European Union Summary Risk Assessment Report

4,4'-ISOPROPYLIDENEDIPHENOL

(BISPHENOL-A)

CAS No: 80-05-7

EINECS No: 201-245-8

NOTE TO THE READER

Please note that this summary contains **only information** from the risk assessment report of 4,4’-isopropylidenediphenol (bisphenol-A) **from 2003** and not from the update from 2008.

To see all information, please refer to the "Final RAR" (Addendum 2008 combined with Volume 2003).

EUROPEAN COMMISSION

JOINT RESEARCH CENTRE

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Summary Risk Assessment Report

2003 **Special Publication I.03.149**

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SUMMARY RISK ASSESSMENT REPORT

Final report, 2003

United Kingdom

This document has been prepared by the UK rapporteur on behalf of the European Union. The scientific work on the environmental part was prepared by the Building Research Establishment Ltd (BRE), under contract to the rapporteur.

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Date of Last Literature Search:	**1998**
Review of report by MS Technical Experts finalised:	**2001**
Final report:	**2003**

(The last full literature survey was carried out in 1998 - targeted searches were carried out subsequently).

PREFACE

This report provides a summary, with conclusions, of the risk assessment report of the substance 4,4'-isopropylidenediphenol (bisphenol-A) that has been prepared by the United Kingdom in the context of Council Regulation (EEC) No. 793/93 on the evaluation and control of existing substances.

For detailed information on the risk assessment principles and procedures followed, the underlying data and the literature references the reader is referred to the comprehensive Final Risk Assessment Report (Final RAR) that can be obtained from the European Chemicals Bureau[1]. The Final RAR should be used for citation purposes rather than this present Summary Report.

[1] European Chemicals Bureau – Existing Chemicals – http://ecb.jrc.it

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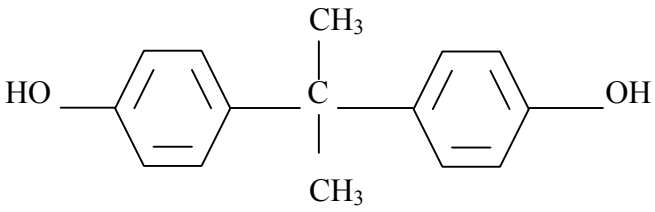
1 GENERAL SUBSTANCE INFORMATION

1.1 IDENTIFICATION OF THE SUBSTANCE

CAS Number: 80-05-7
EINECS Number: 201-245-8
IUPAC Name: 2,2-bis(4-hydroxyphenyl)propane
Common Name: bisphenol-A (abbreviation BPA)
Molecular weight: 228.29
Molecular formula: $C_{15}H_{16}O_2$
Structural formula:



1.2 PURITY/IMPURITIES, ADDITIVES

The purity of bisphenol-A is stated as being 99-99.8% depending upon the manufacturer. Impurities typically include phenol (<0.06%), ortho and para isomers of bisphenol-A (<0.2%) and water (<0.2%).

1.3 PHYSICO-CHEMICAL PROPERTIES

Table 1.1 Physico-chemical properties for bisphenol-A

Parameter	Value
Physical state at normal temperature and pressure	White solid flakes or powder with a mild phenolic odour
Melting point	155-157°C at atmospheric pressure
Boiling point	~360°C with decomposition at atmospheric pressure
Relative density	1.1-1.2 kg/m^3 at 25°C
Vapour pressure	$5.3 \cdot 10^{-9}$ kPa at 25°C
Solubility in water	300 mg/l used at ntp
Partition coefficient (Log Kow)	3.4
Flash point	circa 207°C
Autoflammability	circa 532°C
Oxidising properties	Not an oxidising agent

1.4 CLASSIFICATION

The classification and labelling of bisphenol-A has recently been discussed (January 2002) and provisional agreement has been reached, as follows:

Classification: Repr. Cat. 3; R62
Xi; R37-41, R43

Labelling: Xn
R37-41-43-62
S2-26-36/37-39-46

R62 states: Possible risk of impaired fertility

Toxicity to reproduction category 3 is for substances which cause concern for human fertility, generally on the basis of:

1) results in appropriate animal studies which provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which is not a secondary consequence of the other toxic effects, but where the evidence is insufficient to place the substance in Category 2;

2) other relevant information.

Xi indicates	"irritant"
Xn indicates	"harmful"
R37 states:	Irritating to respiratory system
R41 states:	Risk of serious damage to eyes
R43 states:	May cause sensitisation by skin contact
S(2) states:	Keep out of the reach of children
S26 states:	In case of contact with eyes, rinse immediately with plenty of water and seek medical advice
S36/37 states:	Wear suitable protective clothing and gloves
S39 states:	Wear eye/face protection
S46 states:	If swallowed, seek medical advice immediately and show this container or label

No classification is proposed for the environment based upon the current criteria for classification. It is considered that the observed effects at low concentrations in longer-term studies justify the application of suitable risk and safety phrases to this substance, but that further discussion is needed on what these should be, and more generally on how to include such effects in the classification system.

2 GENERAL INFORMATION ON EXPOSURE

Production

Four companies within the EU manufacture bisphenol-A at six production sites. The total amount manufactured within the EU is approximately 700,000 tonnes/year (based on data up to 1999). Exports from and imports into the EU are 2,000-25,000 tonnes/year and 3,000-8,000 tonnes/year, respectively, depending upon the basis of the estimation. The total EU consumption is estimated at 690,000 tonnes/year.

Uses

Bisphenol-A is primarily used as an intermediate in the production of polycarbonate (71.1%) and epoxy resins (25.0%). Minor uses include as an intermediate for phenoplast resins, unsaturated polyester resin, alkyloxylated bisphenol-A, polyols/polyurethanes and modified polyamides, for can coatings, thermal paper, tyre and brake fluid manufacture, and in PVC production and processing.

Legislative controls

No environmental legislative controls specific to bisphenol-A are known. In recognition of the ability of bisphenol-A to migrate from food contact materials into food, a Specific Migration Limit (SML) of 3 mg bisphenol-A per kg food (3 ppm), has been set for the protection of the consumer in the EU.

3 ENVIRONMENT

3.1 ENVIRONMENTAL EXPOSURE

Environmental releases

The assessment considers the releases of bisphenol-A from its production, and its use in the production of a range of plastic materials - epoxy resins, polycarbonates, PVC and phenoplast resins. Where relevant, emissions from the processing of plastics and the use of plastic articles have been estimated. Emissions from the production and recycling of thermal paper are also included. The estimated releases are based on specific information from industry where available and considered representative, together with default assumptions. The main route of environmental exposure is from use in the thermal paper and PVC industries.

Environmental fate

Bisphenol-A released to the atmosphere is likely to be degraded by reaction with hydroxyl radicals, with a calculated half-life of around 0.2 days. It is not thought to contribute to low-level ozone formation or act as a greenhouse gas.

Abiotic degradation in water is negligible but bisphenol-A does biodegrade. The results from a range of tests suggest that bisphenol-A is best classified as readily biodegradable, possibly following a short period of adaptation.

Experimental data and calculated partition coefficients suggest that bisphenol-A is moderately adsorbed to soil, sludge and sediment upon release to the environment. The air-water partitioning coefficient is low suggesting that volatilisation is unlikely to be a significant removal mechanism for bisphenol-A from water systems. Precipitation of bisphenol-A from the atmosphere is expected to be low due to the relatively low atmospheric emissions and relatively short atmospheric lifetime.

Experimental data suggest bisphenol-A has a low potential for bioaccumulation in aquatic species.

Environmental concentrations

Environmental exposure of bisphenol-A occurs during its production and subsequent use. Predicted environmental concentrations (**Table 3.1**) have been calculated using site-specific data supplemented by defaults as specified by the Technical Guidance Document (TGD) and Emission Scenario Documents where no specific data were available.

Table 3.1 Summary of PECs

	PEC_{stp} (mg/l)	PEC_{water} (µg/l)	$PEC_{sediment}$ (mg/kg wet wt) a)	PEC_{air} (mg/m³) a)	$PEC_{agricultural\ soil}$ (mg/kg wet wt) a)
Site specific					
Production	0.00069-0.19	0.12-0.44	0.007	$3.61 \cdot 10^{-4}$	na
Epoxy Resin Manufacture	0-0.03	0.12-1.32	0.02	na	0.152
Thermal Paper Manufacture	0.0007-0.47	0.19-1.12	0.02	na	na
PVC Production	<0.005	0.62	0.01	na	na
Generic scenarios					
Polycarbonate bottle washing	$1.6 \cdot 10^{-6}$	0.12	0.002	na	na
Thermal paper recycling	2.28	230	3.71	na	1.03
Phenoplast cast resin processing	0.0144	1.56	0.025	na	0.0065
PVC – Inhibitor during production process	3.33	333	5.4	na	1.5
PVC – Anti-oxidant during processing	0.00154	0.27	0.004	na	0.0008
PVC – Preparation of additive packages	0.127	12.8	0.2	na	0.0575
PVC – Anti-oxidant in plasticiser production	0.019	2.0	0.033	na	0.0087
PVC – Plasticiser use	0.00107	0.23	0.0036	na	0.0006
Regional	na	0.12	$1.6 \cdot 10^{-3}$	$2.08 \cdot 10^{-10}$	$9.9 \cdot 10^{-5}$

na not applicable
a) PECs for soil and air are only calculated for uses with environmental releases to those compartments for the generic scenarios, and for site specific scenarios are calculated for the site with the highest environmental release
b) production of PVC resin using bisphenol-A was voluntarily phased out in Europe by the end of 2001 by the major users

3.2 EFFECTS ASSESSMENT

Aquatic compartment (incl. sediment)

Aquatic toxicity data are reported for freshwater and marine fish, Daphnia and algae. The results of the key standard tests are included in **Table 3.2**.

The possible effects of bisphenol-A on endocrine systems have been investigated in a number of species. In fish, effects have been observed with some species on vitellogenin synthesis, secondary sexual characteristics and spermatogenesis. The lowest concentration at which effects have been reported is 1 µg/l, but the quality of the data is not considered suitable to define the PNEC.

Amongst invertebrates, snails appear to be sensitive to bisphenol-A. Effects including increased egg production have been observed in several species, with effects below 1 µg/l reported although the study is not considered suitable to define the PNEC.

Table 3.2 Summary of environmental effects data

Species	Endpoint	Value
Fish		
Pimephales promelas	96-hour LC_{50}	4.6 mg/l
	164-day NOEC (Reproduction)	16 µg/l
Aquatic invertebrates		
Daphnia magna	48-hour EC_{50} (Immobilisation)	10.2 mg/l
	21-day NOEC (Reproduction)	>3.146 mg/l
Aquatic algae		
Selenastrum capricornutum (Fresh water species)	96-hour EC_{50} (Cell count)	2.73 mg/l
Skeletonema costatum (Marine species)	96-hour EC_{50} (Cell count)	1.1 mg/l
	96-hour EC_{10} (Cell count)	0.40 mg/l

An assessment factor of 10 leads to a PNEC of 1.6 µg/l, based on the fish NOEC. To take account of the indications of effects below this level, the risk assessment also considers a "conservative" PNEC of 0.1 µg/l, based on possible effects on spermatogenesis in fish at 1 µg/l. The biological consequences of the effects that the conservative PNEC is based on are currently unknown.

Toxicity data for sediment- and soil-dwelling organisms are not available, but an extrapolation from aquatic toxicity data can be made using the equilibrium partitioning method for screening risk assessment purposes.

Terrestrial compartment

There are no toxicity tests results available for terrestrial species. Therefore the $PNEC_{soil}$ will be derived from the $PNEC_{water}$ by a partitioning equilibrium in line with the recommendations of the TGD.

For bisphenol-A this gives a $PNEC_{soil}$ of 23 µg/kg wet weight based on the conventional aquatic PNEC and 1.3 µg/kg wet weight based on the conservative aquatic PNEC.

Atmosphere

There are no known biotic or abiotic effects of bisphenol-A in the atmosphere, and in particular effects on plants due to atmospheric exposure are unknown. Based on structural considerations, it is unlikely to be an ozone depleter or greenhouse gas, nor is it thought to contribute to low-level ozone formation. It is therefore not possible to derive a PNEC.

Secondary poisoning

A $PNEC_{oral}$ of 33 mg/kg food has been derived for the secondary poisoning assessment from a NOAEL of 50 mg/kg body weight (based on a reduction in litter size) from a three-generation multi-dose level feeding study in rats.

3.3 RISK CHARACTERISATION

Table 3.3 summarises the outcome of the risk assessment for life cycle stages considered and the various environmental compartments.

Table 3.3 Summary of risk assessment conclusions

Life cycle stage [1]	Wastewater treatment plants	Surface water [2] [3]	Soil	Air	Secondary poisoning
Bisphenol-A production	ii	**i**	ii	ii	ii
Epoxy resin production	ii	**i**	**i**	ii	ii
Phenoplast cast resin production	ii	**i**	**i**	ii	ii
Thermal paper production	ii	**i**	ii	ii	ii
Thermal paper recycling	ii	**iii**	**i**	ii	ii
PVC – Inhibitor during production process [4]	ii	**iii**	**i**	ii	ii
PVC – Anti-oxidant during processing	ii	**i**	**i**	ii	ii
PVC – Preparation of additive packages	ii	**iii**	**i**	ii	ii
PVC – Anti-oxidant in plasticiser production	ii	**iii**	**i**	ii	ii
PVC – Plasticiser use	ii	**i**	**i**	ii	ii

1) Four uses only take place on sites where bisphenol-A is produced. Emissions from these processes are included in the site-specific emissions for bisphenol-A production and so are not separately identified. These are: Polyol/polyurethane production; Brake fluid manufacture; Polyamide production; Polycarbonate production.
2) For bisphenol-A the sediment concentrations and the sediment PNECs are both derived from the corresponding PEC and PNEC values from water using the equilibrium partition method. The PEC/PNEC ratios will therefore be the same as the surface water and the same conclusions will apply.
3) The uses identified as a risk based on the "traditional" higher PNEC (1.6 μg/l) are marked conclusion (iii). All other uses give rise to risk when using the "conservative" PNEC and are marked conclusion (i).
4) Production of PVC resin using bisphenol-A was voluntarily phased out in Europe by the end of 2001 by the major users.

In addition the background regional concentration is indicated as a risk with the "conservative" PNEC.

The work required to address the **conclusion (i)** comprises further studies on snails, and work on spermatogenesis in fish, in both cases to clarify the level at which effects occur. The need to investigate effects in terrestrial organisms should be reconsidered once the results of the aquatic studies and any risk management are available (since the principal source of soil exposure is from the spreading of sewage sludge).

4 HUMAN HEALTH

4.1 HUMAN HEALTH (TOXICITY)

4.1.1 Exposure assessment

Occupational exposure

Occupational exposure to bisphenol-A potentially occurs in the following scenarios:

- manufacture of bisphenol-A,
- manufacture of polycarbonate (PC) and of articles produced from PC,
- manufacture of epoxy resins and moderated epoxy resins,
- use of bisphenol-A in PVC manufacture (this use is being phased out),
- manufacture of liquid epoxy paints, lacquers and powder coatings and their use,
- manufacture of thermal papers,
- manufacture of tin-plating additive,
- manufacture of tetrabrominated flame retardants (TBBA).

The total number of persons occupationally exposed to bisphenol-A is not known, but due to its widespread use in epoxy resins and PC it is expected to be thousands. However, the exposure is likely to be negligible in many cases as the residual bisphenol-A in epoxy resins and PC is low. Bisphenol-A has a low-vapour pressure and therefore no significant exposure to vapour occurs. Exposure will be in the form of inhalation or ingestion of dust and by skin contact with flakes or powder.

The highest inhalation exposures were reported for bisphenol-A manufacture, for which a reasonable worst-case 8-hour TWA was estimated at 5 $mg \cdot m^{-3}$. Generally, for all exposure scenarios, short-term exposures rarely exceeded 10 $mg \cdot m^{-3}$.

Dermal exposures were highest for bisphenol-A manufacture and manufacture of epoxy resins (based on EASE model estimations and taking into account the skin surface area exposed). The reasonable worst-case exposure estimate for these scenarios was 1 $mg \cdot cm^{-2} \cdot day^{-1}$.

Consumer exposure

There is no direct consumer exposure to bisphenol-A. However, polycarbonates and epoxy resins that are manufactured using bisphenol-A have many applications in consumer goods, such as food contact containers, adhesives and protective coatings. Potential consumer exposure can arise only under conditions where residual monomer in the polymer matrix becomes available for exposure or where breakdown of the polymer occurs, to generate additional bisphenol-A monomer.

For polycarbonate food contact applications, estimates of daily ingestion of bisphenol-A were derived for infants exposed via feeding bottles and for young children (1.5-4.5 years) exposed via tableware. Intake values of 0.035 mg/day (1-2 month baby), 0.05 mg/day (4-6 month baby) and 0.01 mg/day (young children) were derived. For epoxy resin food contact applications, estimated intake values of 0.1 mg/day for adults, 0.2 mg/day for young children and 0.04 mg/day for infants (6-12 months) were derived. In addition, a very worst-case intake of 0.5 mg/day was estimated for consumption of wine stored in vats lined with epoxy resins.

Potential exposure could also arise from consumer use of epoxy resin based paints ($2 \cdot 10^{-5}$ mg/event for inhalation and 0.0036 mg/event for dermal exposure), wood filler

(0.009 mg/event) and adhesives (0.014 mg/event). Bisphenol-A is a component of restorative materials such as fissure sealant, used in dentistry. However, any exposure from dental fissure sealants is likely to be an infrequent acute event. No other significant sources of consumer exposure were identified.

Humans exposed via the environment

Human intake via the environment is $1.78 \cdot 10^{-5}$ mg·kg^{-1}·day^{-1} at the regional level. The highest local exposure of 0.06 mg·kg^{-1}·day^{-1} arises during the use of bisphenol-A as an inhibitor in PVC production.

Combined exposure

The worst-case combined exposure would be to someone exposed via the environment near to a PVC production plant, who is also exposed via food contact materials. This would result in a maximum combined exposure of 0.069 mg·kg^{-1}·day^{-1}.

4.1.2 Effects assessment

Bisphenol-A is well absorbed orally, but there is limited dermal absorption and no information on inhalation absorption. Following uptake, it is removed rapidly from the blood, with extensive first pass metabolism following oral absorption, limiting bioavailability by this route. Bisphenol-A distributes at least to the liver, bone marrow, testes and the foetus. Enterohepatic circulation occurs. Metabolism mainly involves glucuronide conjugation. Elimination is rapid, primarily via the faeces. There is some evidence from animal studies to suggest that low levels of bisphenol-A may be excreted in breast milk. There is probably no significant bioaccumulation.

Bisphenol-A is of low acute toxicity. It is not a skin irritant, but it can cause serious damage to the eyes and is irritating to the respiratory tract. Bisphenol-A can produce skin sensitisation responses in humans. There is no information on respiratory sensitisation potential. Repeated inhalation studies in rats indicate slight inflammation of the upper respiratory tract epithelium, with a NOAEL of 10 mg/m^3 in a 13-week study. In a 2-year dietary study, a NOAEL of 74 mg·kg^{-1}·day^{-1} has been established for rats. In mice, the liver is the target organ, with a LOAEL (2-year study) of 120 mg·kg^{-1}·day^{-1} in males and a NOAEL of 650 mg·kg^{-1}·day^{-1} in females.

Bisphenol-A appears to have aneugenic potential *in vitro*, possibly mediated by disruption of microtubule formation, but does not have mutagenic potential *in vivo*. The evidence suggests that bisphenol-A does not have carcinogenic potential.

Bisphenol-A has endocrine modulating activity in a number of *in vitro* and *in vivo* screening assays, with a potency generally 3 to 5 orders of magnitude less than that of oestradiol. The effects of bisphenol-A on fertility and reproductive performance have been investigated in good quality studies in rats and mice. An adverse effect on fertility has been seen in both species, with a NOAEL of 50 mg·kg^{-1}. No evidence that bisphenol-A is a developmental toxicant was observed in standard development studies in rats and mice. In a rat multigeneration study, there was evidence of delayed development at a dose level producing maternal toxicity; no maternal or fetal effects were seen at 50 mg·kg^{-1}·day^{-1}. However, additionally, some studies have investigated the potential of bisphenol-A to affect male reproductive tract development in rats and mice. Conflicting results have been reported in these studies, with some studies reporting adverse effects at doses in the µg/kg range. The majority of EU member states felt that these low dose findings could not be dismissed, but

disagreed on how these studies should be used, if at all, in the risk characterisation for this endpoint. The disagreements were based on differing views about the uncertainties surrounding the reproducibility of the findings and their biological significance, if any, to human health.

Following referral to the Competent Authorities it was agreed that further work was required to resolve the uncertainties surrounding the potential for bisphenol-A to produce adverse effects on development at low doses. In the interim, a provisional NOAEL of 50 $mg \cdot kg^{-1} \cdot day^{-1}$ is used in the risk characterisation.

4.1.3 Risk characterisation

Workers

There are concerns for eye and respiratory tract irritation, for liver effects following repeated exposure and for reproductive toxicity during the manufacture of bisphenol-A and the manufacture of epoxy resins: **conclusion (iii)**. There are concerns for skin sensitisation in all occupational exposure scenarios where there is the potential for skin contact: **conclusion (iii)**.

In relation to developmental toxicity, further work is required to resolve the uncertainties and refine the risk characterisation for the manufacture of PC and articles made from PC, powder coating manufacture and use, and in the manufacture of PVC, thermal paper, tin plating additives and TBBA: **conclusion (i)**.

Consumers

There are no concerns in relation to the potential for eye or respiratory tract irritation, skin sensitisation, repeated exposure toxicity or effects on fertility arising from consumer exposure: **conclusion (ii)**. Further information is required in relation to the potential for bisphenol-A to produce adverse effects on development: **conclusion (i)**.

Humans exposed via the environment

There are no concerns for effects on fertility or repeated exposure toxicity: **conclusion (ii)**. Irritation and sensitisation are of low concern where exposure is dissipated throughout the environment. More information on the potential for bisphenol-A to cause developmental effects is required: **conclusion (i)**.

Combined exposure

There are no concerns for fertility or for repeated exposure toxicity: **conclusion (ii)**. More information on the potential for bisphenol-A to cause developmental effects is required: **conclusion (i)**.

4.2 HUMAN HEALTH (PHYSICO-CHEMICAL PROPERTIES)

Given the low vapour pressure at normal temperatures, lack of flammability and the general stability, the risks arising from the physico-chemical properties are small. In common with many organic materials, the finely powdered material is a significant dust explosion hazard. However, this appears to be well known within the manufacturing industry and it is considered that there are adequate controls for this risk in place. Overall, the risk to human health from physicochemical properties is low.

5 RESULTS

5.1 ENVIRONMENT

The environmental risk assessment for bisphenol-A considers all of the life cycle steps identified in Section 3.1.

Results

Conclusion (iii) There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

This conclusion applies to the following scenarios for the water and sediment compartments:

- Thermal paper recycling,
- Use as an inhibitor in PVC production,
- Preparation of additive packages for PVC processing,
- Use as a anti-oxidant in the production of plasticisers for use in PVC processing.

For these uses further refining the PNEC for water will not change the outcome of the assessment. Although these scenarios are referred to as generic in the exposure section, the PEC estimates are based on data from the industry and use areas and are considered representative. It appears unlikely that the provision of further information would alter the conclusions. The use of bisphenol-A in the manufacture of PVC resin is due to be phased out in Europe by the end of 2001 under a voluntary agreement by industry.

Conclusion (i) There is a need for further information and/or testing.

This conclusion applies to the following scenarios for the water and sediment compartments:

- Bisphenol-A production, [2]
- Epoxy resin production,
- Thermal paper production,
- Phenoplast cast resin processing,
- Use as a anti-oxidant in PVC processing,
- Use as a plasticiser in PVC processing,
- Regional concentration.

These scenarios do not give rise to a risk when the PNEC based on the standard endpoint of egg hatchability is used. However, if a "conservative" PNEC based on research studies indicating effects on snails and sperm development in fish is used, all scenarios and the regional concentration give rise to a risk. There is considerable uncertainty over the validity of the lower PNEC. Recent research studies on snails have raised the possibility of effects at still lower concentrations. If these studies were to be used as the basis for a PNEC derivation, the much lower value would have implications for possible risk reduction measures. It is therefore considered that further studies on the toxicity of bisphenol-A to snails are needed, to

[2] Four uses only take place on sites where bisphenol-A is produced. Emissions from these processes are included in the site-specific emissions for bisphenol-A production and so are not separately identified. These are:
Polyol/polyurethane production
Brake fluid manufacture
Polyamide production
Polycarbonate production

provide a more robust basis for the derivation of a PNEC. The re-investigation of the effects on sperm development in fish is also required. The apparently elevated levels measured in sediment will also be considered when the aquatic assessment is refined.

Conclusion (i) also applies to the following uses of bisphenol-A for the terrestrial compartment:

- Epoxy resin production,
- Phenoplast cast resin processing,
- Thermal paper recycling,
- Use as an inhibitor in PVC production,
- Preparation of additive packages for PVC processing,
- Use as an anti-oxidant in the production of plasticisers for use in PVC processing,
- Use as an anti-oxidant in PVC processing,
- Use as a plasticiser in PVC processing,
- Regional concentration.

The equilibrium partitioning method has been used, so testing on terrestrial organisms could revise the PNEC. It is currently not clear what testing would be appropriate, as the most sensitive effects in aquatic organisms appear to be related to endocrine disruption. It is proposed to await the outcome of the further work on aquatic organisms before deciding on testing for the terrestrial compartment. In addition, the UK Department of Environment, Food & Rural Affairs is conducting research into endocrine disruption in the earthworm *Eisenia andrei* and bisphenol-A is one of the test compounds. The project aim is to develop molecular markers of exposure to, and population level effects of, endocrine disruption for use in field and laboratory studies. It is expected that this work will provide relevant information, to a timescale compatible with that of the aquatic tests.

A revision of the PNECoral value will also be considered if additional information on the interpretation of mammalian developmental data becomes available as a result of further studies being conducted for the human health assessment.

Conclusion (ii) There is at present no need for further information and/or testing and for risk reduction measures beyond those which are being applied already.

This conclusion applies to microorganisms in wastewater treatment plants and to the air compartment for all scenarios. It also applies to the terrestrial compartment for the following:

- Bisphenol-A production,
- Thermal paper manufacture.

This conclusion also applies to the water, sediment and terrestrial compartments for the following uses:

- Unsaturated polyester production,
- Can coating production,
- Tyre manufacture,
- Alkoxylated bisphenol-A production,
- Tetrabromobisphenol-A production and use,
- Phenoplast cast resin production.

For these six scenarios, emissions are negligible and PECs have not been calculated in this assessment (these processes are either completely dry, or any aqueous effluent produced is disposed of through incineration).

5.2 HUMAN HEALTH

5.2.1 Human health (toxicity)

The key health effects of exposure to bisphenol-A are eye and respiratory tract irritation, skin sensitisation, repeat dose toxicity to the respiratory tract, effects on the liver and reproductive toxicity (effects on fertility and on development).

Workers

Conclusion (iii) There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

This conclusion applies to the manufacture of bisphenol-A and the manufacture of epoxy resins, in relation to concerns for eye and respiratory tract irritation, effects on liver and toxicity for reproduction (effects on fertility and on development). In addition, there are concerns for skin sensitisation in all occupational exposure scenarios where there is the potential for skin contact.

Conclusion (i) There is a need for further information and/or testing.

This conclusion applies to the manufacture of PC and of articles from PC, powder coating manufacture and use and the manufacture of PVC, thermal paper, tin plating additives and TBBA. Further research is needed to resolve the uncertainties surrounding the potential for bisphenol-A to produce adverse effects on development at low doses.

Conclusion (ii) There is at present no need for further information and/or testing and for risk reduction measures beyond those which are being applied already.

This conclusion is reached in relation to eye and respiratory tract irritation, effects on liver following repeated exposure and effects on fertility for workers in the industry sectors of the manufacture of polycarbonate, manufacture of articles from polycarbonate, powder coatings manufacture and use, manufacture of PVC, thermal paper manufacture, manufacture of tin plating additive and manufacture of TBBA. This conclusion is also reached in relation to repeated dose toxicity to the respiratory tract for all scenarios.

Consumers

Conclusion (i) There is a need for further information and/or testing.

Further research is needed to resolve the uncertainties surrounding the potential for bisphenol-A to produce adverse effects on development at low doses.

Conclusion (ii) There is at present no need for further information and/or testing and for risk reduction measures beyond those which are being applied already.

This conclusion applies to all exposure scenarios, in relation to eye and respiratory tract irritation, skin sensitisation, repeated dose toxicity to the liver and for effects on fertility.

Humans exposed via the environment

Conclusion (i) There is a need for further information and/or testing.

Further research is needed to resolve the uncertainties surrounding the potential for bisphenol-A to produce adverse effects on development at low doses.

Conclusion (ii) There is at present no need for further information and/or testing and for risk reduction measures beyond those which are being applied already.

Given the low levels of exposure for both the regional and local exposure scenarios, there are no concerns for repeated dose toxicity to the liver or for effects on fertility.

Combined exposure

Conclusion (i) There is a need for further information and/or testing.

Further research is needed to resolve the uncertainties surrounding the potential for bisphenol-A to produce adverse effects on development at low doses.

Conclusion (ii) There is at present no need for further information and/or testing and for risk reduction measures beyond those which are being applied already.

This conclusion is reached in relation to repeated dose toxicity and effects on fertility.

5.2.2 Human health (risks from physico-chemical properties)

Conclusion (ii) There is at present no need for further information and/or testing and for risk reduction measures beyond those which are being applied already.

There are no significant risks from physicochemical properties.