177g; 0.5 per cent, 169 g) with recovery during the period of withdrawal of treatment. A significant increase in liver weights (liver weights expressed as per cent bodyweight at 35 days: control, 3.9; 0.1 per cent, 4.0; 0.5 per cent, 4.9) was reported for the 0.5 per cent (estimated nominal dose: 350 mg/kg bw/day) group, although it is not clear whether all five rats per group were included in organ weight determinations or only those killed after 35 days treatment. No treatment-related clinical signs or changes in haematological profile were noted, and necropsy findings were unremarkable. No effects were noted in the group fed 0.1 per cent in the diet (estimated dose: 70 mg/kg bw/day) for the majority of the treatment period, and this level was considered to be the NOEL (Sutton *et al*. 1960). This level of exposure is considered to be the overall NOEL for triphenyl phosphate for this assessment.

Two similarly designed 120-day studies were conducted in Sprague-Dawley rats to investigate possible effects on the immune (Hinton *et al*. 1987) or nervous systems (Sobotka *et al*. 1986). Only details relating to standard repeated dose toxicity parameters are reported here. Results for immunotoxicity and neurotoxicity are discussed later in this section.

Hinton *et al*. (1987) administered triphenyl phosphate (98 per cent pure) at concentrations of 0, 0.25, 0.5, 0.75, and 1.0 per cent w/w in the diet (corresponding to nominal doses of approximately 0, 161, 345, 517 and 711 mg/kg bw/day) to Sprague-Dawley rats (ten/sex/group) for 120 days. The animals were observed daily for clinical signs, as well as changes in bodyweight and food consumption, which were recorded weekly. Blood was obtained on days 0, 30 and 60 to investigate effects on the immune system. Only limited data were reported and a number of standard parameters of repeated dose toxicity were not reported (such as organ weight measurement and histopathology of organs other than lymphoid organs, or haematology and clinical chemistry other than serum proteins). There

was an apparent dose-dependent reduction in the rate of growth (compared with controls) of male rats in the first two months of treatment, with a significant effect on the 1.0 per cent group (above 10 per cent decrease; $p<0.01$) for the first four weeks. Females did not show a dose-dependent reduction in growth rate, and only showed a marginal reduction in body weight at the highest dose level in the first four weeks. There were also non-dose related increases in the relative percentages of alpha-globulins in treated females and beta-globulins in treated males. The authors of the study considered these effects to be possible signs of liver activity of uncertain toxicological significance. However, there was no dose-response, so they were probably not treatment-related. Therefore, the no observed adverse effect level (NOAEL) for general toxicity was 517 mg/kg bw/day, based on the reduced bodyweight gain in males at the highest dose.

Sobotka *et al*. (1986) administered triphenyl phosphate (98 per cent pure) at concentrations of 0, 0.25, 0.5, 0.75, and 1.0 per cent w/w in the diet (corresponding to doses averaged across the 120 days of approximately 0, 161, 345, 517 and 711 mg/kg bw/day) of Sprague-Dawley rats (ten males per group) for 120 days. The animals were observed daily for clinical signs, and bodyweight and food consumption were recorded weekly. Tests for neurotoxic effects are discussed below. There was a treatment-related effect on bodyweight gain but not food consumption. Group cumulative monthly weight gains were significantly reduced in the 0.5 and 1.0 per cent dose groups throughout the study ($p < 0.037$). However, the reductions in cumulative bodyweight observed in the latter parts of the study were concluded to be attributable solely to the reduction during the first month of treatment. Significant ($p < 0.01$) reductions in cumulative weight gains were also apparent in the 0.75 per cent dose group for months one and two, but not three or four, of treatment. There was no effect on weight gain in the lowest dose group. No other overt signs of toxicity were observed. Therefore, the NOAEL for this study was 161 mg/kg bw/day, based on the effects on bodyweight gain.

The toxicity of triphenyl phosphate after repeated dermal exposure was investigated in New Zealand white rabbits. Ten male and ten female animals were assigned, after pre-treatment screening for normality of haematology, clinical chemistry, bodyweight and general health, to groups treated with distilled water (1 ml/kg; control) or with triphenyl phosphate in ethanol (at 0.2 or 2 ml/kg bw/day of a 50 per cent solution) on clipped, intact (half of the animals) or abraded (half of the animals) skin, five times per week for three weeks, giving doses of 0, 100 or 1,000 mg/kg bw/day (assuming the test material was 100 per cent pure). Because the application was not occluded, ingestion of the applied test material was prevented by means of a collar. Observations of the site of treatment, as well as all other endpoints assessed (including mortality, clinical signs, bodyweight, haematology, clinical chemistry, gross pathology, organ weights, and histopathology of more than 30 tissues including the reproductive organs and nervous system) identified no treatment-related differences between treated and control animals. The only treatment-related effect was an unquantified depression of acetyl cholinesterase in plasma, erythrocytes and the brains of triphenyl phosphate treated rabbits; no clinical or histological correlates were found. This effect was not considered by the authors to be of toxicological relevance[8], and the NOEL for this study was considered to be greater than 1,000 mg/kg bw/day (Tierney 1979).

Neurotoxicity

Neurotoxicity is an established adverse effect of many organophosphates. However, in the available studies in hens (see Ciba-Geigy 1980, cited in OECD 2002, 2005, see Section 4.4.2) and cats, pure triphenyl phosphate did not induce acute cholinergic toxicity or delayed neuropathy. These studies, performed prior to the existence of guidelines, do not entirely conform to current standards. Nevertheless, they do allow a weight of

[8] Further information on the extent and nature of this would be helpful to fully assess its toxicological significance.

evidence approach, which indicates a lack of delayed neurotoxicity for triphenyl phosphate (USEPA 2005). The findings of a decreased activity of choline esterase and paralysis, predominantly in cats, in older studies suggestive of a neurotoxic potential were not reproduced in later studies and may be due to contamination of the tested samples by other organophosphorus esters. In addition, at the high doses of triphenyl phosphate used, even small proportions of impurities might have had anticholinesterase activity (OECD 2002).

Although the rat is of limited value as a model for delayed neurotoxicity, the absence of neurotoxic effects after 120 days of treatment with triphenyl phosphate supports the evidence (Sobotka *et al*. 1986) from the other species. As described above, Sobotka *et al*. (1986) administered triphenyl phosphate (98 per cent pure) at concentrations of 0, 0.25, 0.5, 0.75, and 1.0 per cent w/w in the diet (corresponding to doses averaged across the 120 days of approximately 0, 161, 345, 517 and 711 mg/kg bw/day) to Sprague-Dawley rats (ten males per group) for 120 days. A battery of behavioural tests designed primarily to assess multiple indices of neuromotor function were conducted on all animals at monthly intervals, beginning at the end of the first month of dietary treatment. Behavioural tests included measures of mobility and exploratory behaviour (open field activity), balance and general motor coordination (accelerating rod performance) and muscular strength (forelimb grip strength). Measures of vestibular function and motor coordination (negative geotaxis) were also included at the end of the third and fourth months. Test results for neurobehavioral endpoints were not statistically significantly different in treated versus control animals. Only effects on body weight were observed (described above). The NOAEL for neurotoxicity was therefore greater than the highest dose tested (approximately 711 mg/kg bw/day).

Immunotoxicity

The potential immunotoxicity for triphenyl phosphate was investigated in detail in a subchronic dietary study (Hinton *et al*. 1987, described above) in Sprague-Dawley rats, which predates the current guideline for immunotoxicity. At the end of the 120-day treatment period, immunotoxicity was assessed by the humoral response to antigens (anti-sheep red blood cell assay), measurement of the weights of lymphoid organs (spleen and thymus), histopathology of the spleen, thymus and mesenteric lymph nodes, immunohistochemical evaluation of B- and T-lymphocyte regions in these tissues, and measurement of total serum protein and electrophoretic analysis of serum proteins. Besides the effects on bodyweight gain and globulins described above, there were no other effects on the treated animals. Therefore, the NOAEL for immunotoxicity was considered to be greater than the highest dose tested (approximately 711 mg/kg bw/day).

Human data

No reliable human data are available.

Summary and discussion of repeated- dose toxicity

There are no human data on the potential for triphenyl phosphate to cause adverse health effects following repeated exposure by any route.

No repeat dose toxicity studies conducted to current OECD test guidelines are available. However, a number of limited studies allow a weight of evidence approach. Based on the available studies in rats or rabbits, the toxicity after repeated treatment with triphenyl phosphate is low. The majority of studies did not report on the full range of potential endpoints that would routinely be recorded in a study to current test guidelines, but taken together the studies using dietary doses of up to approximately 711 mg/kg bw/day or

dermal doses of up to 1000 mg/kg bw/day address clinical observations, body weight gain, food consumption, haematology, clinical chemistry, organ weights and histopathology. After 35 days of dietary administration, a slight depression of bodyweight gain and an increase of liver weights was noted in rats at a level of 0.5 per cent (estimated dose: approximately 350 mg/kg bw/day). However, a concentration of 0.1 per cent (estimated dose: approximately 70 mg/kg bw/day) in the diet was without any effect. Limited studies with treatment of rats for 120 days at concentrations of up to one per cent in the diet (approximately 711 mg/kg bw/day) confirm this slight effect on growth. The low general toxicity was confirmed after dermal exposure of 100 or 1,000 mg/kg bodyweight in rabbits for 15 days without any apparent sign of toxicity besides an unquantified depression of acetylcholinesterase as the only dose-related effect.

Overall, general wellbeing, immune and nervous systems were not affected by exposure to triphenyl phosphate. The NOAEL for repeated dose toxicity is 70 mg/kg bw/day, based on decreased bodyweight and increased liver weight following administration of triphenyl phosphate at a higher dose of around 350 mg/kg bw/day to rats for 35 days.

4.4.7 Mutagenicity

Studies in vitro

Genetic mutations

Three valid *in vitro* studies investigated the potential for triphenyl phosphate to induce gene mutations. These studies, two in bacteria and one in mammalian cells, were not conducted to current test guideline standards. However, the bacterial mutation test protocols were similar to those of OECD test guideline 471. In most cases, there was no information on whether the studies were conducted to GLP. Other *in vitro* studies are available and are summarised in OECD (2005), but their validity is questionable owing to the poor level of reporting in the reference and/or the use of limited or non-standard protocols. These latter studies are not included in this assessment.

Zeiger *et al*. (1987, cited in OECD 2002, USEPA 2005) conducted a bacterial reverse mutation assay using *S. typhimurium* TA 98, TA 100, TA 1535, TA 1537 and TA 1538 with and without activation by S9 mix from Aroclor 1254 treated rat livers. Bacteria were preincubated for 20 minutes at 37 °C. Experiments were performed in triplicate and repeated. According to USEPA (2005) the test concentrations were 0, 100, 333, 1,000, 3.333 and 10,000 µg triphenyl phosphate/plate (triphenyl phosphate purity of above 98 per cent). However, a precipitate was observed at the two highest concentrations. Positive controls were 2-aminoanthracene for all strains with S9, and sodium azide (TA 1535 and TA 100), 9-aminoacridine (TA 1537), and 4-nitro-o-phenylenediamine (TA 98) in the absence of S9. No cytotoxicity was apparent, but the two highest concentrations exceeded solubility limits (USEPA 2005). There was no evidence of mutagenic activity in any of the tester strains.

In a bacterial reverse mutation study conducted by Monsanto (1978a, cited in OECD 2002, USEPA 2005, WHO 1991), *S. typhimurium* TA 98, TA 100, TA 1535, TA 1537, TA 1538 and *S. cerevisiae D4* were incubated for 48 hours (at 37^{o}C) with and without metabolic activation (S-9 mix from Aroclor 1254-induced adult male Sprague-Dawley rats), and triphenyl phosphate at concentrations of 0, 10, 100, 500 and 1,000 µg/plate (one plate per concentration). Experiments were performed in triplicate and repeated. Negative and positive controls were performed (no further information). There was no increase in revertants compared with controls at any of the tested concentrations, with or without metabolic activation.

In a mammalian cell gene mutation test conducted by Monsanto (1978b, cited in OECD 2002, USEPA 2005, WHO 1991), triphenyl phosphate was tested for its ability to induce mutations at the thymidine kinase (TK) locus in cultured L5178Y mouse lymphoma cells. Tests were conducted in triplicate and repeated, with or without metabolic activation (S9 mix from Aroclor 1254-induced adult male Sprague-Dawley rats), at triphenyl phosphate concentrations of 0, 3.13 (only without S9), 6.25, 12.5, 25, 50 and 75 (only with S9) µg/plate. The highest concentration was selected as it caused reduced bacterial growth in a pre-screening test. Negative and positive controls (ethyl methanesulfonate without S9, and dimethylnitrosamine with S9) were included (no further information available). Triphenyl phosphate did not induce significant mutations at the TK locus.

Cytogenetic effects

The potential for triphenyl phosphate to cause unscheduled DNA synthesis (UDS) was investigated in a study conducted by Schmuck (1989, cited in OECD 2002, 2005) in Syrian hamster embryonic fibroblast cells at concentrations of 0.05 to 10×10^{-5} M. After five hours incubation without activation in the presence of 3H-thymidine, triphenyl phosphate showed no genotoxic effect (no further details given).

Chromosomal effects

No *in vitro* test data concerning chromosomal aberrations are available.

Studies in vivo

No *in vivo* data are available for gene mutations or chromosomal aberrations.

Summary of mutagenicity

Tests for gene mutations in Salmonella bacterial, mammalian cells and yeast did not reveal any sign of mutagenicity (OECD 2002, WHO 1991). In addition, triphenyl phosphate does not appear to induce unscheduled DNA synthesis based on the results of a UDS test. Although the authors of OECD (2002) considered these studies to be valid, the robustness of many of the studies could not be verified due to the limited level of reporting in the latest IUCLID (OECD 2005). There are no data on the potential of triphenyl phosphate to cause chromosomal aberrations *in vitro* or *in vivo*, or gene mutations *in vivo*. Therefore, it is not possible to fully evaluate the genotoxic potential of triphenyl phosphate.

4.4.8 Carcinogenicity

No long-term carcinogenicity bioassays are available for triphenyl phosphate. The Strain A mouse pulmonary adenoma study is not suitable to fully characterise the potential for carcinogenicity of chemicals following repeated long-term oral exposure. However, as the only available study to give any indication of carcinogenic potential for triphenyl phosphate, a study of this type has been included here for completeness.

Theiss *et al*. (1977) tested 16 organic water contaminants, including triphenyl phosphate, in the Strain A mouse pulmonary adenoma test. In this study triphenyl phosphate (purity above 95 per cent) was administered by intraperitoneal injection to mice (20 males per group) at doses of 0 (tricaprylin vehicle), 20, 40 or 80 mg/kg bodyweight. The lowest dose was administered three times per week for six weeks (total of 18 injections), the middle dose three times per week for one week (three injections) and the highest dose was only

administered once. The mice were killed 24 weeks after the first injection and the lungs examined macroscopically for surface nodules. A few of the nodules were examined histologically to confirm whether they were adenomas. Positive controls, which received urethan, responded appropriately. Triphenyl phosphate did not produce a pulmonary adenoma response that was significantly greater than that of the vehicle-treated control mice. Although OECD (2002) assigned this study a Klimisch code of 2 (valid with restrictions), it was considered inadequate by WHO (1991) due to the low survival rate of treated animals (vehicle control, 46/50; 20 mg/kg bw, 18/20; 40 mg/kg bw, 3/20; 80 mg/kg bw, 12/20) and the short duration of the study.

4.4.9 Toxicity to reproduction

Reproductive and developmental toxicity was evaluated in a study (Welsh *et al*. 1987) that does not comply with any OECD test guidelines. However, it does fulfil the requirements of the prenatal developmental toxicity study guideline (OECD test guideline 414). In addition, it has many of the elements of a reproduction/ developmental toxicity screening study (OECD test guideline 421), although the study did not allow animals to deliver and continue to dose until at least four days post-delivery. Dosing stopped at day 20 of gestation when caesareans on killed dams were conducted. A further limitation of the study was the lack of histopathology on male and female reproductive organs. Despite these limitations, the study is considered valid and adequate in exploring whether triphenyl phosphate causes reproductive toxicity.

Fertility and reproductive toxicity

In a good quality study (Welsh *et al*. 1987) that included fertility and developmental toxicity endpoints, Sprague-Dawley rats were randomly assigned to one of five dietary groups (40/sex/group). Four of the groups received 0.25, 0.50, 0.75 or 1.0 per cent (approximate nominal doses of 166, 341, 516 or 690 mg/kg bw/day) triphenyl phosphate in their diets for an initial 91 days. The fifth group, the controls, received untreated diet. Upon completion of this sub-chronic feeding phase, male and female animals from the same feeding groups were cohabited in a 1:1 ratio and left overnight. Females were examined for the presence of sperm the next day. The day that sperm was found was designated as day 0 of gestation. The animals continued to receive test diets throughout mating and gestation. Daily observations for changes in general appearance and health of the dams were recorded throughout gestation. On day 20 of gestation, dams were examined externally and caesarean sections performed. Body weights and food consumption were measured on days 0, 7 and 14 of gestation, and on day 20 prior to caesarean section. At necropsy, dams were subject to examination of their major organs. Ovaries were removed and numbers of corpora lutea noted. The uterus was removed and weighed. The number and position of fetuses (viable and dead) and resorption sites (early and late) were recorded. When animals appeared not to be pregnant, their uteri were stained to enhance visibility of any resorption sites. Fetuses were examined for gross abnormalities and, for each, uterine position, sex, weight and crown-rump length was recorded. Approximately half of the fetuses from each litter were the subjects of visceral examination and the remaining half underwent skeletal examination.

No significant signs of parental toxicity were noted. A slight but statistically significant ($p > 0.05$) lower bodyweight was apparent for females in the highest dose group at the start of mating compared with controls, suggesting possible slight toxicity had occurred during the subchronic phase. However, while intergroup variations in bodyweight increase were found at various times throughout pregnancy, these were not statistically significant and did not demonstrate a strict dose-relationship. During gestation, the treated dams generally consumed slightly more food than the controls although again no consistent

dose-relationship was noted. No differences between treated and control animals in behaviour or gross pathology were noted. The control rats used in this study had a relatively low pregnancy rate (74 per cent); the percentages of pregnancies in treated animals (85 to 95 per cent) were, however, within the expected range for Sprague-Dawley rats. No significant differences between treated and control rats were recorded in the number of corpora lutea, implants, implantation efficiency, viable fetuses or early and late resorptions (see Table 4.7). As there was no effect on the litter size (indirectly measured by the number of viable fetuses and implants) and both sexes were treated in the study, these findings indicate that fertility is not adversely affected by triphenyl phosphate in male and female rats. The NOAELs for maternal toxicity, male and female fertility and reproductive toxicity in this study were therefore greater than the highest dose tested (approximately 690 mg/kg bw/day) (Welsh *et al*. 1987). However, a higher dose of five per cent triphenyl phosphate in the diet of rats leads to food refusal (Sutton *et al*. 1960), and therefore testing of dietary concentrations substantially higher than one per cent triphenyl phosphate would not be advisable. In addition, in a three week dermal repeated dose toxicity study in male and female rabbits, there was no effect on the reproductive organs up to the highest dose of 1,000 mg/kg bodyweight (Tierney 1979, cited in OECD 2002).

Developmental toxicity

In the dietary administration study described above (Welsh *et al*. 1987), no treatment-related changes in fetal survival or the types or numbers of anomalies in the fetuses were identified. There were no specific external or skeletal variations which could be related to treatment with triphenyl phosphate. However, all treated groups showed a significant increase in the incidence of fetuses with moderate hydroureter when compared with the control group. The authors concluded that because of the high baseline incidence in the control group and lack of a clear dose-related response, the biological significance of this finding was unclear. There were also significantly more fetuses with moderately enlarged ureters in the region adjacent to the kidney in the treated groups compared with controls. However, again the incidence was not dose-related, since a greater proportion of fetuses were affected at the lower doses than at the two higher ones. The average number of fetuses having at least two soft-tissue variations was significantly higher in the 0.25, 0.50 and 0.75, but not the 1.0 per cent, triphenyl phosphate groups compared to the control group; the incidence in the 1.0 per cent group was only slightly, non-significantly higher than the controls. In addition, the number of litters with fetuses having at least two soft-tissue variations was statistically significantly greater than controls in the 0.5 and 0.75 per cent groups (Welsh *et al*. 1987). In summary, there were slight increases in the numbers of litters, with fetuses showing soft tissue variations, but no dose-relationship was apparent. The NOAEL for developmental toxicity was therefore considered to be greater than one per cent in the diet (approximately 690 mg/kg bw/day).

Summary of toxicity to reproduction

In study conducted by Welsh *et al*. (1987), there were no significant differences in the number of corpora lutea, implants, implantation efficiency, viable fetuses and number of early or late resorptions following dietary exposure to triphenyl phosphate up to 1.0 per cent (approximately 690 mg/kg bw/day) for 91 days prior to mating, and during mating and gestation. There was also no effect on litter size, thus providing evidence that triphenyl phosphate does not affect fertility in the rat. There were also no treatment-related effects on fetal development. Hence, the NOAEL for fertility and developmental toxicity was greater than the highest dose of 690 mg/kg bw/day.

4.4.10 NOAEL and Margins of Safety (MOS) for assessment of human exposure via the environment

Two studies are considered to provide information potentially suitable for derivation of an overall NOEL. One was a study by Hinton *et al*. (1987) that focused principally on establishing the neurotoxic profile of the chemical, but this did not examine many standard parameters and is therefore considered to be of limited value. In a further repeat dose toxicity study (Sutton *et al*. 1960) that was of a more comprehensive design, a NOEL/NOEC of 0.1 per cent in diet for triphenyl phosphate was determined, and this is selected as the most comprehensive and conservative option. In the Sutton *et al*. study, three groups of five male Holtzman rats were treated by dietary administration of triphenyl phosphate for 35 days. Nominal dietary concentrations were initially zero (control; received untreated commercial rat food), a low dose of 0.5 and a high dose of 5.0 per cent w/w (estimated dose: 350 to 3,500 mg/kg bw/day). However, the dietary concentration for the group initially given a high dose of 5.0 per cent w/w had to be reduced to 0.1 per cent (approximately 70 mg/kg bw/day) after three days because the animals refused the higher diet and lost weight; thus, after the third day animals of this group formed a new low dose, with those given 0.5 per cent w/w throughout being considered as the high dose. Treatment caused a slight depression (no further details given) of bodyweight gain and an increase of liver weights (no further detail given) at the 0.5 per cent dose level. No treatment-related clinical signs or changes in haematological profile were noted, and necropsy findings were unremarkable. At the reduced concentration of 0.1 per cent in the diet (estimated achieved dose: 70 mg/kg bw/day), animals showed no effects, and this dose was considered to be the NOEL/NOEC for general toxic endpoints. The NOEL for other endpoints, such as neurotoxicity, and reproductive and developmental toxicity, was considered greater than 0.1 per cent diet since no effects were observed in any of these parameters at any of the doses tested in these studies.

Table 4.7 Maternal reproductive and necropsy findings in rats fed triphenyl phosphate*

Dosage (% diet)	Number (%) pregnant females	Corpora lutea (mean ± SEM/female)[a]	Implants (mean/female)	Implantation efficiency (average % ± SEM/female)[b]	Viable fetuses (mean ± SEM/female)	Average % resorbed	Early deaths/litter[b]	Mean late deaths/litter[b]
0	29 (74)	16.10 ± 0.38	14.48 ± 0.75	89.16 ± 4.14	13.00 ± 0.69	13.69	1.48	0
0.25	38 (95)	16.29 ± 0.31	14.16 ± 0.44	86.84 ± 2.5	12.97 ± 0.42	7.90	1.18	0
0.5	35 (92)	16.31 ± 043	13.31 ± 0.52	82.59 ± 3.31	12.46 ± 0.50	8.61	0.86	0
0.75	34 (85)	16.09 ± 0.45	13.44 ± 0.42	84.53 ± 2.66	12.12 ± 0.47	9.61	1.32	0
1.00	34 (85)	15.97 ± 057	12.91 ± 0.47	82.75 ± 3.15	12.15 ± 0.46	5.98	0.74	0.03

Notes: * Taken from Welsh *et al*. 1987.
a) Adjusted on the assumption that corpora lutea cannot be less than the number of implants.
b) Statistical analysis was performed on these data after application of the Freeman-Tukey arc-sine transformation.

The quality of the dataset for triphenyl phosphate is variable. However, there are valid data for all endpoints except chromosomal aberrations, *in vivo* genotoxicity and carcinogenicity.

A margin of safety of at least 600-fold is considered necessary to provide reassurance against effects on human health. This is based on applying uncertainty factors for interspecies variation (10), intraspecies variation (10) and extrapolation and from sub-acute to chronic (6).

4.4.11 Derivation of PNEC for secondary poisoning

The lowest NOAEL from the mammalian studies is 0.1 per cent, or 1,000 mg/kg bw/day, as described above. An assessment factor of 300 is appropriate for this study, of 35 days duration. Hence, the PNEC for secondary poisoning is 3.3 mg/kg.

No avian toxicology data relevant to the derivation of a $PNEC_{oral}$ were identified for triphenyl phosphate.

4.5 Hazard classification

4.5.1 Classification for human health

Triphenyl phosphate is not currently included on Annex I of Directive 67/548/EEC for human health. According to the criteria of the EU, triphenyl phosphate does not need to be classified on the basis of its acute toxicity, skin and eye irritancy, sensitizing potential, or corrosivity to the skin or eyes. There are also no data to suggest that triphenyl phosphate should be classified in the EU for specific target organ systemic toxicity following repeated exposure and it is not classifiable as toxic to reproduction.

There are no data on effects via lactation and therefore it is not possible to make recommendations on classification for such effects. Also, there are insufficient data to characterise the carcinogenic potential of triphenyl phosphate and so recommendations for classification on carcinogenicity cannot be made.

Since the base level of testing for mutagenic potential is usually a gene mutation test in bacteria and an *in vitro* mammalian cell test capable of detecting chromosomal aberrations, there is a data gap for triphenyl phosphate. Without information on the potential for triphenyl phosphate to cause chromosomal aberrations, it is not possible to make an informed judgement on the classification of the substance for genotoxicity.

4.5.2 Classification for the environment

Triphenyl phosphate is currently classified as dangerous for the environment on Annex I of Directive 67/548/EEC as follows:

N:	Dangerous for the environment.
R50/53:	Very toxic to aquatic organisms. May cause long-term adverse effects in the aquatic environment.

Based on a fish BCF of around 420 l/kg, a 96-hour LC_{50} of 0.31 mg/l for fish and a 96-hour EC_{50} for invertebrates of 0.25 mg/l, the above classification is appropriate for triphenyl phosphate.

4.6 PBT assessment

The criteria for persistence (P and vP), bioaccumulation potential (B and vB) and toxicity (T) included in the TGD are shown in Table 4.8.

Persistence: triphenyl phosphate is readily biodegradable (Section 3.1.1), which is considered equivalent to a half-life of less than 40 days in freshwater. Hence, the substance does not meet the P criterion.

Bioconcentration: a BCF value of 420 is selected from the available data in Section 3.1.3. Hence, the substance does not meet the B criterion.

Toxicity: the lowest NOEC value from the available tests is 0.037 mg/l; an estimated NOEC for invertebrates is also available, 0.032 mg/l. The substance is not currently classified for mammalian effects. The substance does not meet the T criterion.

The overall conclusion is that the substance does not meet any of the PBT criteria.

Table 4.8 Criteria for identification of PBT and vPvB substances

Criterion	PBT criteria	vPvB criteria
P	Half-life above 60 days in marine water or above 40 days in freshwater* or half-life above 180 days in marine sediment or above 120 days in freshwater sediment*	Half-life above 60 days in marine water or freshwater or above 180 days in marine or freshwater sediment
B	BCF above 2,000	BCF above 5,000
T	Chronic NOEC below 0.01 mg/l or classification for certain human health end points, or endocrine disrupting effects	Not applicable

Notes: * For the purpose of marine environment risk assessment half-life data in freshwater and freshwater sediment can be overruled by data obtained in marine conditions.

5 Risk characterisation

This section identifies the potential risks that triphenyl phosphate might pose for the freshwater and marine aquatic compartments, terrestrial compartment, air compartment and predatory organisms through secondary poisoning. The risk characterisation is performed by comparing the PECs with the PNECs to derive a risk characterisation ratio (RCR). An RCR of less than one implies that any risk resulting from that level of exposure is acceptable. An RCR above one implies a potential risk, and all such values are highlighted in bold in the following tables. Annex C considers the effect of a faster hydrolysis rate on the overall conclusions.

5.1 Aquatic compartment

5.1.1 Surface water

The PNEC for surface water is estimated to be 0.74 μg/l. The resulting PEC/PNEC ratios are summarised in Table 5.1.

Table 5.1 Summary of PEC/PNEC ratios for surface water

Scenario		Predicted concentration (μg/l)	PEC/PNEC
Production of triphenyl phosphate		0.30 and 0.03	0.4 and 0.04
Printed circuit boards	Compounding	5.0	**6.76**
	Conversion	0.52	0.7
	Combined compounding and conversion	5.51	**7.45**
Thermo-plastics/ styrenics	Compounding	14.5	**19.6**
	Conversion	1.48	**2.0**
	Combined compounding and conversion	15.9	**21.6**
Thermosets and epoxy resins	Compounding	5.0	**6.76**
	Conversion	0.52	0.7
	Combined compounding and conversion	5.51	**7.45**
Photographic film	Compounding	4.98	**6.73**
	Conversion	0.52	0.71
	Combined compounding and conversion	5.51	**7.45**
Regional sources		0.01	0.01

The ratios are above one for the combined compounding and conversion and compounding steps in printed circuit boards, thermosets and epoxy resins, and photographic film. For thermoplastics/styrenics the ratios are above one for all three steps. The estimated local emissions from these sources are dominated by the predicted loss from the compounding step, although the conversion step alone also shows a risk for thermoplastics and styrenics.

Further information is needed on process emissions to refine the PECs for these scenarios. The sensitivity analysis in Annex C suggests that a faster hydrolysis rate than assumed here would only have a small impact on surface water concentrations.

No risks to surface water are identified from production sites and regional sources based on the approach taken.

If the alternative PNEC is used (3.2 µg/l, derived when the estimated NOEC for invertebrates is included in the dataset), the conversion-only step for thermoplastics no longer shows a risk (in fact there are no risks for any conversion-only steps). Consideration could be given to carrying out a further toxicity test (such as a long-term *Daphnia magna* reproduction test) to confirm the estimated value. However, the predicted *Daphnia magna* NOEC assumed in the assessment (0.032 mg/l) is close to the lowest NOEC determined experimentally for fish (0.037 mg/l) and so further toxicity testing alone would not remove the risks identified in this assessment (at best, further testing would only increase the PNEC from 0.74 µg/l to 3.7 µg/l, and could even reduce the value if the experimentally determined NOEC for *Daphnia magna* is significantly lower than that predicted). Hence, improvement of the emission estimates is considered the best way to refine this part of the assessment.

As noted in Section 3.1.3, releases to water are assumed to arise from the deposition of volatile emissions onto the floor and surfaces of the process area and subsequent washing. Information from two users of triphenyl phosphate (actual uses were not identified) indicates that floor cleaning in some cases may not involve the use of water (but rather sawdust), and that in other cases washing may take place intermittently. This may mean that the calculations of exposure levels are not applicable to all sites in the industry. However, no specific information on emissions or resulting concentrations was provided, and so calculated concentrations are retained as a reasonable worst case. The information does suggest that any risk reduction measures may need to be site-specific.

The highest river concentration measured in a recent Environment Agency monitoring exercise is 0.434 µg/l, which is below the range of PNECs, although the sample locations are not necessarily closely associated with processes that use the substance.

Triphenyl phosphate in other products

As noted in Section 1.2.3, triphenyl phosphate is present in most of the other aryl phosphate flame retardants assessed in this series. PECs in this assessment are based on emissions from the use of triphenyl phosphate itself, and so do not include releases from the use of other substances. To consider these possible sources, the fraction of other substances which may be triphenyl phosphate (from Table 1.1) was applied to overall emissions estimated for other substances in their respective assessments. This does not take any account of the different properties, which may mean that triphenyl phosphate has higher or lower releases than the other substance, but is intended to provide an order of magnitude estimate. The result is that air emissions would increase by 35 per cent, while emissions to water and soil would be trebled. The resulting PEC for regional water is still only a small fraction of the PEC values, and would have no influence on the results. None of the regional concentrations (water, soil or sediment) give rise to PEC/PNEC ratios above one. So although the additional release is significant in terms of quantity, it is not significant in terms of the resulting concentrations.

5.1.2 Waste water treatment

The PNEC for waste water treatment processes is 0.51 mg/l. The resulting PEC/PNEC ratios are summarised in Table 5.2.

Table 5.2 Summary of risk characterisation ratios for waste water treatment plants

Scenario		Predicted concentration (mg/l)	PEC/PNEC
Production of triphenyl phosphate		0.01	0.02
Printed circuit boards	Compounding	0.05	0.1
	Conversion	5.17×10^{-3}	0.01
	Combined compounding and conversion	0.06	0.11
Thermo-plastics/ styrenics	Compounding	0.15	0.29
	Conversion	0.01	0.03
	Combined compounding and conversion	0.16	0.32
Thermosets and epoxy resins	Compounding	0.05	0.1
	Conversion	5.17×10^{-3}	0.01
	Combined compounding and conversion	0.06	0.11
Photographic film	Compounding	0.05	0.1
	Conversion	5.21×10^{-3}	0.01
	Combined compounding and conversion	0.06	0.11

The PEC/PNEC ratios are all less than one. On this basis, no risk to waste water treatment plants would be expected from the production and use of triphenyl phosphate. The alternative PNEC considered in Section 4.1.6 is higher, and so this too would not show any risks.

The highest WWTP effluent concentration measured in a recent Environment Agency monitoring exercise is 0.122 µg/l, which is well below the PNEC, although the sample locations are not necessarily closely associated with processes that use the substance.

5.1.3 Sediment

The PNEC for sediment is estimated to be 0.16 mg/kg wet weight by the equilibrium partition method. The substance is expected to sorb significantly to sediments (the log K_{oc} used is 10,000), and uptake via the solid phase is possible. This is not accounted for in the equilibrium partition method. In these circumstances, the TGD indicates that an extra factor of 10 should be applied to PEC/PNEC ratios to take account of possible uptake in the solid phase[9]. Resulting PEC/PNEC ratios are summarised in Table 5.3.

All of the ratios are above one with the exception of production (at site 2) and the regional assessment. Without the additional factor of 10, the results would be the same as for the water compartment.

Further information on exposure identified for the aquatic compartment would also have an influence on the risk ratios here. However, the extra factor of 10 used for sediment means that the emission estimates would have to be significantly reduced to remove all of the concerns.

[9] The TGD indicates a threshold of log K_{ow} of five for the extra factor. Although the log K_{ow} of triphenyl phosphate is below this, the measured K_{oc} is equivalent to a higher K_{ow} (a value of 5.8 would be required to estimate the K_{oc} of 10,000 using the phosphates equation for K_{oc} in the TGD). Hence, in this case the extra factor of 10 is indicated.

The sensitivity analysis in Annex C suggests that a faster hydrolysis rate than assumed in this assessment could have a significant effect on the local and regional sediment PECs. It may therefore be possible to refine the PECs by carrying out further testing[10] to investigate the actual degradation (mineralization) half-life in sediment under relevant environmental conditions.

Toxicity data for sediment organisms would allow a PNEC to be derived directly, and would remove the need for the additional factor. It is likely that three long-term tests on sediment organisms would be required.

A possible alternative approach would be to measure the sorption coefficient of triphenyl phosphate in a range of sediments, to investigate the sediment properties which govern the sorption and to determine a suitable value for this assessment. This might indicate that the additional factor of ten is unnecessary, depending on the outcome.

Table 5.3 Summary of risk characterisation ratios for sediment

Scenario		Predicted concentration (mg/kg wet wt.)	PEC/PNEC ratio
Production of triphenyl phosphate		0.06 and 6.29×10^{-3}	**4.01** and 0.39
Printed circuit boards	Compounding	1.09	**67.6**
	Conversion	0.11	**7.03**
	Combined compounding and conversion	1.2	**74.5**
Thermo-plastics/ styrenics	Compounding	3.16	**196**
	Conversion	0.32	**20**
	Combined compounding and conversion	3.48	**216**
Thermosets and epoxy resins	Compounding	1.09	**67.6**
	Conversion	0.11	**7.03**
	Combined compounding and conversion	1.2	**74.5**
Photographic film	Compounding	1.09	**67.3**
	Conversion	0.11	**7.09**
	Combined compounding and conversion	1.2	**74.5**
Regional sources		2.43×10^{-3}	0.15

5.2 Terrestrial compartment

The PNEC for soil is estimated as 0.13 mg/kg wet weight by the equilibrium partition method. The substance is expected to sorb significantly to soils (the log K_{oc} used is 10,000), and uptake via the solid phase is possible. This is not accounted for in the equilibrium partition method. In these circumstances, the TGD indicates that an extra factor of 10 should be applied to the PEC/PNEC ratios to take account of possible uptake in the solid phase. The resulting PEC/PNEC ratios are given in Table 5.4.

All of the ratios are above one with the exception of production and the regional assessment.

Further information on exposure identified for the aquatic compartment would also have an influence on the risk ratios here. However, the extra factor of 10 used for soil means that the emission estimates would have to be significantly reduced to remove all concerns. Like sediment, the sensitivity analysis in Annex C suggests that a faster hydrolysis rate

[10] The half-life determined in such a test would be the result of degradation by both biodegradation and hydrolysis to biodegradable substances.

than assumed here could have a significant effect on the local and regional soil PECs. It may therefore be possible to refine the PECs by carrying out further testing to investigate the actual degradation (mineralization) half-life in soil under relevant environmental conditions.

Toxicity data for terrestrial organisms would allow a PNEC to be derived directly, and would remove the need for the additional factor. As for sediment, it is likely that three long-term tests would be required.

5.3 Atmosphere

No information is available on the toxicity of triphenyl phosphate to plants and other organisms exposed via air. The low vapour pressure of the substance means that volatilisation to the atmosphere is likely to be limited and the resulting concentrations are likely to be low (predicted concentrations are all below 2×10^{-4} mg/l). The possibility of triphenyl phosphate contributing to atmospheric effects such as global warming and acid rain is thus likely to be small. In addition, as the substance does not contain halogen atoms, it will not contribute to ozone depletion.

Table 5.4 Summary of risk characterisation ratios for the terrestrial compartment

Scenario		Predicted concentration (mg/kg)	PEC/PNEC ratio
Production of triphenyl phosphate		negligible[a]	negligible[a]
Printed circuit boards	Compounding	1.58	**121**
	Conversion	0.16	**12.3**
	Combined compounding and conversion	1.74	**133**
Thermo-plastics/ styrenics	Compounding	4.58	**350**
	Conversion	0.47	**35.6**
	Combined compounding and conversion	5.04	**386**
Thermosets and epoxy resins	Compounding	1.58	**121**
	Conversion	0.16	**12.3**
	Combined compounding and conversion	1.74	**133**
Photographic film	Compounding	1.57	**120**
	Conversion	0.16	**12.4**
	Combined compounding and conversion	1.74	**133**
Regional sources	Agricultural soil	7.71×10^{-5}	<0.01
	Natural soil	1.9×10^{-5}	<0.01
	Industrial soil	5.07×10^{-3}	0.39

Notes: a) Sewage sludge from the production sites is not applied to land.

5.4 Secondary poisoning

The PNEC for secondary poisoning is estimated to be 3.33 mg/kg food. The resulting PEC/PNEC ratios are summarised in Table 5.5.

Risk characterisation ratios are all below one, apart from the combined compounding and conversion steps, and the conversion step for thermoplastics and styrenics. This indicates a generally low risk of secondary poisoning through the terrestrial food chain from the current uses of triphenyl phosphate. The scenarios that pose a potential risk have ratios

that are only slightly above one, so revised emission estimates may refine the assessment. As noted in Section 3.3.4, triphenyl phosphate has been found in the blubber of dolphins (*Torsions truncates*) which may indicate possible accumulation via the food chain (Kuehl and Haebler 1995, cited in OECD 2002). This appears to be in contrast with the data on uptake in fish, and the OECD report indicates that the substance is degraded by hydrolysis in rat liver to diphenyl phosphate. In the absence of any information on specific exposure conditions (the dolphins were collected during an unusual mortality event in the Gulf of Mexico; the cause of death was not established), this information cannot be used further in this assessment.

Table 5.5 Summary of risk characterisation ratios for secondary poisoning

Scenario		Fish food chain		Earthworm food chain	
		Predicted concentration (mg/kg)	**PEC/PNEC ratio**	**Predicted concentration (mg/kg)**	**PEC/PNEC ratio**
Production of triphenyl phosphate		0.06 and 7.64×10^{-3}	0.02 and <0.01	negligible	<0.01
Printed circuit boards	Compounding	0.14	0.04	1.81	0.54
	Conversion	0.02	<0.01	0.19	0.06
	Combined compounding and conversion	0.15	0.05	2.0	0.6
Thermo-plastics/ styrenics	Compounding	2.5	0.75	5.25	**1.58**
	Conversion	0.26	0.08	0.53	0.16
	Combined compounding and conversion	2.76	0.83	5.78	**1.73**
Thermosets and epoxy resins	Compounding	7.4×10^{-3}	<0.01	1.81	0.54
	Conversion	0.11	0.03	0.19	0.06
	Combined compounding and conversion	0.95	0.29	2.0	0.6
Photo-graphic film	Compounding	0.86	0.26	1.8	0.54
	Conversion	0.09	0.03	0.19	0.06
	Combined compounding and conversion	0.95	0.29	2.0	0.6

5.5 Risks to human health following environmental exposure

A NOAEL of 70 mg/kg bw/day in rats was identified in Section 4.4.10 as the most appropriate value for use in this assessment. A margin of safety of at least 600 is considered necessary to provide sufficient reassurance against effects on human health with this result (see Section 4.4.10). The estimated human exposures via the environment were calculated in Section 3.3.4 and are included in Table 5.6 together with the resulting margins of exposure.

Table 5.6 Margin of exposure between daily human doses and the NOAEL (70 mg/kg bw/day)

Scenario		Total daily human intake (mg/kg bw/day)	Margin of exposure
Production of triphenyl phosphate		2.43×10^{-4} and 1.96×10^{-5}	288,100 and 3,571,000
Printed circuit boards	Compounding	0.02	3,500
	Conversion	1.72×10^{-3}	40,700
	Combined compounding and conversion	0.02	3,500
Thermoplastics/ styrenics	Compounding	0.05	1,400
	Conversion	7.68×10^{-3}	9,114
	Combined compounding and conversion	0.06	1,170
Thermosets and epoxy resins	Compounding	0.02	3,500
	Conversion	2.92×10^{-3}	23,970
	Combined compounding and conversion	0.02	3,500
Photographic film	Compounding	0.02	3,500
	Conversion	2.69×10^{-3}	26,020
	Combined compounding and conversion	0.02	3,500
Regional sources		9.73×10^{-6}	7,194,000

All of the margins of exposure are above the required value, and so no risks are indicated for any scenario.

5.6 Marine risk assessment

Although a PEC/PNEC approach can be applied to the marine environment, there are additional concerns which may not be adequately addressed using the above methods. Chief among these concerns is the possibility that hazardous substances may accumulate in parts of the marine environment. The effects of such accumulation are unpredictable in the long term, and once such accumulation has occurred it may be practically difficult to reverse. The properties which lead to substances behaving in this way also lead to greater uncertainty in estimating exposures and/or effect concentrations, and so make a quantitative risk assessment more difficult. In order to identify substances which are likely to behave in this way, criteria have been developed relating to the persistence, accumulation and toxicity of the substance. The first part of the marine assessment is therefore a comparison of the properties of the substance with these criteria. This is presented in Section 4.6.

PEC values for the marine assessment are presented in Sections 3.3.1 and 3.3.4. These were calculated using EUSES. PNECs for marine aquatic species are included in Section 4.1.6. The PNEC for secondary poisoning for the marine environment is the same as that for the freshwater fish and terrestrial food chains (Section 4.4.11). The resulting PEC/PNEC ratios are in Table 5.7. The production step is not included in these calculations as the production sites do not discharge to the marine environment.

Table 5.7 Summary of risk characterisation ratios for marine environment

Scenario		PEC/PNEC ratio			
		Marine water	**Marine sediment**	**Fish food chain**	**Top predators**
Printed circuit boards	Compounding	**81.6**	**816**	0.05	<0.01
	Conversion	**8.33**	**83.3**	<0.01	<0.01
	Combined compounding and conversion	**89.9**	**899**	0.05	0.01
Thermoplastics/ styrenics	Compounding	**236**	**2,360**	0.91	0.18
	Conversion	**24**	**240**	0.09	0.02
	Combined compounding and conversion	**260**	**2,600**	0.99	0.2
Thermosets and epoxy resins	Compounding	**81.6**	**816**	<0.01	<0.01
	Conversion	**8.33**	**83.3**	0.04	<0.01
	Combined compounding and conversion	**89.9**	**899**	0.34	0.07
Photographic film	Compounding	**81.2**	**812**	0.31	0.06
	Conversion	**8.4**	**84**	0.03	<0.01
	Combined compounding and conversion	**89.9**	**899**	0.34	0.07

Risks are indicated for all scenarios for marine waters and marine sediments. The regional concentrations in marine waters (8.85×10^{-4} μg/l) and sediments (1.1×10^{-4} mg/kg wet weight) do not indicate a risk.

Further information on process emissions indicated for the freshwater environment would also help to refine these conclusions. More specifically for the marine assessment, information on whether any of these processes avoid discharging to the marine environment or do so only after effluent treatment (the calculations above assume a direct discharge to the marine environment without waste water treatment), would be useful.

Testing on freshwater organisms would also affect the marine PNEC, although the same comments on this as in Section 5.1.1 apply. Testing on sediment organisms would be of more value for the sediment assessment. There is also the possibility of testing on marine species, which would allow the assessment factor to be reduced.

The size of the PEC/PNEC ratios suggests that no one part of the further information requirements would be sufficient on its own to reduce the ratios to below one.

No risks are indicated for marine predators.

6 Conclusions

Triphenyl phosphate can enter the environment from its production and use, and from the use of articles made from materials containing it. Based on the available information, potential risks are identified for all of the life cycle steps for one or more of the protection goals. The overall conclusions are summarised in Table 6.1 in a simplified form. In particular, the different steps in the use of each material have been combined here. Section 5 should be consulted for the detailed results.

Table 6.1 Summarised potential environmental risks identified for triphenyl phosphate

Life cycle stage	Surface water	Sediment	WWTP	Air	Soil	Aquatic food chain	Terrestrial food chain	Marine water	Marine sediment
Production	-	✸	-	-	-	-	-	-	-
Printed circuit boards	✸	✸	-	-	✸	-	-	✸	✸
Thermoplastics/styrenics	✸	✸	-	-	✸	-	✸	✸	✸
Thermosets/epoxy resins	✸	✸	-	-	✸	-	-	✸	✸
Photographic film	✸	✸	-	-	✸	-	-	✸	✸
Regional	-	-	-	-		-	-		

No risks are identified for humans exposed via the environment, or for marine food chain exposure.

Monitoring data confirm the presence of triphenyl phosphate in surface water and sediments; the levels found are generally lower than those calculated, although the measurements cannot be related to specific activities.

The potential risks identified here could be reassessed following additional work, in particular:

- Collation of further site and industry-specific information on releases of triphenyl phosphate from use in the different types of plastic materials indicated. This work could include:
    - Improved description of practices at sites using triphenyl phosphate, to determine the realism of the emission estimates, ideally through surveys of representative sites.
    - Targeted monitoring to confirm or replace the calculated PEC values (especially in water, sediment and WWTP sludge). Further environmental monitoring for triphenyl phosphate is taking place in England and Wales, at one WWTP per Environment Agency region, in both final effluent and associated receiving waters (6 samples at 4 week intervals). The sites are different from those used in the previous monitoring exercise. Sampling is expected to take place from September 2008 until March 2009.
    - Information on the fate of sludges from sites using the substance.
    - Surveys to locate user sites, especially in relation to marine discharges.
- Long-term sediment and soil organism testing.

- Measurement of the sorption coefficient of triphenyl phosphate in a range of sediments.
- Studies on the fate of the substance in WWTP (municipal and industrial).
- Further testing to investigate the actual degradation (mineralization) half-life in sediment and soil under relevant environmental conditions.

A possible risk to sediment organisms is identified for production (for only one of two sites). This conclusion could be refined through the sediment testing indicated above, but it is more appropriate for the local control authority to consider this outcome.

There may be opportunities to read across information and test results from this substance to the other aryl phosphates assessed in this group (and vice versa). Therefore, the additional work indicated above should be considered in relation to that proposed for other members of the group. The overview document should be consulted for more information on this.

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8 Glossary of terms

Term	Description
Biochemical oxygen demand (BOD)	A measure of degradation potential
Bioconcentration factor (BCF)	A measure of chemical uptake, being the ratio between the concentration in an organism and the concentration in an environmental compartment (usually water)
CAS number (no.)	An identifying code number assigned to chemicals by the Chemical Abstract Services. The CAS number is a generally recognised identification reference for a chemical; a substance can have more than one such number
Inherently biodegradable	Some potential for environmental degradation to carbon dioxide and water, and so on, as measured by laboratory screening tests involving microorganisms
Lowest observed effect concentration (LOEC)	The lowest concentration in a toxicity test that gives rise to adverse effects (relative to a control)
Median effective concentration (EC_{50})	The concentration in a toxicity test at which a particular effect is observed in half of the organisms exposed for a specified time
Median lethal loading (LL_{50})	The loading of substance in a water-accommodated fraction that leads to death in half of the organisms exposed for a specified time
Median lethal concentration/dose (LC/D_{50})	The concentration in a toxicity test that can be expected to cause death in half of the organisms exposed for a specified time
No observed effect concentration (NOEC)	The highest concentration in a toxicity test that does not give rise to adverse effects (relative to a control)
Octanol-water partition coefficient (K_{ow})	This parameter gives an indication of the partitioning behaviour of a substance between water and lipid-containing materials such as cell membranes or organic matter in soils and sediments
Readily biodegradable	Rapid environmental degradation to carbon dioxide and water, and so on, as measured by laboratory screening tests involving microorganisms

9 Abbreviations

Acronym	Description
ABS	Acrylonitrile-styrene-butadiene
ASTM	American Society for Testing and Materials
B	Bioaccumulative
BCF	Bioconcentration factor
BMF	Biomagnification factor
BOD	Biochemical oxygen demand
bw	Bodyweight
CAS	Chemical Abstract Services
CMR	Carcinogenic, mutagenic and toxic to reproduction
CPDPP	Cumylphenyl diphenyl phosphate
DEHP	Di(2-ethylhexyl)phthalate
DIN	Deutsche Industrie Norm (German norm)
EC	European Communities
EC_{50}	Median effect concentration
EC_x	As EC_{50}, but for x% effect; x usually being 0, 10, or 100
ECB	European Chemicals Bureau
EEC	European Economic Communities
EINECS	European Inventory of Existing Commercial Chemical Substances – this lists all chemical substances that were supplied to the market prior to 18th September 1981
EPA	Environmental Protection Agency (USA)
ESD	Emission Scenario Document
ESR	The Existing Substances Regulation – Council Regulation (EEC) 793/93 on the evaluation and control of the risks of 'existing' substances.
EU	European Union
EUSES	European Union System for the Evaluation of Substances (software tool in support of the TGD on risk assessment)
HPLC	High performance liquid chromatography
HPTLC	High performance thin layer chromatography
HPV	High Production Volume (supply above 1,000 tonnes per year)
IUCLID	International Uniform Chemical Information Database: contains non-validated tonnage, use pattern, property and hazard information for chemicals, submitted by industry under the Existing Substances Regulation (ESR)
K_{oc}	Organic carbon normalised distribution coefficient
K_{ow}	Octanol-water partition coefficient

Kp	Solids-water partition coefficient
L(E)C_{50}	Median lethal (effect) concentration
LD_{50}	Median lethal dose
LL_{50}	Median lethal loading
LO(A)EL	Lowest observed (adverse) effect level
LOEC	Lowest observed effect concentration
log K_{ow}	Log of the octanol-water partition coefficient (K_{ow})
NO(A)EL	No observed (adverse) effect level
NOEC	No observed effect concentration
NPDPP	Nonylphenyl diphenyl phosphate
n.t.p.	Normal temperature and pressure
OECD	Organisation for Economic Cooperation and Development
P	Persistent
PBT	Persistent, bioaccumulative and toxic
PEC	Predicted environmental concentration
pH	Logarithm (to the base 10) of the hydrogen ion concentration [H+]
pK_a	Logarithm (to the base 10) of the acid dissociation constant
PNEC	Predicted no effect concentration
Ppt	Parts per trillion
SCAS	Semi-continuous activated sludge unit
SIAR	SIDS Initial Assessment Report, OECD
SIDS	Screening Information Data Set, OECD
TGD	Technical Guidance Document
TLC	Thin layer chromatography
TOC	Total organic carbon
TPP	Triphenyl phosphate
TSCA	Toxic Substances Control Act (USA)
USEPA	Environmental Protection Agency, USA
UV	Ultraviolet region of the electromagnetic spectrum
vB	Very bioaccumulative
vP	Very persistent
vPvB	Very persistent and very bioaccumulative
w/w	Weight per weight ratio
wt	Weight
wwt	Wet weight
WWTP	Waste water treatment plant

10 Data collection and peer review process

This report has been produced using publicly available data gathered and assessed by the contractor for the Environment Agency. Additional information has been submitted voluntarily by member companies of the Phosphate Ester Flame Retardant Consortium (PEFRC, http://www.pefrcnet.org/), and the Environment Agency would like to thank them for their cooperation.

The Environment Agency has been keen to ensure that the data used in this report are as complete and accurate as possible. Original reports and literature articles for key studies were retrieved and assessed for reliability wherever possible (it is clearly indicated where this was not the case).

The main scientific literature search was performed in 2002, with some further limited searching to consider specific issues up to 2007.

Drafts of this report have been circulated to key stakeholders in UK and European Industry for comment on several occasions, as well as members of the UK and European chemical regulatory community in July 2007. The Advisory Committee on Hazardous Substances has also provided helpful comments as part of its own deliberations on this substance group (their last review was in September 2007).

In addition, certain technical aspects of the report were peer-reviewed by an independent expert group set up by the Environment Agency for this purpose in April 2007. The experts were:

- Dr Kay Fox (independent consultant);
- Dr Tamara Galloway (University of Plymouth).

Their comments have not been published but are available on request. All comments received have been addressed in the final report where appropriate.

The Institute for Environment and Health wrote the human health effects assessment, and this was peer-reviewed by colleagues at the Health and Safety Executive and Health Protection Agency.

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