European Union Risk Assessment Report

TRIS[2-CHLORO-1-(CHLOROMETHYL)ETHYL] PHOSPHATE (TDCP)

CAS No: 13674-87-8

EINECS No: 237-159-2

RISK ASSESSMENT

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RISK ASSESSMENT

May 2008

Ireland (lead) and United Kingdom

Rapporteur for the risk assessment of TDCP is Ireland (lead) and United Kingdom

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Final report:	**2008**

Foreword

This Draft Risk assessment Report is carried out in accordance with Council Regulation (EEC) 793/93[1] on the evaluation and control of the risks of "existing" substances. "Existing" substances are chemical substances in use within the European Community before September 1981 and listed in the European Inventory of Existing Commercial Chemical Substances. Regulation 793/93 provides a systematic framework for the evaluation of the risks to human health and the environment of these substances if they are produced or imported into the Community in volumes above 10 tonnes per year.

There are four overall stages in the Regulation for reducing the risks: data collection, priority setting, risk assessment and risk reduction. Data provided by Industry are used by Member States and the Commission services to determine the priority of the substances which need to be assessed. For each substance on a priority list, a Member State volunteers to act as "Rapporteur", undertaking the in-depth Risk Assessment and recommending a strategy to limit the risks of exposure to the substance, if necessary.

The methods for carrying out an in-depth Risk Assessment at Community level are laid down in Commission Regulation (EC) 1488/94[2], which is supported by a technical guidance document[3]. Normally, the "Rapporteur" and individual companies producing, importing and/or using the chemicals work closely together to develop a draft Risk Assessment Report, which is then presented at a Meeting of Member State technical experts for endorsement. The Risk Assessment Report is then peer-reviewed by the Scientific Committee on Toxicity, Ecotoxicity and the Environment (CSTEE) which gives its opinion to the European Commission on the quality of the risk assessment.

This Draft Risk Assessment Report is currently under discussion in the Competent Group of Member State experts with the aim of reaching consensus. During the course of these discussions, the scientific interpretation of the underlying scientific information may change, more information may be included and even the conclusions reached in this draft may change. The Competent Group of Member State experts seek as wide a distribution of these drafts as possible, in order to assure as complete and accurate an information basis as possible. The information contained in this Draft Risk Assessment Report does not, therefore, necessarily provide a sufficient basis for decision making regarding the hazards, exposures or the risks associated with the priority substance.

This Draft Risk Assessment Report is the responsibility of the Member State rapporteur. In order to avoid possible misinterpretations or misuse of the findings in this draft, anyone wishing to cite or quote this report is advised to contact the Member State rapporteur beforehand.

[1] O.J. No L 084, 05/04/199 p.0001 – 0075

[2] O.J. No L 161, 29/06/1994 p. 0003 – 0011

[3] Technical Guidance Document, Part I – V, ISBN 92-827-801 [1234]

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Note regarding EU enlargement

Work on this risk assessment began before enlargement of the EU to 27 member states in 2006. All tonnage data, and references to the 'EU' in this risk assessment report, therefore refer to the former EU of 15 Member States.

Reasons for prioritisation for risk assessment

Chlorinated alkyl phosphate esters (particularly TCPP) were identified as possible substitutes for pentabromodiphenyl ether in the risk reduction strategy for that substance (EC 2001). A risk assessment of this group is therefore important as that substance has now been banned from the EU market. It has since become clear, from discussion with the industry, that in the EU these chemicals are not direct replacements for pentaBDE, and that changes in TCPP consumption are linked mostly with the decline in TCEP use and increase in the market for polyurethane (PUR) generally (pers. comm., 1st March 2004). They appear to be relatively persistent substances, and there is some human health concern (the substance manufacturers have voluntarily classified TDCP as a category 3 carcinogen).

Four substances in this group are listed in IUCLID, and were ranked according to the EURAM method (EU Risk Ranking Method); their priority scores (PS) are shown in **Table i**.

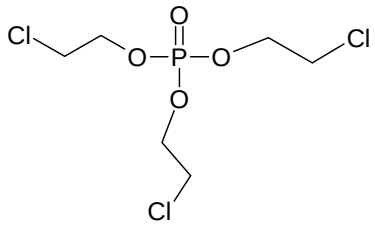
Table i Priority scores of chlorinated alkyl phosphate esters

Name	CAS No.	Aquatic PS	Health PS
tris(2-chloroethyl) phosphate (TCEP)	115-96-8	15.3	61.2
tris(2-chloro-1-methylethyl) phosphate (TCPP)	13674-84-5	10.5	58.1
tris[2-chloro-1-(chloromethyl)ethyl] phosphate (TDCP)	13674-87-8	42.6	39.8
2,2-bis(chloromethyl)trimethylene bis(bis(2-chloroethyl)phosphate) (V6)	38051-10-4	34.2	39.8

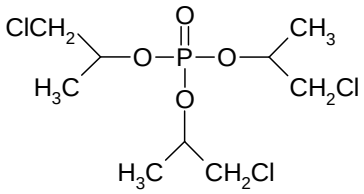
Note: A priority score of 100 is the highest priority.

The substance structures are shown below.

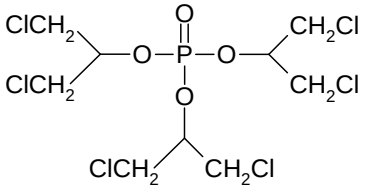
Tris(2-chloroethyl) phosphate (TCEP)



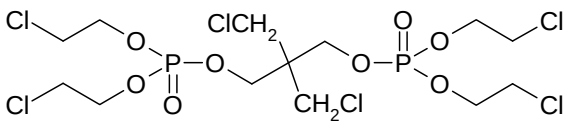
Tris(2-chloro-1-methylethyl) phosphate (TCPP)[4]



Tris[2-chloro-1-(chloromethyl)ethyl] phosphate (TDCP)



2,2-Bis(chloromethyl)trimethylene bis(bis(2-chloroethyl)phosphate) (V6)



[4] Structure shown is the main isomer present

A previous assessment in 1995 concluded that there was insufficient exposure and hazard information to perform a risk assessment for some of these substances ("The Flame Retardants Project Final Report, KEMI Report No. 5/96"). V6 in particular was data poor. A 1998 OECD SIDS assessment concluded that TCPP was a low priority for further work (the environmental exposure was said to be 'minimal') (UNEP, 1999). Nevertheless, the pentabromodiphenyl ether risk reduction strategy indicated that TCPP use is increasing owing to new technologies in both rigid and flexible foam systems. An in depth ESR assessment is a useful check of OECD conclusions.

The substances TDCP, TCPP and V6 are therefore good candidates for a concurrent assessment in view of their similar use pattern and structures. Other flame retardant substances (from Environmental Health Criteria document (WHO, 1998) or UK review) within this group that do not appear to be EU HPV substances are shown in **Table ii**. The substance with CAS number 6145-73-9 is an isomer of TCPP and is present in the commercial substance. The substance with CAS number 78-43-3 is an isomer of TDCP. Both of these CAS numbers may have in the past been erroneously applied to the respective substances.

Table ii Chlorinated alkyl phosphate esters which are not EU HPV substances

Name	CAS No.	Status	Data availability (according to EHC)	Use
tris(2-chloro-1-propyl) phosphate	6145-73-9	LPV	poor	rigid urethane foams
tetrakis(2-(chloroethyl)ethylene-diphosphate	33125-86-9	Believed not to be available[1]	poor	"plastics"
tris(2,3-dichloro-1-propyl) phosphate	78-43-3	Believed not to be available[1]	poor	"plastics"

Note: None of these substances as such are commercially available from, or produced as isolated products by, EU manufacturers.

[1]These substances are not listed as either HPV or LPV substances by the ECB.

TCPP, TDCP and V6 all appear on the 4th ESR Priority List and their risk assessments have been completed by Ireland (leading the work and assessing human health) and the UK (leading on the environmental assessment). See HSA/EA 2008a and b for the other assessments. TCEP, from the 2nd ESR Priority List, has been assessed by Germany. There is some overlap between the substances in both properties and use pattern, and hence this risk assessment report contains references to the assessments of these other substances.

Physicochemical, environmental and ecotoxicological data for all four substances are presented together for comparison in Appendix C to this risk assessment.

OVERALL RESULTS OF THE RISK ASSESSMENT[5]

CAS Number: 13674-87-8
EINECS Number: 237-159-2
IUPAC Name: Tris[2-chloro-1-(chloromethyl)ethyl] phosphate

Environment

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

Conclusion (ii) applies at the regional scale in all compartments and to all current local life cycle stages. TDCP does not meet all of the PBT criteria (it meets the screening criteria for P or vP).

It is understood that the life cycle stages associated with Confidential Use C (i.e. C1a, C1b and C2) are no longer relevant in Europe, on the basis of industry information. Should it be the case that supply for Use C resumes in future, conclusion (i) or (iii) would apply for some compartments and some life cycle stages.

Human Health

Human health (toxicity)

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

A conclusion (i) "on hold" applies to effects on female fertility for all worker exposure scenarios.

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

Conclusion (ii) applies to all worker exposure scenarios for the endpoints acute toxicity, irritation, sensitisation, mutagenicity, effects on male fertility and developmental toxicity.

Conclusion (ii) applies to typical dermal exposure and inhalation exposures, both reasonable worst case and typical, during the manufacture of TDCP (worker scenario 1), manufacture of flexible PUR foam – slabstock (worker scenario 2a) and manufacture of flexible PUR foam – moulded (worker scenario 2b) in relation to repeated dose toxicity and carcinogenicity.

Conclusion (ii) also applies to all other worker exposure scenarios (worker scenarios 3, 4 and 5) for both reasonable worst case and typical exposures in relation to repeated dose toxicity and carcinogenicity.

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5 Conclusion (i) There is a need for further information and/or testing.
Conclusion (ii) There is at present no need for further information and/or testing and no need for risk reduction measures beyond those which are being applied already.
Conclusion (iii) There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

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

Conclusion (iii) applies to reasonable worst case dermal exposure during the manufacture of TDCP (worker scenario 1), manufacture of flexible PUR foam – slabstock (worker scenario 2a), and manufacture of flexible PUR foam – moulded (worker scenario 2b) in relation to repeated dose toxicity and carcinogenicity.

Consumers

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

A conclusion (i) "on hold" applies to effects on female fertility for all consumer exposures.

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

Conclusion (ii) applies to all consumer exposure scenarios for the endpoints acute toxicity, irritation, sensitisation, repeated dose toxicity, mutagenicity, carcinogenicity, effects on male fertility and developmental toxicity.

Humans exposed via the environment

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

A conclusion (i) "on hold" applies to effects on female fertility for both regional and local exposures.

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

Conclusion (ii) applies to both regional and local exposures for the endpoints acute toxicity, irritation, sensitisation, repeated dose toxicity, mutagenicity, carcinogenicity, effects on male fertility and developmental toxicity.

Combined exposure

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

A conclusion (i) "on hold" applies to effects on female fertility for combined exposure.

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

Conclusion (ii) applies to combined exposure for the endpoints acute toxicity, irritation, sensitisation, repeated dose toxicity, mutagenicity, carcinogenicity, effects on male fertility and developmental toxicity.

Human health (physico-chemical properties)

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

Conclusion (ii) applies to all endpoints**.**

CONTENTS

EUSES Calculations can be viewed as part of the report at the website of the European Chemicals Bureau: http://ecb.jrc.it

TABLES & FIGURES

Note: There are two further Annexes to this risk assessment report:

Confidential use pattern and exposure annex: this annex presents confidential details of the release scenarios for production and uses of TDCP, used in the risk assessment. It is available to competent authorities as part of the ESR review process, on request from the Rapporteur. This is referred to in the text as the 'Confidential Annex'.

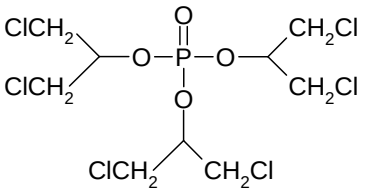
Confidential analytical data annex: this presents confidential details of the purity and impurities of commercially available TDCP together with various spectra. It is available to competent authorities as part of the ESR review process, on request from the Rapporteur. This is referred to in the text as the 'confidential annex of compositional data'.

The Rapporteur can provide the confidential annexes on request, as appropriate.

1 GENERAL SUBSTANCE INFORMATION

1.1 IDENTIFICATION OF THE SUBSTANCE

CAS Number: 13674-87-8
EINECS Number: 237-159-2
IUPAC Name: Tris[2-chloro-1-(chloromethyl)ethyl] phosphate
Molecular formula: $C_9H_{15}Cl_6O_4P$
Structural formula:



Molecular weight: 430.91
Synonyms[6]: 2-Propanol, 1,3-dichloro-, phosphate (3:1)
Tris(1,3-dichloro-2-propyl) phosphate
Tris(1-chloromethyl-2-chloroethyl) phosphate
1,3-Dichloro-2-propanol phosphate (3:1)
Phosphoric acid, tris(1,3-dichloro-2-propyl)ester
TDCP: this common acronym is used throughout this report

Smiles notation O=P(OC(CCl)CCl)(OC(CCl)CCl)OC(CCl)CCl

1.2 PURITY/IMPURITIES, ADDITIVES

Purity

TDCP is 93 – 99.9% pure (w/w).

Impurities:

The impurity profile of the commercial product TDCP is specific to individual manufacturers. Details are given in the confidential annex of compositional data. The impurity profile does differ between suppliers but the impurity content is low. Their structures do not suggest that they would have had a strong influence on any of the test results.

Additives

No additives are used.

[6] For the sake of simplicity, company trade names are not listed here, since they may be subject to change.

1.3 PHYSICO-CHEMICAL PROPERTIES

The physico-chemical property values of TDCP that have been reviewed are summarised in **Table 1.1**. The values selected for use in the risk assessment are justified as follows:

Melting / freezing

The preferred value is <-20°C, which was obtained in a modern GLP study in accordance with Directive 92/69/EC (Cuthbert and Mullee, 2002a). TDCP exists as a supercooled liquid. It can crystallise at temperatures below 27°C. However, crystallisation is difficult to induce, and only occurs under abnormal conditions. Material stored in a refrigerator or freezer rarely crystallises.

Boiling

The preferred value is 326°C, although decomposition occurred, which was obtained in a modern GLP study in accordance with Directive 92/69/EC (Cuthbert and Mullee, 2002a).

Density at 20°C

The preferred value of the relative density is 1.513, which was obtained by the pycnometer method in a modern GLP study in accordance with Directive 92/69/EC (Cuthbert and Mullee, 2002a).

Vapour pressure

The preferred value is 5.6×10^{-6} Pa at 25°C, which was obtained by the vapour pressure balance method in a modern GLP study in accordance with Directive 92/69/EC (Tremain, 2002).

Surface tension

Based upon the chemical structure and the known physico-chemical properties of the substance, TDCP it is not expected to exhibit surface activity and there is no indication in use that it has 'surfactant-like' surface energy lowering potential.

A derogation in respect of this test was requested by industry and accepted by the TCNES.

Water solubility

The preferred value is 18.1 ± 1.1 mg/l at 20°C, which was obtained by the flask method in a modern GLP study in accordance with Directive 92/69/EC (Cuthbert and Mullee, 2002b). The ± value is the range of results reported.

Octanol-water partition coefficient

A modern GLP study according to the HPLC estimation method (A8, 92/69/EEC) has been carried out[7] (Cuthbert and Mullee, 2002b). In full compliance with the test guideline, TDCP

[7] It is noted in a later section of this report (3.1.3.2.1 – Adsorption) that K_{oc} values estimated using the HPLC method tend to be overestimated for TDCP and related substances. The problem with K_{oc} by HPLC estimation probably lies with the column type, a proposal which is discussed in more detail in Section 3. A different column type is used to measure log K_{ow} and there is no reason to suspect that a similar issue might occur. The K_{ow} by

was shown to have a log K_{ow} of 3.69 ± 0.36 at 20°C. The ± value is the 95% confidence interval.

Flash point (closed cup)

No closed cup result is available. Read-across from TCPP (HSA/EA, 2008a) suggests that the result is likely to be above 245°C

Flammability (contact with water)

Based on the known chemical and physical properties of the substance TDCP and its chemical structure, negative results are predicted for the following flammability test of Commission directive 84/449/EEC, hence it is considered justified to omit; Method A12 flammability in contact with moisture.

In contact with water or damp air, this substance will not react to produce hazardous gases.

A derogation in respect of this test was requested by industry and accepted by the TCNES.

Pyrophoric properties

The chemical substance of concern TDCP has use as a flame retardant, it does not support combustion.

In a fire, the mechanism of action of the flame retardant is primarily one by which phosphorus interferes with the combustion process, in the solid and gas phases, to produce a 'char' via formation of phosphoric acid. This char acts as a barrier and in turn prevents further oxygen reaching the site of combustion and the fire is 'starved' of fuel. The presence of the halogen – chlorine atoms – also aids this process in that they scavenge free radicals formed in the gaseous phase of the fire and consequently decreases the release of flammable volatiles.

The substance is not "extremely flammable" or "flammable" as referenced by the flash point (Method A9) and auto ignition temperature (Method A15).

A derogation in respect of this test was requested by industry and accepted by the TCNES.

Explosivity

Based upon the chemical structure of the substance TDCP and the known synthetic route of manufacture via an exothermic chemical reaction, there is no indication that this substance is thermodynamically unstable.

The DSC test used for boiling point measurement showed no exotherms.

The structure does not contain any of the more commonly known endothermic groups such as: azides, cyano-, dienes, acetylenic, peroxide or chlorate groups.

It is industry's opinion that this plus oxygen balance calculation supports the contention that this substance is unlikely to possess explosive properties.

A derogation in respect of this test was requested by industry and accepted by the TCNES.

HPLC agrees with shake flask data (of lower reliability) and with the EPIWIN prediction. The physicochemical data for the four related substances TCPP, TCEP, TDCP and V6 appear to be consistent and there is no reason to doubt any of the log K_{ow} values.

Autoignition temperature

A value of 513°C is stated, though the reliability of this result is not known (Akzo Nobel, 2000).

For TCPP, a single reliable GLP study (Tremain and Bartlett, 1994; see HSA/EA, 2008a) is available, giving an autoignition temperature of >400°C, although the composition of the sample used is not known. The value of 513°C is not inconsistent with this result.

Oxidising properties

By reference to the structural formula, it can be seen that TDCP contains highly electronegative atoms of chlorine, however the fact that these elements are only bonded to carbon and/or hydrogen renders it unlikely that this will confer oxidising properties on the substance. Furthermore, in order for a substance to have oxidising properties, a stable reduced form of the substance would need to exist, which is considered to be unlikely for TDCP.

Based upon information submitted in relation to A1 and A14 of Commission Directive 84/449/EEC and by analogy with similar existing chemicals, it is industry's opinion that the evidence supports the contention that the substance is unlikely to possess oxidising properties.

A derogation in respect of this test was requested by industry and accepted by the TCNES.

Henry's Law Constant

The Henry's Law constant has been derived from the values of vapour pressure and water solubility.

$$H = \frac{\text{Molecular weight} * \text{Vapour pressure}}{\text{Water solubility}}$$

A value of 1.24×10^{-4} Pa.m^3/mol is used in the risk assessment, based on EUSES adjustments of the properties for temperature dependence.

Table 1.1 Summary of physico-chemical properties

The values chosen for use in the risk assessment are presented in bold type.

Property	Value	Reliability[1]	Comments
Physical state	Liquid		
Melting point	27°C	(4) not assignable	Cited in a MITI report, origin unknown
	Melting point -58°C; freezing point -40°C	(4) not assignable	Melting point determination by DSC (compliant with OECD Guideline 102) Akzo-Nobel, Inc. 2001a and b, cited in USEPA, undated
	26.66°C	(4) not assignable	Akzo Nobel, 2003, cited in USEPA, undated
	<-20°C**	(1) valid without restriction. Modern GLP study in accordance with 92/69/EC	Cuthbert and Mullee, 2002a

Property	Value	Reliability[1]	Comments
Boiling point	>200°C	(4) not assignable	Cited in a MITI report. HSDB cites this value as peer-reviewed
	200°C (at 533 Pa)	(4) not assignable	Reduced pressure value Akzo Nobel, 2003, cited in USEPA, undated
	~326°C (decomp.)**	(1) valid without restriction. Modern GLP study in accordance with 92/69/EC	Boiled with decomposition. Cuthbert and Mullee, 2002a
Relative density	1.52	(4) not assignable	
	1.52	(4) not assignable	
	1.5022 at 20°C	(4) not assignable	Specific gravity. Budavari, 2001 (The Merck Index); Lewis, 2000 (Sax's Dangerous Properties of Industrial Materials), cited in USEPA, undated
	1.48 kg/l at 25°C	(4) not assignable	Bulk density. HSDB, 2003, cited in USEPA, undated
	1.513 at 20°C**	(1) valid without restriction. Modern GLP study in accordance with 92/69/EC	Cuthbert and Mullee, 2002a
Vapour pressure	12 Pa at 20°C	(4) not assignable	More information required.
	1.3 Pa at 30°C	(4) not assignable	Peer-reviewed reference, although value is much higher than might be expected for the main component.
	3.2 Pa at 20°C	(4) not assignable	Result certificate only
	5.6 x 10^{-6} Pa at 25°C **	(1) valid without restriction. Modern GLP study in accordance with 92/69/EC, vapour pressure balance method	The result is consistent with the chemical structure of the main component and the other properties, in particular the boiling point. Tremain, 2002.
Surface tension			No study available, but not expected to exhibit surface activity
Water solubility	110 mg/l	(4) not assignable	Cited in a MITI report
	7 mg/l at 24°C	(4) not assignable	HSDB cites this value as peer-reviewed (source AQUASOL database). May originate in a paper by Hollifield (1979)[2]
	100 mg/l at 30°C	(4) not assignable	May originate in a paper cited by Sasaki *et al*,.
	42 mg/l	(4) not assignable	Akzo-Nobel, Inc. 2001a and b, cited in USEPA, undated
	18.1 mg/l at 20°C**	(1) valid without restriction. Modern GLP study in accordance with 92/69/EC	Cuthbert and Mullee, 2002b.

Property	Value	Reliability[1]	Comments
Partition coefficient n-octanol/water (log value)	3.6 – 3.7	(4) not assignable	Cited in a MITI report.
	3.65	(4) not assignable	HSDB cites this value as peer-reviewed.
	3.76	(3) invalid.	Sasaki et al, 1981. Does not comply with good practice.
	1.59 – 3.65	(4) not assignable	Estimates
	2.4	(4) not assignable	Akzo-Nobel, Inc. 2001a and b, cited in USEPA, undated
	3.8	(4) not assignable	WHO, 1998, cited in USEPA, undated
	3.65	(2) valid with restrictions	Accepted calculation method (SRC KOWWIN v. 1.67)
	3.69 ± 0.36**	(1) valid without restriction. Modern GLP study in accordance with 92/69/EC, HPLC method	Cuthbert and Mullee, 2002b
Flash point		(4) not assignable	252°C is an open cup result.
	>107.22°C	(4) not assignable	Seta closed cup method Akzo Nobel, 2003, cited in USEPA, undated
Autoflammability (autoignition temperature)	**513°C**	(4) not assignable	Akzo Nobel, 2000
Flammability			Not expected to be flammable. Derogation accepted by TC NES
Explosive properties			Not expected to be explosive. Derogation accepted by TC NES
Oxidizing properties			Not expected to be oxidising. Derogation accepted by TC NES
Viscosity	1,800 cP at 25°C 2,200 cP at 0°C 540 cP at 40°C	(4) not assignable	Akzo Nobel, 2003, cited in USEPA, undated
Henry's law constant	1.24 x 10^{-04} $Pa.m^3/mol$ at 25°C	(4) not assignable	By calculation from VP and WS results

Studies marked ** were performed with a composite sample of purity 94.2%, derived from recent representative commercial products from the main producers.

[1] Klimisch code

[2] Hollifield (1979) sets out an approach to determine water solubility of various highly insoluble substances of environmental interest, based on plotting the turbidity of a series of solutions.

CLASSIFICATION

Current classification

Classification for the environment (N, R51-53) was agreed at EU level in 2005[8].

Basis of classification for the environment

Data presented in this report are consistent with the classification N R51-53 (toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment). This is based on the lowest acute E(L)C_{50} of 1.1 mg/l (fish) and the lack of ready biodegradability.

It is not possible to determine the relationship between the actual and nominal exposure concentrations from the fish test study report. Whilst it is possible that actual concentrations were lower, it is not known whether they were below 1 mg/l. However, this was a semi-static test and this regime would have favoured the maintenance of exposure concentrations. In addition, it is notable that the LC_{50} value is well below the water solubility value of 18.1 mg/l. It has therefore been used at face value for classification purposes.

The classification is also supported by QSAR estimates of fish acute LC_{50} (values range between 4.5 and 8.1 mg/l), and also by four other fish tests, that gave acute LC_{50} values in the range 1.4 to 5.1 mg/l (though these were nominal concentrations from static tests so are not wholly reliable).

Proposed classification

Basis of proposed classification for human health

It was agreed to classify TDCP as Carc. Cat 3; R40 in 2005[9].

The classification for effects on fertility and developmental toxicity are not yet agreed. Based on the information available, it is considered that there is no concern for effects on male fertility or developmental toxicity and therefore no classification for these endpoints is proposed.

The classification and labelling proposal for TDCP will be considered by the Risk Assessment Committee (RAC) in due course.

[8] Commission Working Group on the Classification and Labelling of Dangerous Substances Meeting on Environmental Effects of Existing Chemicals, Pesticides & New Chemicals September 28-30, 2005

[9] Commission Working Group on the Classification and Labelling of Dangerous Substances Meeting on the Health Effects of Pesticides, Existing Chemicals & New Chemicals November 14-18, 2005

GENERAL INFORMATION ON EXPOSURE

It should be noted that there are only two producers of TDCP in Europe. Therefore, only limited information on the life cycle in the EU has been included in this assessment report on grounds of confidentiality. Further information on the life cycle is given in a Confidential Annex, which also describes how research into the life cycle was carried out.

Tonnages and environmental concentrations derived from them have not been corrected for purity of the substance.

The two producers (see below) have participated in the industry consortium working on the risk assessment of TDCP. This consortium assisted in the early stages of the study by sending out a questionnaire to users of TDCP. The results were collated confidentially by the Rapporteur. More recently, the consortium has assisted with further consultation with the confidential downstream users.

Relationship between TCPP, TDCP and V6

As noted in the Foreword, the substances TDCP, TCPP and V6 are good candidates for a concurrent assessment in view of their similar use pattern and chemical similarity. All three substances are used predominantly in various types of polyurethane foam applications in the EU (>97.5% of TCPP; >85% of TDCP and >75% of V6). Chlorinated alkyl phosphate esters (particularly TCPP) were identified as possible substitutes for pentabromodiphenyl ether (pentaBDE) in the risk reduction strategy for that substance (EC 2001). However it has since become clear, from discussion with the industry, that in the EU these chemicals are not direct replacements for pentaBDE, and that changes in TCPP consumption are linked mostly with the decline in TCEP use and increase in the market for polyurethane (PUR) generally (pers. comm., 1st March 2004). As discussed in Section 2.1.2, consumption levels appear to have stabilised in recent years; this risk assessment represents a realistic upper limit of EU production and consumption and significant increases are not anticipated in the near future.

PRODUCTION

Production processes

The process is carried out by reacting phosphorus oxychloride with an organic epoxide chemical in the presence of a catalyst. The crude product is washed and dehydrated to remove acidic impurities and residual traces of water and volatile chemicals. The product is then filtered, transferred to storage tanks for dispatch in road tankers or packed into drums (pers. comm. 30th April 2001, Rhodia).

Production capacity

There are two producers of TDCP in the EU: Supresta (whose TDCP business was owned earlier in the ESR process by Akzo Nobel) and Albemarle, (whose TDCP business was owned earlier in the ESR process by Rhodia and previously Albright and Wilson). References are made in accordance with the company that supplied information at the time. Total EU production in 2000 was less than 10,000 tonnes, with production taking place in Germany and the UK. Between 1998 and 2003, production has fluctuated slightly but the total EU sales

tonnage has remained reasonably stable within approximately 10%. The EU consumption used in the risk assessment represents the upper limit of sales in the six year period for which data are available. The Rapporteur has no reason to anticipate significant tonnage increases in the near future, based on industry information and general research.

Neither producer imported TDCP into the EU in the year 2000. Both are of the opinion that TDCP is not imported into the EU by any other party (pers. comm. 26th February 2002, Akzo Nobel and pers. comm. 6th March 2002, Rhodia).

In respect of automotive and furniture use, by far the most significant applications of TDCP, it is known that there is some import/export of finished articles, but overall the EU is a net exporter. There is no specific information regarding the movements of TDCP-containing furniture and vehicles. It is possible that finished goods containing TDCP in rebonded foam may be imported into the EU. This is not accounted for in the assessment as there is too little information, although it is not likely to be significant.

Both producers exported TDCP from the EU in the year 2000. It is assumed that no handling (e.g. repackaging) takes place and that no losses of TDCP arise through export.

As a result of exports, consumption is somewhat less than production.

Table 0.1 Production and consumption of TDCP in the year 2000

Life Cycle Stage	Tonnes in Year 2000
Production	< 10,000
Imports	None
Exports	Yes

Full details are given in the Confidential Annex

USES

Introduction

TDCP is an additive flame retardant, i.e. it is physically combined with the material being treated rather than chemically combined. The amount of flame retardant used in any given application depends on a number of factors such as the flame retardancy required for a given product, the effectiveness of the flame retardant and synergist within a given polymer system, the physical characteristics of the end product (e.g. colour, density, stability, etc.) and the use to which the end product will be put.

Somewhat less than 10,000 tonnes of TDCP were consumed in the EU in the year 2000. Most TDCP is used in the production of flexible polyurethane (PUR) foam. TDCP is added directly at the point of production of flexible foams. Most foams containing TDCP are used in the automotive industry, with some use in furniture.

TDCP operates in the same marketplace as the flame retardant TCPP. Owing to the price differential between these products (TDCP is around twice the price of TCPP), TDCP is only used in those applications where a more efficient flame retardant is required to meet specific standards (pers. comm. 19th March 2002, Rhodia).

Use of TDCP in products other than PUR tends to be associated with single users who have tried the product of their own accord and have decided to use it (pers. comm. 19th March 2002, Rhodia). The low tonnage associated with these other uses confirms that TDCP is not widely used outside the polyurethane industry.

The use pattern and life cycle stages considered in this assessment are reported in **Table 2.2** and shown in **Figure 2.1**. Further information including information on the confidential life cycle stages is given in the Confidential Annex. Given that there are only two producers and that both producers have provided a detailed breakdown of tonnage, the life cycle is well defined.

Table 0.2 Use pattern for TDCP

Ref. Env[1]	Ref. HH[2]	Industry Category	Use category	Description	Percentage of total use
A	5	11	22	PUR foam for use in automotive applications	< 80%
B	2, 3	11	22	PUR foam for use in furniture	< 25%
C	-	Confidential	22	Confidential	<15%
D[3]	-	Confidential	22	Confidential	
E	-	Confidential	22	Confidential	
F	-	Confidential	22	Confidential	
G	-	Confidential	47	Confidential	
H	-	Confidential	22	Confidential	
I	4	11	22	Rebonding of flexible foam	This is a form of recycling
J	-	11	22	Recycling as loose crumb	This is a form of recycling
Total					100%

Industry Category 11 = polymers industry Use category 22 = flame retardants and fire preventing agents Use category 47 = softeners

Notes:

1 – Reference letter used in the Environmental risk assessment

2 – Reference number used in the Human Health risk assessment

3 – Consultation suggests that supply has ceased; however it is not clear how long ago, and therefore it is assumed that the scenario could still be relevant. This is discussed in more detail in the Confidential Annex.

Product Register Data

Data from product registers have been provided by Denmark, Sweden and Switzerland. This information is summarised in **Table 2.3**, together with data from the SPIN database (data about the use of substances in Norway, Sweden, Denmark and Finland). Data for Sweden in 1999 are for TDCP combined with TCPP and are therefore of limited use. In this regard, data for Sweden for the year 2000 indicate only limited products containing TDCP while data presented for TCPP for the year 2000 indicate that the diversity of usage reported in 1999 is owing to the inclusion of TCPP in the data (see HSA/EA, 2008a). Overall, the product register data do not provide new information concerning uses of TDCP.

It is notable that the industry's view is that not all uses here are current or recommended uses. In particular, both producers have indicated that uses in concrete and as a resin hardener do

not apply to TDCP. Neither of these applications is included in the risk assessment as no further evidence of these applications has come to light in the research and consultation procedures.

Table 0.3 Product register and SPIN data

Country	Year	Tonnage	Number of Products	Concentration*	Description
Denmark	-	226	4	10% to 100%	Industry group and product types are confidential
Sweden	1999	350**	45**	-	Plastics, concrete, textiles & insulation materials. 9/45 products available to consumers **
	2000	-	3	-	Use: raw material (fire prevention additive in plastics). Trade code: Industry for plastic products. No consumer products.
Switzerland	-	-	1	1%	Hardener in resin

* Intervals used in the Danish Product Register are 0-1%, 1-5%, 5-10%, 10-20%, 20-50%, 50-80% and 80-100%. If limited data indicate confidential information, broader intervals are used.

** Combined data with TCPP

A life cycle assessment study by SP, Sweden and IVL-Swedish Environmental Research Institute, Sweden (Simonson *et al*, undated) investigated emission of pollutants associated with different life cycle stages of sofas. Three sofas were tested. The purpose was to assess pollutant emissions at all stages of the sofas' life cycle, including in the event of fire. Emissions of the flame retardant (FR) itself were not investigated. The information and assumptions regarding the life cycle are useful for comparison with the assessment made in the current risk assessment. A schematic representation shows the life cycle stages of relevance for the flame retardant as:

flame retardant production; material (i.e. foam) production

production of primary product (i.e. item of furniture)

use of primary product (i.e. in-service)

recycling processes (see below)

incineration

landfill/landfill fire

fire of primary products.

Service lives of ten and fifteen years were used in the LCA, though this appears to have been used as a half-life in the assessment. The mode of recycling is of interest; the schematic indicates mechanical/feedstock recycling, but this is not believed by the Rapporteur to be a valid route and is not assessed in the present ESR RAR. Interestingly, elsewhere in the report, the only route of 'recycling' investigated for releases is for heat recovery (i.e. incineration).

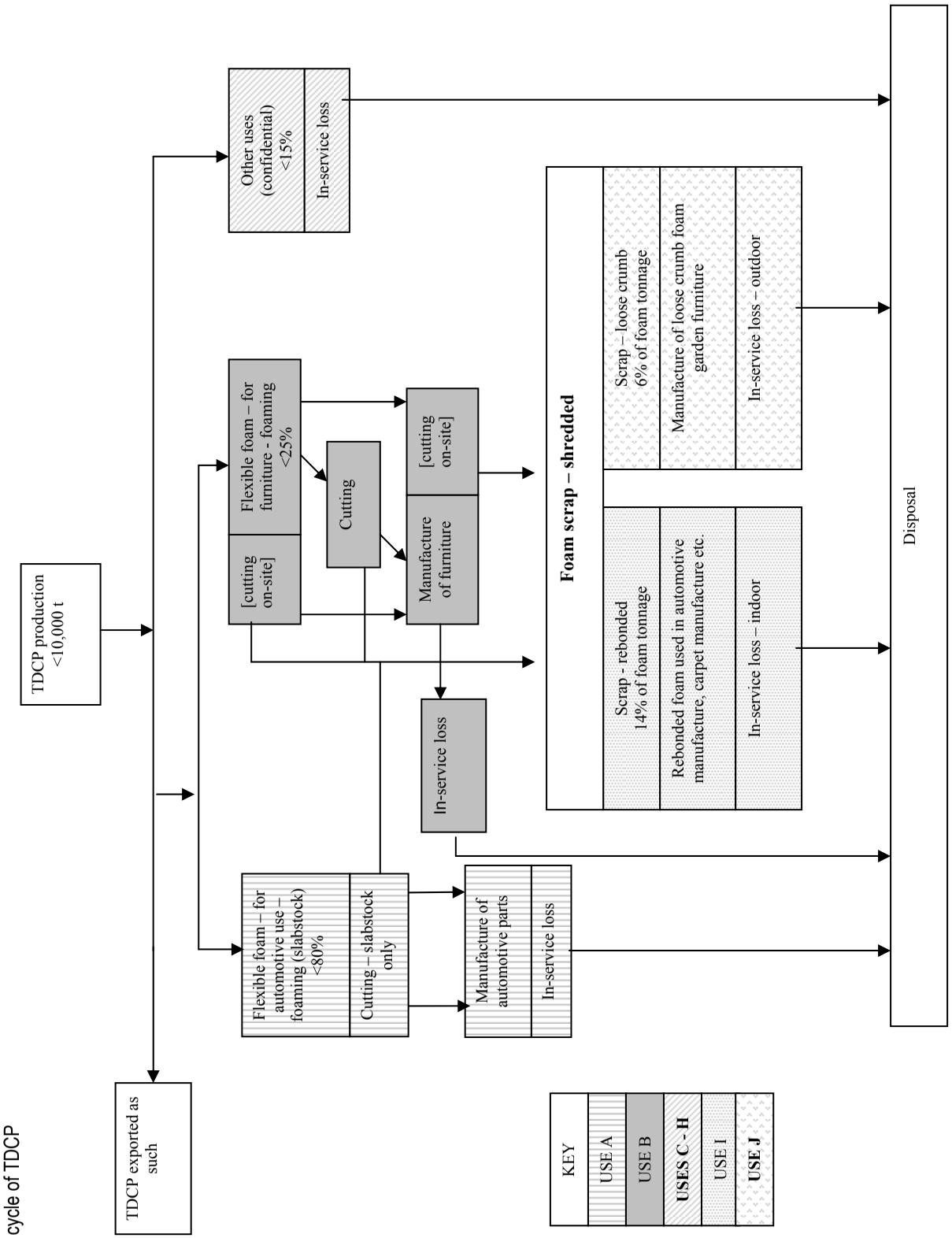


Figure 2.1 Life cycle of TDCP

Scenarios

A longer, more general, discussion of relevant industries is provided in Appendix A.

Flexible foam

Flexible foam production

Flexible foams are produced by pouring a blend of the two raw materials (polyol containing additives including flame retardants such as TDCP, and di-isocyanate) onto a rolling conveyor belt (in the case of slabstock foam) or into a mould (moulded foam). Moulded foam is mainly used in the automotive industry (seat cushions, headrests), with some use for office furniture. Slabstock foam is cut in accordance with the specifications demanded by customers, the main application being for furniture (EC, 1997). Slabstock foams are also used for rear car seats and fabric lining for seat covers and roofing in cars. The market for slabstock foams is around seven times larger than the market for moulded foams for car seats (Mark and Kamprath, 2000).

Note that the PUR industry uses the term "conversion" to describe the cutting of foam. In the Emission Scenario Document (ESD) for additives used in the plastics industry (OECD, 2004), however, the term "conversion" is used to describe manufacture of products (i.e. foaming). For the purposes of clarity in this assessment the term "conversion" is used only as defined in the ESD.

For further information on slabstock foams, moulded foams and polyether versus polyester foams, refer to section 1 of Appendix A. The majority of the description of foam production presented in this section is taken from the risk assessment for pentabromodiphenyl ether (EC, 2000).

Cutting

Blocks of PUR foam generally have to be cut into the required size/shape of the final product. This operation usually occurs after the blocks have cured and cooled. For some applications (e.g. seats for office furniture), PUR foam can be produced in a mould of the desired shape and so cutting is not required.

When fabricating a block, the first stage is usually to trim the sides and top of each block to give a block with uniform faces. This is carried out using vertical and horizontal band knives. The amount of scrap foam removed from the block depends on the size of the block and the type of machine used to produce it. For instance, it has been estimated for a block of foam of density 22 kg/m^3 and having dimensions 2 m x 1.5 m x 1 m, the scrap foam generated from trimming will vary from around 15% to <5%, depending on the machine used. The highest wastage figures are from "domed-topped" blocks made in machines with unrestrained tops, with lower figures being obtained from machines/processes designed to minimise the formation of a domed top (Woods 1982 in EC 2000).

Blocks are passed on to "converters" (hereinafter called "cutters") who cut these into the required size and shape. Foam producers operate their own cutting facilities, but also sell to a

large number of other cutters, most of which (in the UK at least) are small, privately owned companies. In the UK alone there are hundreds of foam cutters (pers. comm., not attributable[10]). Cutting is carried out using band saws. Dusts are collected at the point of cutting by extractors attached to the blade. Hot wire cutting methods are not used any more in this industry (pers. comm., 2nd July 2004).

Overall, for any flexible slabstock foam, scrap foam from cutting totals around 20% of the final product (pers. comm., not attributable):

half (10%) is lost in terms of skins when the block is first cut (when a block is made it has a skin like a loaf of bread which needs to be removed); and

the other half (10%) comes from cutters, for example when cushions are cut. In this regard not all cushions are regularly shaped, and some shapes create more scrap than others.

The collection rate for scrap produced by cutters is "very high" as rebonding facilities pay for the scrap foam, the alternative being for the cutter to pay for disposal of the foam (pers. comm., not attributable). Scrap foam may be sold as second quality foam, or will be granulated (to form 'crumb') and made into rebonded foam.

Furniture manufacture

Cutters sell foam of the required size and shape to furniture makers, i.e. furniture makers do not need to re-cut the foam. That said, some foam is sold directly to furniture makers who cut their own foam. Therefore end product manufacturers may carry out cutting of polyurethane foam (EC 2000). In contrast some cushions arrive at the furniture manufacturer pre-covered with polyester fibre (pers. comm., not attributable).

Flame bonding is a method for laminating polyurethane foam sheet to materials such as textiles. The foam sheet is passed across a propane/air flame and the foam is then brought together with the textile material between pressure rollers. The flame treatment generates a chemically active surface that facilitates bonding to the textile substrate (HMIP 1995). The high temperature used in flame bonding leads to emission of volatile organic compounds (VOCs), including benzene, together with hydrogen cyanide and particulate matter as a result of pyrolysis. Free di-isocyanates including toluene di-isocyanate (TDI), are also present in the fumes which are given off in the process, as a result of oxidation and chain scission (HMIP 1995). Flame lamination companies within the EU have to comply with national emission regulations and most facilities achieve these requirements by the use of appropriate attenuation techniques. Activated carbon scrubbing techniques are often used to meet the more stringent national emission legislation (pers. comm. 22nd January 2007).

[10] In all cases of a non-attributed personal communication it is not possible to reveal the source of the data. The information was provided by industry during the consultation process.

Recycling of PUR foams

Rebonding

In a typical process, foam scrap is fed through a shredding machine and then into a granulator. The granules are screw-conveyed into a vessel where the material is sprayed with pre-polymer and mixed to ensure a thorough coating. The coating granules are then screw conveyed into a rectangular or circular moulding press where the mix is compressed and consolidated as the pre-polymer cures. Curing is facilitated by steam injection (HMIP 1995). The condensate is ultimately removed under vacuum and vented to the air (pers. comm. 29th April 2004). The rebonded blocks are removed and allowed to stand in order to cool (HMIP 1995). The foam product is then either cut (converted) in the usual way (EUROPUR, 2005a), or can be "peeled" from the block at the desired thickness and have a suitable backing applied (EC 2000).

A survey carried out by EUROPUR (pers. comm. 7th December 2005) accounted for approximately 45 kilotonnes of rebonded foam produced in the EU, and it was estimated that approximately 60 kilotonnes are rebonded in total. A high proportion of this is produced in the UK (approximately 22 kilotonnes) (pers. comm. 7th December 2005). Across the EU, only a low proportion of this will contain flame retardants. Cheaper non-FR foam trim can be obtained exclusively but it is likely that a site rebonding FR-PUR will also be handling non-FR foam. It has been estimated that a typical site might rebond 3-5 kilotonnes of foam per year in total (pers. comm. 29th April 2004).

Use of Rebonded Foam

The relative high density and resilience of rebond make it suitable for applications including vibration sound dampening, sport mats, cushioning, packaging and carpet underlay and new applications are constantly being developed (ISOPA 2001a). In cars, rebond can be used for sound insulation, for example under the carpet in the boot. In cushioning, a strip of rebonded foam is used along the front of some cushions on the basis that it is more hard wearing. There is also some use in office furniture (ISOPA 2003).

Re-bonders in mainland Europe now handle the two lines of scrap together (the flame retarded foam from the UK, and foam produced elsewhere in Europe, a smaller proportion of which contains flame retardants), avoiding the need to clean out the machines in between a run of each type (pers. comm., not attributable).

In the risk assessment of pentabromodiphenyl ether (EC 2000), losses from re-use or disposal of scrap foam were not separated from losses during use and disposal of finished articles. In this risk assessment, the rates of release from the two types of foam will be evaluated in the same way.

Loose crumb

Shredded scrap foam is used directly for some applications. This is referred to as 'loose crumb' and is used in deep-buttoned soft-cushions for garden furniture and in some low-grade furniture applications. In Europe, the major use of loose crumb is reported to be in garden furniture. The foam industry has indicated that the market for reuse of scrap foam in this way is small and is deteriorating (Bürgi, 2002). To give a realistic worst case, and in the absence

of firm information, it is assumed in this assessment that 70% of the scrap foam remaining in the EU will be rebonded and 30% will be recycled as loose crumb[11].

While all such furniture used to be returned to the UK to meet the demand generated by UK regulations, 50% now stays in mainland Europe. For the purposes of this risk assessment it is assumed that 75% of scrap foam generated in the EU remains here, with the remaining 25% being exported to the US. Thus it is assumed that 75% of the TDCP in scrap foam remains in the EU. The risk assessment is not very sensitive to this assumption, because daily use rate at the main site is not affected by the total. To assess the reasonable worst case (since the rate of loss is higher from outdoor service), it is assumed that all loose crumb is used in garden furniture.

For a full summary of recycling options for PUR foams, including further details on the rebonding process and use of rebonded foam, refer to section 2 of Appendix A.

Automotive use: Use A

Production and use

Data have been provided by producers of TDCP and by companies using TDCP in the production of foams for automotive applications. The number of sites using TDCP is known. Data indicate that both automotive foam and foams for use in furniture can be produced on the same site.

There are three distinct production processes for automotive applications:

slabstock foam produced on conveyor (as for foams used in furniture), involving a continuous or semi-continuous batch process, for use in the lamination of textiles

hot cure PUR foam which is moulded

cold cure moulded PUR foam.

In the absence of any specific information it is assumed that half of the TDCP used in automotive applications is associated with slabstock foams, and that the remainder is used in moulded foams. Hence, only half of the automotive foam containing TDCP is subject to cutting, with associated scrap proceeding into rebonding and loose crumb applications.

For further information on use of TDCP in automotive applications, refer to section 3 of Appendix A.

Rebonding and loose crumb

As discussed in section 2.2.2.1.4, the vast majority of scrap slabstock foam produced during cutting is rebonded or recycled as loose crumb. On average 20% of foam produced will end up as scrap. It is assumed that 75% of the scrap foam generated in the EU remains within the

[11] Note: industry (EUROPUR) has indicated that 30% recycling in the form of loose crumb may be an overestimate (pers. comm., 27th March 2006). Therefore it is possible that a higher proportion may be rebonded. However, due to the similarities between the release levels from loose crumb and rebonding processes, and the similarity of site distribution (information provided in the EUROPUR survey) (pers. comm. 7th December 2005), this has no significant implications for the risk assessment at the processing stage.

EU (and therefore is relevant to recycling in the risk assessment); the remainder is assumed to be exported.

As TDCP is used in some slabstock foams for automotive applications, some of the scrap foam from the cutting operations will be rebonded or recycled as loose crumb. Thus it is assumed that 7.5% of TDCP will be recycled in these ways (i.e. 50% use in slabstock x 20% waste x 75% remaining in the EU). In the absence of firm information, it is assumed that 70% of this scrap foam is rebonded and 30% recycled as loose crumb[8].

Imports and exports of motor vehicles and parts

The level of automotive imports and exports into the EU were examined to indicate whether additional TDCP could be entering via this route. European Commission data (EC 2002) indicate that in 1999, EU imports of cars, light commercial vehicles and components were worth EUR 46.58 billion. During the same period, the EU exported the equivalent of EUR 61.35 billion. Thus there was a net trade surplus for the EU with the rest of the world amounting to EUR 14.8 billion in 1999. On this basis it could be argued that there is likely to be a net export from the EU of TDCP in automotive goods. To be conservative, no attempt has been made to account for this trade in the assessment.

End of life

The risk assessment allows for some landfilling of end-of-life automotive foam. Some will be incinerated for energy recovery, though the proportions are not clear.

For further information on end-of-life, the current and future situations for automotive plastics, refer to section 3 of Appendix A

Furniture foams: Use B

Production and use

TDCP is used in the manufacture of furniture and bedding in those applications where the less expensive and more volatile flame retardant TCPP cannot meet the required standards, which vary globally. Where TDCP is required, it is used in settees, armchairs and other furnishings (EUROPUR, 2002) and also in mattresses for special purposes, e.g. for use in hospitals and prisons (KEMI, 1996).

California Bulletin of Home Furnishings 117 is a US standard applying to public buildings and to domestic situations. Some companies operating in Europe choose to adopt this standard (e.g. US-owned hotel chains). The standard requires that foam is heat-aged at 104^{o}C for 24 hours. TCPP cannot meet this heat-ageing requirement owing to its volatility. TDCP can meet the standard in some circumstances (pers. comm. 19th March 2002, Rhodia). These observations support the view that losses from foam (e.g. in-service) must be related in some way to volatility.

ISOPA data (undated 1) indicates that 400 foamers/moulders are involved in the production of furniture and bedding from PUR foam in Europe each year, consuming 530,000 tonnes of polyurethane. Given the price and specialist nature of TDCP compared with TCPP, only a limited number of foamers will use this flame retardant. Data has been provided by producers of TDCP and by companies using TDCP in the production of furniture. The number of sites

using TDCP is known. Data indicate that both automotive foams, and foams for use in furniture can be produced on the same site.

Rebonding and loose crumb

As discussed in Section 2.2.2.1.4, the vast majority of scrap foam from foam cutting and furniture production is rebonded or recycled as loose crumb. On average 20% of foam produced will end up as scrap. It is assumed that 75% of the scrap foam generated in the EU remains within the EU (and therefore is relevant to recycling in the risk assessment); the remainder is assumed to be exported.

It is thus assumed that 15% of TDCP will be recycled in the EU in these ways (i.e. 20% scrap x 75% remaining in the EU). In the absence of firm information, it is assumed that 70% of this scrap foam is rebonded and 30% recycled as loose crumb[8].

Imports and exports of furniture into the EU

Imports of furniture into the EU were examined to identify whether additional TDCP may be entering the EU via this route. Imports of upholstered furniture from outside the EU-15 amounted to 848 million Euros in 1997. Most of these were sourced from Poland (more than 50%). Imports have been increasing continuously since 1993 to satisfy a growing internal demand. Extra-European exports of upholstered furniture stood at 1.17 billion Euro in 1997, an increase of 25% on the previous year. Two countries were accounted for more than half of these exports: the United States (39%) and Switzerland (15%) (UEA 2002). Thus there was a net trade surplus for the EU with the rest of the world amounting to 322 million Euro in 1997.

On this basis, it could be argued that there is likely to be a net export from the EU of TDCP in furniture products, especially as the main export market is the US and TDCP is used to meet the US standard (California 117). In order to be conservative, no attempt has been made to account for this trade in the assessment, which affects the amount of foam in service and disposed of at the end of life.

End of Life

At the end of its useful life, furniture in the EU is sent to landfill or incinerated. Most furniture in the UK goes to landfill at the end of service life (pers. comm., not attributable). In this regard the Landfill Directive (1999/31/EC) calls for decreasing amounts of waste to be sent to landfill in all EU countries. As far as possible, waste is to be used for energy recovery with another potentially important route in the future being gasification of plastics including PUR (pers. comm. 31st July 2002, producers and downstream users).

TRENDS

The above discussion, and that described in Appendix A have identified the following trends:

a trend away from exporting scrap foam to the US

a trend towards increased recycling and recovery of PUR foams in general and towards automotive foams in particular, driven by the End of Life Vehicles Directive (ELV) (see Appendix A). This Directive will necessitate large increases in recycling and recovery rates for automotive PUR.

LEGISLATIVE CONTROLS

The use of the flame retardant TDCP in automotive and furniture applications is driven by fire safety standards. The key standards, applicable globally, are:

the Federal Motor Vehicles Safety Standard No. 302 for automotive applications (see section 3 of Appendix A); and

the California Bulletin of Home Furnishings 117 for furniture applications (see section 2.2.2.1.6).

In the UK there are The Furniture and Furnishings (Fire) (Safety) Regulations 1988 (SI 1988 No. 1324) as amended by The Furniture and Furnishings (Fire) (Safety) (Amendment) Regulations 1989 (SI 1989 No. 2538). The equivalent legislation in Ireland is the Industrial Research and Standards (Fire Safety) (Domestic Furniture) Order 1995 (S.I. 316 of 1995).

While these regulations are important in driving the market for TCPP, they are not important for TDCP (further information on the UK regulations can be found in the risk assessment for TCPP, see HSA/EA, 2008a).

There is currently no harmonised set of standards for fire safety testing of furniture in the EU.

For the parts of the life cycle associated with polyurethane foaming, emissions of TDCP will be restricted. All vapours produced in this reaction must be extracted, because potentially dangerous di-isocyanate vapours are produced in the course of the polymerisation. Release of di-isocyanate is highly controlled under a range of international and national regulations. More information is given in the risk assessment report for methylene di-isocyanate (Federal Public Service for Public Health, Safety of the Food Chain and the Environment, 2003).

In respect of flame retardants used in the manufacture of toys, European Standard EN 71-9 (Safety of Toys – Part 9: Organic Chemical Compounds – Requirements) states that certain specified flame retardants, including TCEP, which are used in textiles of toys and accessible components of toys intended for children under 3 years of age should not be found above the limit of quantification of the test method and therefore should not be detected in toys. More generally, Directive 88/319/EEC specifies that toys must not contain dangerous substances or preparations within the meaning of Directives 67/548/EEC and 88/379/EEC (repealed by 1999/45/EC) in amounts which may harm the health of children using them. TDCP is not specifically covered by this legislation beyond this general aspect.

ENVIRONMENT

ENVIRONMENTAL EXPOSURE

Consultation with key downstream users was used to supplement the information provided by producing companies. The producing companies co-operated fully with the assessors and provided information on the number of downstream users associated with each life cycle stage. Associations representing the many downstream users have also been involved with the consultation.

In the assessment of some life cycle stages, it has been necessary to implement appropriate defaults in order to characterise a reasonable worst-case release pattern. Site-specific data have been used where known, to refine the exposure assessment. Defaults set out in this document originate in the A-tables of the Technical Guidance Document (TGD) (EC 2003), or the Emission Scenario Document (ESD) for Additives Used in the Plastics Industry (OECD, 2004). For plastics applications, the ESD defaults override those presented in the A-tables. The ESD gives rates of release only to air and wastewater. The TGD defaults also include rates of release to industrial soil. Exposure of industrial soil to TDCP has not been evaluated in this risk assessment, since 1) the substance is subject to relatively high levels of control on industrial sites, and 2) a rate of release from handling is already calculated in accordance with the ESD. However, exposure of agricultural or grassland soil is foreseeable as a result of weathering and wear in service or at disposal, or by spreading of sewage sludge. This is described in section 3.1.2.2.4.

Most release rates for foam-related stages originate from new models, described in a report (Appendix B), which brings together theoretical modelling with the results of various published studies of releases of FRs from foams.

EUROPUR has sponsored a study to investigate volatile losses of the related substance TCPP from small pieces of PUR foam at ambient temperature (Hall, 2005). Pieces of foam were spread out on a tray under conditions of controlled air flow. The TCPP contents of the pieces were measured analytically over time. Three sizes of fragments of foam were studied in separate runs. Further details are available in Appendix B. A key finding from the experimental data is that initial rapid losses occurred followed by approach to a consistent plateau at around 40% loss, suggesting that only 40% of TCPP in the matrix is available. Losses were fastest from the smallest pieces, but the plateau was the same in each case. Therefore, as a consequence of this study, percentage loss figures associated with possible overall volatile releases from foams or foam particles have been multiplied by a correction factor, representing that which is ‘available’ for release, i.e. is not very strongly bound. The available fraction is estimated to be 0.4 for TCPP, based on the experimental data. For TDCP, which is a more adsorbing, higher molecular weight molecule containing proportionately more chlorine, it is realistic on grounds of structure and properties that a smaller proportion will be available for release. TCPP is not used in automotive applications due to a phenomenon called fogging where a film forms on the interior glass of the car (Patel, 2001). The phenomenon of fogging is not seen with TDCP and V6. TDCP also has a much lower level of volatility than TCPP, expressed as rate of loss (Appendix B). These factors have been used to estimate that the available fraction for TDCP is 10% at the most, although it could be lower than that.

The B-tables and ESD site-size methods are not used in most cases; sufficient information was available about specific aspects of the market to allow representative fractions in the main region and fractions of the main local source to be estimated. The number of days is then evaluated to give a reasonable operational rate given the size of the main site.

In this report and the Confidential Annex, 'R' refers to the fraction of total tonnage in the main region, and 'FMLS' is the fraction of the main local source, i.e. the fraction of the regional tonnage associated with the largest site. In accordance with the TGD definitions, a 'region' is a semi-industrialised European area with surface area 40,000 km^2, with standard default environmental properties and a population of 20 million people. All the figures are based on the most recent edition of the Technical Guidance Document (EC, 2003).

Note regarding environmental releases: There are no reasons to suspect these substances contribute directly to dioxin formation (e.g. there are no aromatic groups). Like all organohalogens the possibility exists that they could act in an indirect way as a source of halogen in high temperature processes. Since most incinerators should have measures in place to control halogenated dioxin emissions, this is mentioned for information only.

Properties of TDCP in the context of the ESD (OECD, 2004)

The main desired activity of TDCP is as a flame retardant. As TDCP is an additive flame retardant, there is the possibility that it may diffuse out of the treated substrate to some extent. It is a liquid at room temperature. Its vapour pressure (5.6E-06 Pa at 25°C, 1.8E-06 Pa at 20°C) falls within the bracket identified as 'low' within the ESD (OECD, 2004).

The ESD envisages flame retardants as being either organic solids or inorganic solids. As stated above, TDCP is a liquid, with a 'low' vapour pressure. For this reason it would be inappropriate to simply apply the organic flame retardants sections of the ESD, as the loss scenarios will be different:

the potential for dust formation is not present for TDCP
process controls may be different.

These factors are thought to have a significant effect upon the handling and compounding stages, though once the additive is formulated, its original physical state is irrelevant. Having said that, it is noteworthy that losses from the stage of conversion (e.g. foaming) are (for some additive types) dependent on the volatility of the additive, according to the ESD.

Variation of loss rate based on volatility in the ESD

For conversion (i.e. foaming), the rates of loss given in the ESD/UCD conform to a pattern; a ratio of 1:5:25 between rates of loss of low: medium: high vapour pressure additives is established. This relationship is applied in some cases here (e.g. for some in-service loss stages) to derive default rates of loss for TDCP (low volatility) based on corresponding known rates of loss for a medium-volatility additive.

Distinction between conversion at large and small sites in the ESD

The ESD, which sets out default rates of loss from all stages of the life cycle, also indicates that 'small' sites tend overall to have a higher rate of loss:

"*As is noted specifically for some of the processes, fume elimination equipment is commonly used to reduce emissions... All the* [release estimates from conversion] *relate to situations where fume elimination equipment is in operation, i.e. larger sites. For smaller sites (<…~750 tonnes of plastic) the emission factors should be increased by a factor of 10*".

It is notable that industry has consistently indicated that this assumption is overly conservative, since exposure to di-isocyanate fumes is always closely controlled. The evidence has been carefully considered and the factor of ten is not applied to life cycle stages of PUR foaming in this risk assessment.

Environmental releases

Release from production

Defaults

It is not considered necessary to seek default rates of loss, or fractions of the main local source. The two manufacturing sites within the EU have been identified and site-specific release data have been provided by the industry.

Extent of site-specific data

Site-specific data provided by the producers of TDCP is set out in the Confidential Annex.

Release from flexible foams

For all life cycle stages following production, it could be considered that the releases associated with one life cycle stage should be subtracted from the tonnage taken forward to subsequent life cycle stages. However, it is considered that for this substance, such variations will be within the range of error in the risk assessment. Therefore, no such correction has been used in the risk assessment.

Foam production

Information on the number of sites is given in the Confidential Annex.

The ESD for plastics additives (OECD, 2004) has been consulted extensively in the course of preparation of this risk assessment. However, the magnitude of releases are based on a report (Appendix B), which brings together theoretical modelling with the results of various published studies of releases of FRs from foams.

The possible sources of environmental release during the manufacture of flexible polyurethane foam are likely to be associated with:

volatilisation from the foam while at elevated temperatures (curing); and

volatilisation from the foam in storage.

Site visits and information received from the industry (see Section 2 and Appendix A) indicate that volatilisation in the foaming process and cleaning of equipment (both of which could theoretically be sources of release of a plastics additive) are not relevant in this case. Furthermore, consultation of TDCP foamers in a questionnaire process undertaken by the consortium through EUROPUR has indicated that the handling of TDCP in storage and prior to use is rigorously controlled to prevent spillage, and hence there is no need for the risk assessment to account for releases to wastewater associated with handling specifically.

Mixing of the components required for the foam is usually carried out in a mixing head immediately prior to feeding into the moulding system. The flame retardant additive can either be metered directly to the mixing head or may be premixed with the polyol component of the foam before feeding to the mixing head. Two main types of mixing head are commonly used: low pressure and high pressure. Low pressure mixing heads need to be cleaned out between cycles by flushing with a suitable solvent (e.g. dichloromethane) or may be flushed with further polyol which can then be reused if the formulation allows. High-pressure (impingement) mixing heads do not require solvent flushing between batches (HMIP 1995).

Releases from curing and storage

The proposed rate of release in curing and storage, accounting for the finding that for TDCP, only 10% of the substance present is available for release, is 3E-05% to air and to wastewater. This is based on a model which brings together theoretical modelling with the findings of various published studies of TCPP in the main, with one result for TDCP (See Section 3.1 above for further details and Appendix B).

While some internal parts of the foam blocks reach a high temperature during curing, this is not expected to have a significant influence on the release rate. This is because the blocks are large and the exterior of the block soon cools.

An additional release of 0.01% to wastewater from handling of raw materials would normally be included for small sites. However a questionnaire survey (pers. comms., 20th – 26th July 2005) has demonstrated that precautions taken when handling, storing, loading and transferring TDCP are such that there it would be overly conservative to account for such a release here.

Releases to air: 3E-05%

Releases to wastewater: 3E-05%

A discussion of the consequences of using ESD defaults is presented in the ESR RAR for TCPP (HSA/EAa).

Foam cutting and manufacture of end products

There may also be losses to the environment associated with the cutting of slabstock foams during cutting and trimming processes and manufacture of furniture and automotive furnishings. Releases associated with the generation of foam dusts must be assessed, since modelling shows that FR contained in foam dusts will be volatilised very rapidly (Appendix B). While it is known from consultation that dusts are collected at the point of cutting by extractors attached to the blade, it could still be the case that a small proportion of dusts and small pieces of foam are exposed to air and hence that some FR could be released on a local scale. A study undertaken by EUROPUR (EUROPUR, 2005b) has established that up to

0.1% of foam is lost as dust and non-recycled offcut pieces. It is estimated that 1% of this material might not be collected by the extractor systems. These pieces of FR foam could then release FR into the workplace air and could reach the environment via air and also wastewater (via adsorption and cleaning). A release rate of 5E-05% to air and 5E-05% to water is proposed, accounting for the finding that for TDCP, only 10% of the substance present is available for release. This is based on a model which brings together theoretical modelling with the findings of various published studies (Appendix B).

Information on the number of sites is given in the Confidential Annex.

Rebonding and loose crumb

Rebonding

Elevated temperature processing applies to what is essentially an additional processing stage in the life cycle.

It is assumed that 5.25% of the TDCP in automotive foams (see section 2.2.2.1.5) and 10.5% of the TDCP in furniture foams (see section 2.2.2.1.6) will be rebonded in the EU (this is based on the combination of 20% of furniture foam and 10% of automotive foam being available for recycling; 75% remaining in EU for recycling; and 70% of recycling being in the form of rebonding[12]). (Neither the quantity of TDCP-containing foam that is recycled nor the concentration of TDCP in the foam is relevant to this assessment as releases are estimated on the total amount of TDCP present which depends on the levels of scrap foam).

The granulation and rebonding processes are contained within equipment, therefore rates of loss are anticipated to be much lower than the theoretical model might suggest. Granulating machines are fitted with dust extraction equipment. Taking the same approach as for cutting at furniture and automotive manufacturing sites, it could be estimated that up to 0.1% of foam is lost as dust, and that 1% of this material is not collected by the extractor systems and could be released to the local air compartment. Releases are therefore 1E-04% to air, accounting for the finding that for TDCP, only 10% of the substance present is available for release. There are no releases to wastewater (Appendix B).

Information on the number of sites is given in the Confidential Annex. A survey carried out by EUROPUR investigated the number of sites and quantities of rebonded foam associated with various EU15 countries (pers. comm. 7th December 2005). The survey data relate to total PUR, including non-FR foam. For TDCP, where there is no distinctive geographical concentration within Europe, the risk assessment parameters can be based directly on rebonding site size distribution. The following set of values are used in the risk assessment:

Fraction in the main region = 0.4
Fraction of the main local source = 0.55

[12] Note: industry (EUROPUR) has indicated that 30% recycling in the form of loose crumb may be an overestimate (pers. comm., 27th March 2006). Therefore it is possible that a higher proportion may be rebonded. However, due to the similarities between the release levels from loose crumb and rebonding processes, and the similarity of site distribution (information provided in the EUROPUR survey) (pers. comm. 7th December 2005), this has no significant implications for the risk assessment at the processing stage.

Loose crumb

It is assumed that 2.25% of the TDCP in automotive foams (see section 2.2.2.1.5) and 4.5% of the TDCP in furniture foams (see section 2.2.2.1.6) will be recycled as loose crumb in the EU (this is based on the combination of 20% of furniture foam and 10% of automotive foam being available for recycling; 75% remaining in EU for recycling; and 30% of recycling being in the form of loose crumb[13]).

The granulation process is contained within equipment, therefore rates of loss are anticipated to be much lower than the theoretical model might suggest. Granulating machines are fitted with dust extraction equipment. Taking the same approach as for cutting at furniture manufacturing sites, it could be estimated that up to 0.1% of foam is lost as dust, and that 1% of this material is not collected by the extractor systems and could be released to the local air compartment. Releases are therefore 1E-04% to air, accounting for the finding that for TDCP, only 10% of the substance present is available for release. There are no releases to wastewater (Appendix B).

It has been indicated that granulation associated with loose crumb recycling generally does not take place at the same sites as rebonding (pers. comm., 27th March 2006). However, since both rebonding and loose crumb are dependent on the availability of scrap foam from the same sources, site distribution may be expected to follow the same distribution pattern. Information on the number of sites is given in the Confidential Annex.

In-service losses

Default rate of release

Based on measured releases, the ESD estimates loss to air and to water. It is known that all of the rates of loss used in the ESD were derived from measurements of medium-volatility additives, therefore it is appropriate to divide these rates by 5 (in accordance with the correction applied to rates of loss from conversion) to obtain the rate of loss of TDCP. Therefore the default release rates can be taken to be:

Indoor service:

Loss to air	0.01% over lifetime
Loss to wastewater	0.01% over lifetime

Outdoor service:

Loss to air	0.01% over lifetime
Loss to wastewater	0.03% per year

Values used in the risk assessment: Furniture and automotive foam

The ESD gives lifetimes for furniture of five to ten years. ISOPA (1997) gives PUR-specific lifetimes for furnishing/mattresses of greater than ten years. This is supported by reports that

[13] Note: industry (EUROPUR) has indicated that 30% recycling in the form of loose crumb may be an overestimate (pers. comm., 27th March 2006). Therefore it is possible that a higher proportion may be rebonded. However, due to the similarities between the release levels from loose crumb and rebonding processes, and the similarity of site distribution (information provided in the EUROPUR survey) (pers. comm. 7th December 2005), this has no significant implications for the risk assessment at the processing stage.

50% of households change their upholstered furniture every eight to sixteen years (DTI undated. In the risk assessment, a lifetime of ten years is used.

All in-service losses are evaluated on a regional basis (over 365 days per year) because no specific local source can be identified for these releases. All service is taken to be indoors.

Given that the air surrounding the foam is likely to be slow moving, and the foam is covered in service by fabrics and upholstery, an annual rate of release of 1E-04% per year to air is proposed, accounting for the finding that for TDCP, only 10% of the substance present is available for release. This is based on a model which brings together theoretical modelling with the findings of various published studies (Appendix B). All in-service losses are evaluated on a regional basis because no specific local source can be identified for these releases.

Since TDCP is an additive flame retardant it may be subject to volatilisation or leaching from the polymer matrix during the lifetime of the use of an article. Given that the parts are unlikely to be washed, the actual potential for leaching from the foam during use would appear to be minimal.

Rebond and loose crumb foams

The application of rebonded foam is assumed to be in indoor applications (such as furniture, mats, cushions and sound insulation, as described in section 2.2.2.1.4). The proportion in the main region is assumed to be 0.1 and a lifetime of ten years is used in the risk assessment.

Given that the air surrounding the foam is likely to be slow moving, and the foam is covered in service by fabrics and upholstery, an annual rate of release of 1E-04% per year to air is proposed, accounting for the finding that for TDCP, only 10% of the substance present is available for release. This is based on a model which brings together theoretical modelling with the findings of various published studies (Appendix B).

Loose crumb foam is assessed as outdoor service (garden furniture). A fraction of 10% in the main region is considered acceptable.

Given that the foam is covered in service by fabrics and upholstery, an annual rate of release of 1E-03% per year to air is proposed, accounting for the finding that for TDCP, only 10% of the substance present is available for release. This is based on a model which brings together theoretical modelling with the findings of various published studies (Appendix B). (Note: as described in Appendix B, the rate of release from loose crumb is ten times higher due than that from rebonded foam, due to its use in outdoor applications with higher air turnover).

Waste remaining in the environment

In keeping with the requirements of the TGD, some consideration of release through weathering and wear over the service life and at disposal is appropriate. A total of 2% release over the lifetime of the article is assumed for most life cycle stages. The release of TDCP is limited by the available fraction (for TDCP, only 10% of the substance present is available for release). Since modelling indicates immediate volatilisation from small particles (Appendix B), in this risk assessment the release is assessed as being entirely to air in the first instance. Hence the release rate used in the risk assessment is 0.2% to air. Redistribution of the substance via fugacity modelling is then dealt with by EUSES. These releases, which are

associated with physical erosion of the polymer, are additional to 'in-service loss', which is associated with volatile releases from the article itself.

It is important to differentiate this route of release from the assessment of in-service loss. Waste remaining in the environment is associated with physical weathering and wear and hence release of FR from foam particles. In-service loss is simple volatilisation out of the foam article itself.

Not all life cycle stages will be subject to weathering and wear processes: these releases are assessed only for TDCP used in flexible foams used for automotive and furniture applications, rebonded foam and loose crumb furniture. The releases are evaluated on a regional scale, in keeping with the in-service distribution of the polymer between the regions for these applications.

In reality the potential for release of particulate waste from weathering, wear, etc., during the service life of furniture and automotive foams may be lower than this estimate, because the foam will have a protective covering. Furthermore, the scenario described above is theoretical only and it has not been possible to test its validity.

End of life

Disposal to landfill is considered likely to be the most significant route of disposal of flexible foam and other articles containing TDCP. As described in section 3.1.2.4, available data suggest that releases of TDCP via landfill leachate are negligible. Its contribution to the $PEC_{regional}$ values is considered to be zero in this risk assessment.

Release from other uses

Releases from other uses is discussed in the Confidential use pattern and exposure Annex.

Release from disposal

It is highly likely that flame retardants, such as TDCP, will find their way to landfill. This could be via disposal of domestic waste, or in water or dust from cleaning homes or commercial premises. The available monitoring data confirm all of these possibilities. There is a very limited amount of data on releases of TDCP from landfill sites in the EU. The Environment Agency of England and Wales analysed the concentration of TDCP in leachate from 22 landfills in southern England and Wales during spring 2005. The data obtained are presented with further detail in section 3.1.4.2. These data show that TDCP was not detected in any samples, with a detection limit of 10 µg/l (pers. comm., 3rd August 2005).

The absence of any definite measured concentrations for TDCP suggests that it can be assumed that there are no significant releases from landfill. In contrast, the related substance TCPP was detected in landfill leachates at a mean concentration of 11±4 µg/l (HSA/EA, 2008a). The finding for TDCP is consistent with its lower solubility and higher adsorption to solids when compared with TCPP, as well as the lower volumes likely to be found in municipal landfills.

End of life for automotive foams

The ESD indicates that plastics constitute 6% of automotive wastes of which 3% is mechanically recovered, and the remaining 97% is landfilled or incinerated (without heat recovery).

Data from APME (2000) for 1998 indicate that of the 728,000 tonnes of plastic present in automotive wastes in Europe, 77% is landfilled, 10% mechanically recovered (and a further 0.14% exported for mechanical recovery) and 13% used for energy recovery.

Section 2.2.2.1.2 reports on current levels of recovery and recycling for automotive PUR as stated in Mark and Kamprath (2000). There is reported to be 70,000 tonnes of PUR available for recovery each year, of which approximately:

5% is recovered and recycled (3% in the Netherlands and an estimated 2% in Italy)

5% (present in ASR[14]) is used for energy recovery, i.e. incineration

90% (present in ASR) is sent to landfill

These values are PUR-specific and are used in the risk assessment.

The future implications of the End of Life Vehicles Directive (2000/53/EC) are discussed in section 3 of Appendix A.

End of life for furniture foams

The ESD indicates that plastics constitute 72% of municipal solid waste arisings. Of this waste stream:

20% is incinerated and the heat recovered

1% is mechanically recovered

79% is landfilled or incinerated (without heat recovery).

Data from ISOPA (1997) indicate the following for post-user plastics waste in West Europe:

6% mechanical recycling

3% incineration without energy recovery

13% incineration with energy recovery

78% landfill

Data from APME (2000) for 1998 indicate that of the 11,370,000 tonnes of plastic present in municipal waste in Europe:

4% is incinerated

66% landfilled

3% consumed in feedstock recycling

4% mechanically recovered (and a further 0.25% exported for mechanical recovery)

[14] Automotive Shredder Residue

22% used for energy recovery.

Industry indicates that at end of life most furniture goes to landfill (see section 2.2.2.1.6). For the purposes of this risk assessment it is assumed that all furniture is landfilled.

Release from landfill

As described in section 3.1.2.4, available data suggest that releases of TDCP via landfill leachate are negligible. Its contribution to the $PEC_{regional}$ values is considered to be zero in this risk assessment.

Release from recycling

The method for calculating levels of loss from recycling is given in the Confidential Annex.

Regional and continental total releases

Total releases at the regional and continental scale include contributions both from local sites and from several life cycle stages evaluated only at the regional and continental scales. In total the release rates to the various compartments are as shown in **Table 3.1** below.

Table 3.1 Total releases to the regional and continental environmental compartments

Endpoint	Emission in kg/d
Total regional emission to air	2.49
Total regional emission to wastewater	0.95
Total regional emission to surface water	0.24
Total regional emission to industrial soil	0.021
Total continental emission to air	15.33
Total continental emission to wastewater	1.07
Total continental emission to surface water	0.27
Total continental emission to industrial soil	0.18

Environmental fate

Degradation in the environment

Atmospheric degradation

A half-life in air of 21.3 hours has been proposed based on an OH radical concentration of 5×10^5 molecules/ml, which is the default in the TGD (EC 2003).

As shown below, the Syracuse Research program AOPWIN gives a predicted reaction rate constant of 18.08×10^{-12} cm^3/molecule.sec. With the TGD model for photodegradation, this is equivalent to a half-life of 21.3 h.

```
SMILES : O=P(OC(CCL)CCL)(OC(CCL)CCL)OC(CCL)CCL
CHEM   : 2-Propanol, 1,3-dichloro-, phosphate (3:1)
MOL FOR: C9 H15 CL6 O4 P1
MOL WT : 430.91
------------------- SUMMARY (AOP v1.90): HYDROXYL RADICALS ----------------
Hydrogen Abstraction       =  18.0819 E-12 cm3/molecule-sec
Reaction with N, S and -OH =   0.0000 E-12 cm3/molecule-sec
Addition to Triple Bonds   =   0.0000 E-12 cm3/molecule-sec
Addition to Olefinic Bonds =   0.0000 E-12 cm3/molecule-sec
Addition to Aromatic Rings =   0.0000 E-12 cm3/molecule-sec
Addition to Fused Rings    =   0.0000 E-12 cm3/molecule-sec

   OVERALL OH Rate Constant =  18.0819 E-12 cm3/molecule-sec
```

Aquatic degradation

Abiotic degradation

The hydrolysis of TDCP was investigated (Kendall and Nixon, 2000) in a study that complied with GLP. The test was performed using Fyrol FR2 formulated product, lot no. 9102C-1 as provided by Akzo Nobel Chemical Inc.; no information on test substance purity is given.

Details of the preliminary test were not reported. The implication is that no significant hydrolysis was observed at pH 4 or 7 although this is not definitively stated; the full test was carried out at pH 9 only. At 50°C and pH 9: $t_{1/2} \approx 14.7$ days.

The test was performed at a nominal concentration of 10 mg/l, representing approximately one-half of the water solubility. Only one test vessel was studied for each of two temperatures (20°C and 40°C), each sampled and analysed in triplicate. Calculations are based on measured t_0 concentration.

Analysis was by GC; presumably the substance monitored is TDCP although the study consistently refers only to "Fyrol FR2" in respect of both the test substance and analyte. The results indicate that at 20°C and pH9, Fyrol FR2 hydrolyses with a half-life of 120 days.

Gerlt (1992) describes the two known mechanisms for non-enzymatic hydrolysis of phosphate esters, and reviews enzymatic catalysis relevant to biological systems. No information on rates is given. Discussion is general only.

It is very unlikely that the rate of hydrolysis at environmentally-relevant pH values is fast enough to have any influence on predicted environmental concentrations.

Photodegradation

Echigo *et al* (1996) investigated aqueous phase decomposition of TDCP (and two other substances) by ozone, UV light and hydrogen peroxide, singly and in combination. The influence of pH was examined briefly.

Whilst the work was aimed primarily at waste treatment, the results showed that reaction with oxidative species such as ozone or hydroxyl radicals can proceed rapidly. The paper did not relate the data to typical environmental conditions.

Biodegradation studies

In a MITI study (CITI, 1992) performed in compliance with OECD 301C, sludge was collected from ten sites in Japan: four sewage plants and six surface waters (three rivers, a lake and two 'bays'). Samples were taken regularly and fresh and old samples were mixed. The amounts used were 100 mg/l test substance and 30 mg/l sludge.

No information is supplied describing the purity of the test substance, conditions of storage, etc. Degradation after 28 days was 0 – 4%. The report does not set out results in any detail. Aniline was used as the reference substance, present at 100 mg/l. Aniline degradation fulfilled the required criteria for validity.

In a GLP-compliant study of ready biodegradability (modified Sturm test), (Jenkins, 1990a), sludge was sampled from a sewage treatment plant treating predominantly domestic waste. This was used to inoculate test vessels at a relatively low level (1%).

The test substance was present at a concentration of 10 and 20 mg/l (this is at a higher dose than the microbial inhibition screen performed before the biodegradation study, which tested only up to 10 mg/l). No degradation was observed at either loading rate of test substance. The reference substance was sodium benzoate, present at 20 mg/l. Sodium benzoate degradation fulfilled the required criteria for validity. The test report is very clear and provides a great deal of detail about the conditions used.

In a study of biodegradability (SafePharm, 1996, brief report only available for review), mixed population sewage sludge was sampled from 10 UK sites. Sludge was present in test vessels at a concentration of 100 mg/l dry weight. The test substance was present at 30 mg/l. No degradation was observed over 28 days based on 0.78 mg O_2/mg ThOD (DOC analysis was not carried out for the test substance due to low solubility). Aniline was used as a reference substance, present at 100 mg/l in test vessels. Degradation of aniline fulfilled the required criteria for validity.

This study refers to inherent biodegradability and OECD 302C. However, no acclimation period was used and hence, in this context, this study can only be seen as a short screening test, from which no conclusions regarding inherent biodegradability of TDCP can be drawn.

The degradation of what is believed to be an isomer of TDCP in environmental water was studied during 1979-1980 (Hattori *et al*., 1981, cited in WHO, 1998). The test waters were taken from the Oh River (test concentration 20 mg TDCP/litre), Neya River (1 mg TDCP/litre), and seawater from two locations in Osaka Bay. Degradation was measured by analysis of the increase of phosphate ions, using the molybdenum blue colorimetric method. Degradation levels at 7 and 14 days were 12.5 and 18.5% respectively in Oh River water; 0 and 5.4% respectively in Neya River water, 0 and 22% respectively in seawater from Osaka Bay (Tomagashima), and 0% in seawater from Osaka Bay (Senboku).

Biodegradation under anaerobic conditions in sewage sludge has been explored in a non-GLP investigative study (van Ginkel, 2005a). Anaerobic sludge from a municipal WWTP treating predominantly domestic sewage was used in a 60-day study, performed in triplicate with reference blanks, in which biodegradation was assessed on the basis of release of chloride. TDCP was present at 234 µM (equivalent to 100 mg/l) and the inoculum was added at 2 g dwt/l; lactate was also present to act as an electron donor (468 µM). Test bottles were flushed for 10 minutes with nitrogen gas and incubated in the dark at 100 rpm and 30ºC. At the start of the test, the chloride concentrations in the blank and test bottles were below the detection limit <30 mg/l. After 60 days, the chloride concentration was still < 30 mg/l in the blank and

test bottles. Complete mineralisation of TDCP would have produced 50 mg/l chloride. Due to the relatively high limit of quantification of chloride, it cannot be discounted that some biodegradation took place but there is no definitive evidence for it. Comparable quantities of biogas (methane and carbon dioxide) formed in the test vessels both in the presence and absence of TDCP, indicating that TDCP was not inhibitory to the methanogenic micro-organisms.

TDCP has been shown to be not readily biodegradable. No definitive conclusion can be reached regarding inherently biodegradability or biodegradation under anaerobic conditions. For the purposes of modelling the rate constants for degradation in wastewater treatment and surface waters are set at 0 h^{-1}, in accordance with the TGD.

Degradation in soil

Very little degradation (<6%) occurred in soil in a 17-week study (Schaefer and Stenzel, 2005). The study used four soil types: sand, loam, clay loam and sandy loam. ^{14}C-radiolabelled TDCP was applied to the soil surface and the soils were incubated at 20 ± 2 °C, and two test chambers of each soil were analysed at intervals of 0, 7, 14, 35, 63 and 122 days. ^{14}C-labelled substances were analysed through extraction and combustion of soil, CO_2 traps and charcoal traps, using liquid scintillation counting.

Total ^{14}C recovery was very good with material balances for individual test chambers ranged from 90.6% to 104%. A small portion (mean value ≤5.5%) of the total ^{14}C was found in the CO_2 traps. Ethanol extracts were characterized using HPLC analysis with radiochemical detection. TDCP was the only radiolabelled material found in the extracts.

There was no inhibition of the soil micro-organisms.

For the purposes of modelling the rate constant for degradation in soil is set at 0 h-1.

Summary of environmental degradation

Key information is summarised in **Table 3.2**.

Table 3.2 Environmental degradation rates for TDCP

Endpoint	Year test completed	Protocol cited	Results	Reliability	Study reference
Hydrolysis	2000	OECD 111	Most rapid $t_{1/2}$ at pH 9: >120 d at 20 deg C	(2) valid with restrictions	Kendall and Nixon, 2000
Photodegradation			The output of SRC AOPWIN program k for reaction with hydroxyl radicals = 18.1 x 10^{-12} cm^3 molec. s-1		
Ready biodegradability	1990	Modified Sturm test	Not readily biodegradable	(1) valid without restrictions	Jenkins, 1990a
Degradation in river and sea waters	1980	None	River waters: 0 – 12.5% degradation at day 7; 5.4 – 18.5% degradation at day 14. Sea waters: 0% degradation at day 7; up to 22% degradation at day 14.	(4) not assignable	Hattori *et al.*, 1981, cited in WHO, 1998
Anaerobic biodegradation	2005	None	Complete mineralisation would have produced 50 mg/l chloride After 60 days, result <30 mg/l chloride (limit of quantification)	(2) valid with restrictions	Van Ginkel, 2005a
Degradation in soil	2005	OECD 307	Very little degradation (<6%)	(1) valid without restrictions	Schaefer and Stenzel, 2005

These data show that the rate constants in water, sediments, sewage sludge and soil can all be set to zero.

Distribution

A summary of studies related to the environmental distribution of TDCP is given in **Table 3.3**.

Table 3.3 Studies related to environmental distribution of TDCP

Endpoint	Year test completed	Protocol cited	Results	Reliability	Study reference
Adsorption to 3 soils, sediment and sludge[1]	2006	OECD 106	K_{oc} = 1780 (range 1540 – 2010), log K_{oc} = 3.25	(1) valid without restriction. GLP study	Schaefer and Ponizovsky, 2006
Adsorption to soil[2]	2002	Method C.19 of 2001/59/EC	Log K_{oc} = 4.09$\pm$0.29	(1) valid without restrictions[3]. GLP study	Cuthbert, J.E. and D.M. Mullee, 2002a

Notes

1 – Test sample was radiolabelled TDCP with radiochemical purity 99.9%

2 – Test sample was a composite sample of purity 94.2%, derived from recent representative commercial products from the main producers.

3 – It is important to note that while this result is of reliability (1), the results are not suitable in this case for application in risk assessment, for reasons expanded upon in the text (see Section 3.1.3.2.1). The method used is a screening study.

Adsorption

The understanding of the adsorption behaviour of TDCP, and the structurally-related substances TCPP and V6, is based on a number of items of data. These are:

Measured adsorption coefficient in 3 soils, sediment and sludge for TDCP, in accordance with OECD guideline 106
Estimated adsorption coefficient by HPLC measured with all three substances, in accordance with OECD guideline 121
Prediction by standard QSAR methods, from the TGD.

Detailed review of OECD 106 study (Schaefer and Ponizovsky, 2006)

The study was conducted to a high standard, in full compliance with all three tiers of the OECD 106 method and in accordance with the principles of GLP. TDCP in 0.01 M calcium chloride was equilibrated with each of three soils (a clay loam, a loamy sand and a clay), one sediment and one activated sludge solid. Study of the kinetics of adsorption was made which showed that the equilibration time was adequate. The solids and aqueous phase were separated by centrifugation. Method checks on the adsorption to glassware were made and this was found to be insignificant. The stability of the substance was checked.

Both adsorption and desorption were studied, and the equilibrium constants (K_d) were sufficiently similar to show that there was reversibility. Kinetic studies showed that the processes were rapid. The determination of Freundlich isotherms was made which showed that the processes were not highly concentration-dependent (results not reported herein).

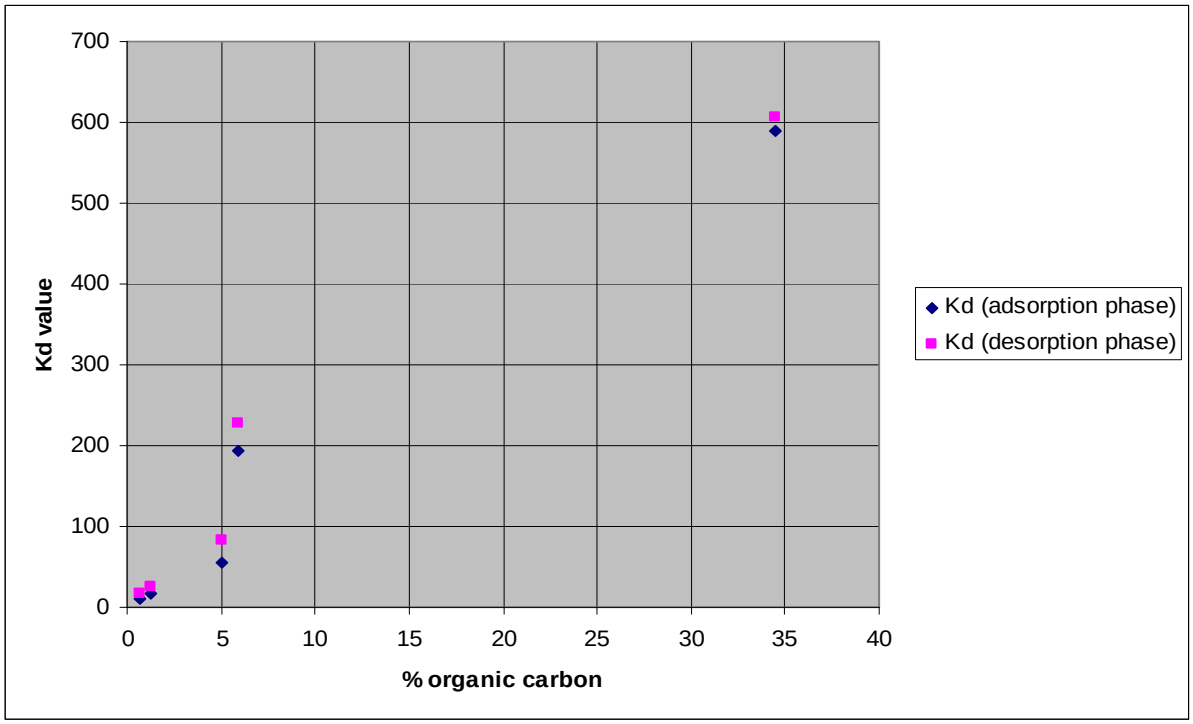
The data are presented in **Table 3.4**.

Table 3.4 TDCP OECD 106 study: partition data

Substrate	% organic carbon	K_d (adsorption phase)	K_d (desorption phase)
Clay loam (TB-PF)	5	56	82
Loamy sand (Roger Myron)	1.3	18	26
Clay (Montana clay)	0.7	11	16
Sediment (Turkey Creek)	5.9	193	227
Sludge solids	34.46	590	606

In the case that adsorption of TDCP to soil is primarily to the organic matter in soil, then K_d is expected to be directly proportional to the fraction of organic carbon. The simplest way to examine this is by a graph of K_d vs. the fraction of organic carbon (OC) (**Figure 3.1**).

Figure 3.1 Adsorption and desorption of TDCP



Linear regression of these data with no constraints gave the following statistics:

Regression Statistics	
Multiple R	0.977608819
R Square	0.955719003
Adjusted R Square	0.950183879
Standard Error	51.66746504
Observations	10

ANOVA

	Df	*SS*	*MS*	*F*	*Significance F*
Regression	1	460932.2844	460932.28	172.66441	1.07E-06
Residual	8	21356.21555	2669.5269		
Total	9	482288.5			

	Coefficients	*Standard Error*	*t Stat*	*P-value*	*Lower 95%*	*Upper 95%*
Intercept	21.82087835	20.40780427	1.0692418	0.3161584	-25.239603	68.881359
% organic carbon	16.9635897	1.290970594	13.140183	1.07E-06	13.986606	19.940573

It can be seen that the 95% confidence interval of the intercept spans zero, and it is logical to constrain the intercept to be zero. This gives:

Regression Statistics	
Multiple R	0.984918346
R Square	0.970064147
Adjusted R Square	0.858953036
Standard Error	52.07710877
Observations	10

ANOVA

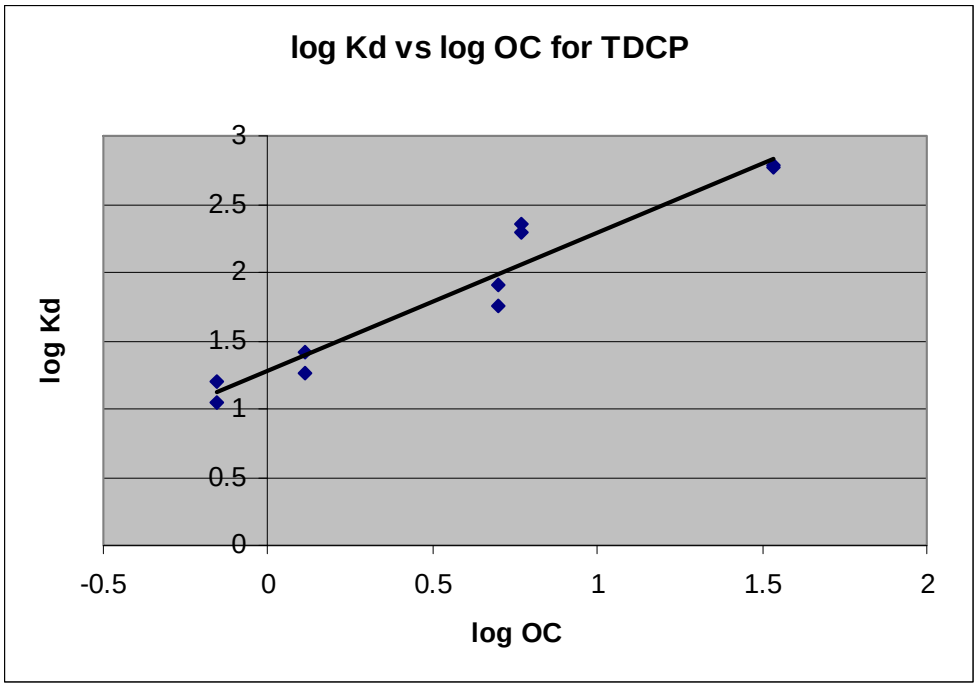
	Df	*SS*	*MS*	*F*	*Significance F*
Regression	1	790942.7727	790942.77	291.64285	1.404E-07
Residual	9	24408.22732	2712.0253		
Total	10	815351			

	Coefficients	*Standard Error*	*t Stat*	*P-value*	*Lower 95%*	*Upper 95%*
Intercept	0	#N/A	#N/A	#N/A	#N/A	#N/A
% organic carbon	17.79068215	1.041758217	17.077554	3.639E-08	15.434061	20.147303

From this regression, K_{oc} = 1780 (range 1540 – 2010). However, due to the nature of linear regression, the gradient of the line, which equates to K_{oc}, is heavily controlled by the highest x value. For a zero intercept, the logarithm of K_d vs the logarithm of OC should also be a straight line, with unit gradient.

This possibility is shown in **Figure 3.2**:

Figure 3.2 Adsorption and desorption of TDCP, expressed as log values



The regression statistics are:

Regression Statistics	
Multiple R	0.9676066
R Square	0.9362625
Adjusted R Square	0.9282953
Standard Error	0.173636
Observations	10

ANOVA

	df	*SS*	*MS*	*F*	*Significance F*
Regression	1	3.5430092	3.5430092	117.51486	4.633E-06
Residual	8	0.2411956	0.0301495		
Total	9	3.7842048			

	Coefficients	*Standard Error*	*t Stat*	*P-value*	*Lower 95%*	*Upper 95%*
Intercept	1.2758165	0.0780591	16.344247	1.978E-07	1.095812	1.455821
log OC	1.0138471	0.0935246	10.840427	4.633E-06	0.7981789	1.2295153

The intercept is log K_{oc}, and with the correction for use of % rather than a fraction gives K_{oc} = 1890 (range 1250 – 2860). The graph and the results show that in fact no individual data point is having an excessive influence on the results obtained without taking logarithms. Therefore the regression without taking logarithms should be preferred, i.e. K_{oc} = 1780, log K_{oc} = 3.25.

The result is K_{oc} = 1780 (range 1540 – 2010), log K_{oc} = 3.25

Interpretation of the data shows that TDCP adsorption to the tested substrates was proportional to organic carbon content. Therefore the value of K_{oc} can be used directly in EUSES. The uncertainty in the value has been considered, and it should be noted that variation within this range of K_{oc} has only a small influence on the resulting PEC/PNEC ratios, and hence it is considered that there is no need to explore alternative values in detail in the risk assessment report.

Extrapolation of the measured K_{oc} to TCPP and V6

The log K_{ow} of TDCP is 3.69. Based on the measured log K_{ow} of 3.69 and the measured log K_{oc} of 3.25 from the OECD 106 study, the following empirical relationship can be used for read across to structurally related substances: log K_{oc} = -0.44 + log K_{ow}. Justification of the use of such a relationship is made within the respective reports (HSA/EA 2008a and b), see also **Table 3.5**.

Review of the other K_{oc} data

A reliable modern measurement of the soil adsorption coefficient K_{oc} obtained by the HPLC estimation method is available (Cuthbert and Mullee, 2002a). The result is $K_{oc} = 1.23 \times 10^4$, log K_{oc} = 4.09±0.29. The +/- value is the 95% confidence interval. It should be noted that the calibration substances were general substances, not related structurally to TDCP, there being insufficient reliable calibration substances containing the phosphate group. For this reason, estimates of K_{oc} from the EPIWIN program are not considered to be reliable enough for phosphates and are not included here.

The HPLC screening test resulted in a 7-fold higher K_{oc} than was found in the OECD 106 study. This suggests that some specific interaction with the HPLC column had occurred, possibly involving the phosphate group. This interpretation is further supported in that V6, which has two phosphate groups, is the substance for which the HPLC estimate is most out of line, relative to the K_{ow}. Adsorption behaviour in the OECD 106 study was proportional to organic carbon content which suggests that adsorption to components other than organic carbon was not significant.

The TGD gives a method for estimating the value of K_{oc} based on log K_{ow}. The most appropriate equation is that for phosphates:

Log K_{oc} = 0.49 log K_{ow} + 1.17 (n = 41, r^2 = 0.73, s.e. = 0.45)

The log K_{ow} for TDCP is 3.69 ± 0.36. On the basis of the uncertainty on this value, a range of log K_{oc} can be estimated. From the above equation, K_{oc} = 950.8 (range 633.4 – 1427.2). Estimates made using the hydrophobics equation are also provided for reference in **Table 3.5**.

A comparison of the measured and estimated K_{oc} values for all chloroalkylphosphates being evaluated under ESR is given in **Table 3.5**.

Table 3.5 Comparison of measured and estimated K_{oc} for chloroalkylphosphates in the ESR process

Substance (CAS)	K_{oc} derived from OECD 106 result for TDCP	K_{oc} measured [l/kg] by HPLC estimation	K_{oc} estimated [l/kg] from log K_{ow} (Phosphates)	K_{oc} estimated [l/kg] from log K_{ow} (Hydrophobics)
TCPP (13674-84-5)	174	576	304	187
TDCP (13674-87-8)	**1780**	12300	951	1230
V6 (38051-10-4)	245	11000	360	247
TCEP (115-96-8)	-	-	110	-

Conclusions

For TDCP, good agreement is found between the QSAR predictions of K_{oc} from K_{ow} (K_{oc} = 950.8 (range 633.4 – 1427.2) and the value measured in the OECD 106 study (K_{oc} = 1780 (range 1540 – 2010). The HPLC screening estimates of K_{oc} appear to consistently over-estimate this value for the chloroalkylphosphates. For TDCP, the K_{oc} measured in the OECD 106 study will be used for the risk assessment, K_{oc} = 1780 log K_{oc} = 3.25. The regression equation derived from this study will be used to derive K_{oc} values for TCPP and V6 based on their measured log K_{ow} values.

The measured value of K_{oc} = 1780 is used in the risk assessment of TDCP. The range of the value does not have a significant impact on the conclusions.

The coefficients in **Table 3.6** are derived by the EUSES program from this value of K_{oc}, using default conversion factors. As discussed in detail above, all the results from the study show good correlation and so normalisation on organic carbon content gives a very reliable K_{oc} result. Given the use of substrates with a wide variety of characteristics in the study, the results can be applied with confidence as the basis of adsorption coefficients for environmental substrates in the EUSES model. Therefore it is not necessary to apply single results measured in the OECD 106 study for sediment, sludge or soil independently in the risk assessment.

Table 3.6 Adsorption coefficients used in the environmental risk assessment

Partition coefficient	Symbol	Values used
Organic carbon - water partition coefficient	K_{oc}	1780 l/kg
Solids - water partition coefficient for soil	Kp_{soil}	35.6 l/kg
Solids - water partition coefficient for sediment	Kp_{sed}	89 l/kg
Solid - water partition coefficient for suspended matter	Kp_{susp}	178 l/kg
Soil - water partition coefficient	$K_{soil\text{-}water}$	53.6 m^3/m^3
Sediment - water partition coefficient	$K_{sed\text{-}water}$	45.3 m^3/m^3
Suspended matter - water partition coefficient	$K_{susp\text{-}water}$	45.4 m^3/m^3

Precipitation

The low volatility and relatively high adsorption coefficient suggest that most TDCP found in the atmosphere will adsorb to particulate matter, which may then be washed out by rainfall. The TGD estimates this from vapour pressure, leading to a similar conclusion.

Volatilisation

A Henry's Law constant of 1.24 x 10^{-4} Pa.m^3/mol can be calculated from the vapour pressure and water solubility. This indicates a preference for water compared to air, and hence a low rate of volatilisation from surface water to air.

Distribution in wastewater treatment plants

It is assumed that no biodegradation occurs during wastewater treatment.

Based on the physico-chemical properties of TDCP (vapour pressure = 5.6 × 10^{-6} Pa, water solubility = 18.1 mg/l, Henry's law constant = 1.24 x 10^{-4} Pa m^3/mole and K_{oc} = 1780 l/kg) the predicted behaviour of the substance during wastewater treatment (as estimated by the SIMPLETREAT program within EUSES) is:

Fraction to air	0%
Fraction to surface water	82.1%
Fraction to sludge	17.9%
Fraction degraded	0%

Distribution in the environment

Distribution according to fugacity modelling

The approach to distribution modelling is described below. Two models have been used:

The 1997 EQC model, at Level I

The 1999 Level III model, using the EU default parameters.

The physicochemical properties entered were as given in section 1; K_{oc} is estimated by the program from K_{ow} as 2008, which is sufficiently close to the measured K_{oc} value that no adjustment is required to the input value of log K_{ow}.

The reaction half-lives have been set at negligible reaction in all compartments. For purposes of examining the importance of the value of K_{ow} and K_{oc}, the emissions were to air, water and soil.

The results obtained are shown in **Table 3.7**.

Table 3.7 Environmental distribution of TDCP for various models

	EQC Level I	Level III
% in air	0.0004	0
% in soil	79.8	98.1
% in water	18.4	1.74
% in sediment	1.77	0.14

The results for EQC level I (the simplest model) indicate that water, soil and sediment are all significant should TDCP be stable in the environment. Furthermore, the outputs of the model are sensitive to the K_{ow} (i.e. K_{oc}) input. The Level III result shows less substance in water because it accounts for mass flow of water out of the region being modelled.

The Level III model has been used to indicate the fate modelled for separate releases into different compartments. No inflow from outside the modelled area (the whole EU) has been included. The results are in **Table 3.8**.

Table 3.8 Output of fugacity model for various release scenarios

Release:	To air, water and soil	To air	To water	To soil
% in air	0	0.002	0	0
% in soil	98.1	98.8	0.057	98.9
% in water	1.74	1.08	92.7	1.04
% in sediment	0.14	0.084	7.22	0.081

The results reflect that most TDCP found in air would be precipitated to soil, and that there is very little movement between soil and water, because transfer via the air compartment is very slow. In water, the modelled adsorption to sediment is low.

Accumulation and metabolism

Aquatic organisms

Bioaccumulation studies available are summarised in **Table 3.9**.

Table 3.9 Studies of bioaccumulation of TDCP in fish

Year test completed	Protocol cited	Results	Reliability	Study reference
1992	MITI (OECD 305C)	42d BCF 0.3 – 22 at two concentrations over 6 weeks	(4) not assignable. Only a brief summary available.	CITI, 1992
1981		96h BCF 3-5 for goldfish, 77-113 for killifish	(3) invalid	Sasaki *et al,* 1981
1982		BCF 31-59 (continuous flow through system), 50-89 (static system)	(2) valid with restrictions. Acceptable though not clearly reported	Sasaki *et al*, 1982

Bioaccumulation

Bioaccumulation in fish has been assessed (CITI, 1992). The fish species used for this test was the carp (*Cyprinus carpio*). Test concentrations appear to be acceptable, being approximately 2 and 0.2% (20 and 2 μg/l) of LC_{50}, though LC_{50} values relate to other species.

Fish were kept in flow-through conditions for 28 days prior to exposure to test substance. The exposure period was 6-8 weeks following which the concentration in fish was determined (method not stated). BCFs of 0.3 – 3.3 and <2.2 – 22 were obtained for the two concentrations respectively. Bioconcentration is calculated as (concentration in fish)/(concentration in water).

TDCP and three other phosphates were investigated (Sasaki *et al,* 1981) in studies to estimate the log K_{ow}, acute toxicity to and bioconcentration in two species of fish, goldfish (*Carassius auratus*) and killifish (*Oryzias latipes*). .

The bioconcentration test was only 96 h long (the usual term of exposure is more than one week). Test fish were not fed or test vessels aerated in this time. The test system appears to be that used for the toxicity test (see section 3.3.1). The test concentration of 1 mg/l is unacceptably close to the LC_{50} reported in other sources (though this toxicity level was not observed in the species concerned). Estimated BCF is 3-5 for goldfish, 77-113 for killifish. Bioconcentration is here calculated as (concentration in fish)/(concentration in water).

The estimation of acute toxicity is very unusual and the results should not be considered further as it appears that only one test concentration was used for each test substance, the LC_{50} being estimated based on the number of survivals after 96 hours. Fish exposed to TDCP suffered a "characteristic manifestation of [organophosphorus] toxicity" – deformation of the spine. The estimated LC_{50} is 3.6 ppm in killifish, 5.1 ppm in goldfish.

A study of phosphate ester behaviour in killifish (Sasaki *et al*, 1982) discusses bioconcentration in this species of various substances using both static and flow-through systems. A 30-day exposure period was used. Bioconcentration ratios of 31-59 for TDCP were recorded for the continuous flow system; the paper quotes a previous static study in which BCFs of 50-89 were obtained; this has not been reviewed. A biological half-life of 1.65 hr was reported. Bioconcentration is calculated as (concentration in fish)/(concentration in water).

The short half-life is consistent with the rapid elimination seen in metabolic studies in the rat. Full details will be given in section 4.1.2.1.

The TGD gives a method for estimating the value of BCF in fish based on log K_{ow}. The appropriate equation is the linear equation for substances with log K_{ow} <6:

$$\text{Log } BCF_{fish} = 0.85 \log K_{ow} - 0.70$$

The log K_{ow} for TDCP is 3.69± 0.36. On the basis of the uncertainty on this value, a range of log BCF can be estimated. From the above equation, BCF_{fish} = 273.2 (range 135.1 – 552.7). The measured BCFs for TCPP and TDCP are relatively low in comparison with the predictions and with other substances of similar log K_{ow} values. There could be various causes for such a result, including the observed rapid metabolism in the organism. There is evidence for metabolism of both TDCP (which is discussed in Section 4.1.2.1) and TCPP (refer to HSA/EA, 2008a). TCEP has a similarly low measured BCF value and metabolism occurred in both *in vivo* toxicokinetics and *in vitro* studies.

The measured BCF of 45 l/kg is used in the risk assessment; this is the arithmetic mean of the range 31 to 59 l/kg. Since the values are in a narrow range, a mean is considered acceptable and representative.

Terrestrial organisms

The revised TGD gives a new method for estimating the value of BCF in earthworms based on log K_{ow}, using the method of Jager (1998):

$$BCF_{earthworm} = \frac{(0.84 + 0.012.K_{ow})}{RHO_{earthworm}}$$

For $RHO_{earthworm}$ by default a value of 1 kgwwt.L^{-1} can be assumed. The log K_{ow} for TDCP is 3.69± 0.36. On the basis of the uncertainty on this value, a range of log BCF can be estimated. From the above equation, $BCF_{earthworm}$ = 59.61 (range 26.50 – 135.48).

Aquatic compartment (including sediment)

$PEC_{sediment}$ is calculated using the equilibrium partitioning approach.

The value $Clocal_{effluent}$ for wastewater treatment plants is used as the value of PEC for WWTP micro-organisms.

Calculation of predicted environmental concentrations (PEC_{local})

The PECs for TDCP are calculated using the methods given in the Technical Guidance Document, except where site-specific assessment is appropriate and suitable acceptable data have been provided (more information is given in the Confidential Annex). Where a default local assessment applies, the usual models, equations and assumptions apply.

Some notes on the basis of PEC are given in **Table 3.10**.

Table 3.10 Notes on the basis of PECs for specific life cycle stages

	Life cycle stage	Basis of release rates to the environment
	Producer 1	Site specific data
	Producer 2	Site specific data
A1a	Flexible foam - automotive - foaming large site	Appendix B
A1b	Flexible foam - automotive – foaming	Appendix B
A2	Foam cutting	Appendix B
B1	Flexible foam - furniture – foaming	Appendix B
B2	Foam cutting	Appendix B
C1	CONFIDENTIAL	Estimates from relevant ESDs; read across from relevant previous published risk assessments; site specific info and WWTP details in some instances Note for application C (life cycle stages C1a and b and C2): industry has indicated that supply ceased in 2006 (pers. comm.. 3rd October 2007 and 30th October 2007, confidential).
C2	CONFIDENTIAL	
D1	CONFIDENTIAL	
D2	CONFIDENTIAL	
E1a	CONFIDENTIAL	
E1b	CONFIDENTIAL	
F1	CONFIDENTIAL	
G1	CONFIDENTIAL	
I1	Flexible foam - Furniture, seating, mattresses - re-bonding of scrap	Appendix B
J1	Loose Crumb	Appendix B

Calculation of PEC_{local} for production

PEC_{local} for production is based on site-specific, confidential details of effluent concentration and wastewater treatment plant size and function.

Table 3.11 Values used in calculation of PEC for production

	$C local_{effluent}$ [mg.l-1]	$C local_{water}$ [mg.l-1]	PEC_{water} [mg.l-1]	$PEC_{sediment}$ [mg.kg wwt-1]
Producer 1	0.0299	2.98E-04	3.20E-04	0.0126
Producer 2	1.19E-03	1.18E-05	3.41E-05	1.35E-03

Calculation of PEC_{local} for formulation

Formulation is not a relevant life cycle stage for TDCP.

Calculation of PEC_{local} for industrial/professional use

PEC_{local} values for industrial and professional use are calculated for all life cycle stages. Calculated PECs are summarised in **Table 3.12**.

Table 3.12 Values used in calculation of PEC for industrial and professional use

	$C_{local_{effluent}}$ [mg.l-1]	$C_{local_{water}}$ [mg.l-1]	PEC_{water} [mg.l-1]	$PEC_{sediment}$
A1a: Flexible foam – automotive - foaming large site	7.38E-05	7.36E-06	2.97E-05	1.17E-03
A1b: Flexible foam – automotive - foaming	9.24E-06	9.21E-07	2.32E-05	9.17E-04
A2: Foam cutting	3.28E-05	3.27E-06	2.56E-05	1.01E-03
B1: Flexible foam – furniture - foaming	7.36E-05	7.34E-06	2.96E-05	1.17E-03
B2: Foam cutting	1.37E-05	1.36E-06	2.37E-05	9.34E-04
C1a: CONFIDENTIAL[1]	*2.65E-03*	*2.64E-04*	*2.86E-04*	*0.0113*
C1b: CONFIDENTIAL[1]	*0.0684*	*6.82E-03*	*6.84E-03*	*0.27*
C2: CONFIDENTIAL[1]	*0.409*	*0.0408*	*0.0408*	*1.61*
D1: CONFIDENTIAL	0	0	2.23E-05	8.81E-04
D2: CONFIDENTIAL	0	0	2.23E-05	8.81E-04
E1a: CONFIDENTIAL	0.328	8.18E-05	1.04E-04	4.11E-03
E1b: CONFIDENTIAL	8.21E-03	8.18E-04	8.41E-04	0.0332
F1: CONFIDENTIAL	6.14E-03	6.13E-04	6.35E-04	0.0251
G1: CONFIDENTIAL	8.62E-04	8.59E-05	1.08E-04	4.27E-03
I1: Flexible foam – Furniture, seating, mattresses - re-bonding of scrap	0	0	2.23E-05	8.81E-04
J1: Loose Crumb	0	0	2.23E-05	8.81E-04

Note 1 The industry has confirmed that the confidential application C of TDCP (life cycle stages C1a and b and C2) is no longer applicable in Europe and supply has ceased. While risk characterisation has been performed for these life cycle stages, it should be recognised that the risks are no longer believed to be relevant.

Calculation of PEC_{local} for private use

Not applicable. In-service loss and waste remaining in the environment are characterised on a regional scale.

Calculation of PEC_{local} for disposal

Not included in the present assessment. As discussed in Section 3.1.4.2.1, in a survey of landfill leachate samples in the UK TDCP was not detected in any samples, with a detection limit of 10 µg/l. Emissions from landfill sites are therefore considered to be negligible.

Measured levels

All available data are summarised in **Table 3.21**.

Since no laboratory reports are supplied, validation and good laboratory practice cannot be verified by the Rapporteur. Therefore all results must be treated as of non-assignable reliability. Older results are of little value for comparison with any environmental concentrations predicted by modelling, although they do at least indicate that TDCP can be detected in the environment.

Monitoring data provided by regulatory authorities in England and Wales

The Environment Agency has provided some data on the environmental concentration of TDCP and its isomers in various media (pers. comm. 22nd December 2005). This information comprises five measurements of concentrations in groundwaters, taken between September 2004 and May 2005. The measurements range between concentrations of 20 – 240 ng/l in various locations in Sussex, Wiltshire, Devon and Hertfordshire. The values were collected as part of a screening assessment. The reliability is not assignable, but they should be considered for the exposure of man via drinking water.

Landfill leachate

As described in section 3.1.2.4, the Environment Agency of England and Wales has conducted some limited studies of the concentration of TDCP in leachate from 22 landfills in southern England and Wales. The data obtained (pers. comm., 3rd August 2005) show that TDCP was not detected in any samples for 18 different landfill sites, with a detection limit of 10 µg/l.

Freshwater sediments

In a study conducted on behalf of DEFRA (CEFAS, 2002), various samples were collected from around England and Wales during or prior to 2002. Freshwater sediments (50 samples) were analysed using LC-MS for selected chemicals including TDCP (lower limit of quantitation 10 ng/g ww for all matrices). TDCP was not detected in any samples.

Measured levels reported in the open literature

All available measured data are summarised in **Table 3.21**.

Measured levels in the EU

Water

TDCP has been detected in river water (Hendriks *et al,* 1994). Samples were taken in 1989 at various sampling points in the Netherlands Rhine delta. The sites of sampling are described relative to town and city outlets but without a fuller description of local industries it is difficult to judge the scale represented. The dissolved organics present in the water were concentrated using an XAD column (XAD is a methyl methacrylate ester resin).

Concentrations of 0-0.055 µg/l were found. However, due to the concentration/extraction procedure there are uncertainties in the concentrations determined and they 'may reflect a minimum'. The paper clearly identifies by CAS number the isomer of TDCP that is the subject of this risk assessment.

Eleven WWTP receiving waters were sampled and analysed as part of a wider study (Kuch *et al*, undated). The surface waters were sampled upstream and downstream of the receiving point of treated effluent from the respective WWTP. Details of the sampling regime and analytical methods are not presented. Chloroalkylphosphate FRs were predominantly detected in trace concentrations. TDCP was detected in surface waters at up to 0.74 µg/l. The concentrations of FRs were lower upstream of the WWTP receiving points than downstream, which clearly indicates that the FRs were being introduced to the river via the WWTP.

River water of the Ruhr and its tributaries and WWTP effluents were sampled at 38 locations in the Ruhr river system (Andresen *et al*, 2004). Samples were taken in September 2002, at a time of low water flow due to low rainfall. Some samples had also been sampled in July 2002 and comparative results are available. Analysis was by GC-MS; TDCP had a recovery rate of 95% and a limit of quantification of 14 ng/l.

Detailed analysis of TDCP in the river waters (Ruhr, Möhne, Lenne, and other tributaries) is not presented, however it is stated that near the mouth of the river Ruhr concentrations of ~50 ng/l were analysed. Samples of river water were also taken from the Rhine and Lippe rivers, for comparison with the above results. Analysis showed that TDCP was present at 13-36 ng/l and 17 ng/l respectively in Rhine and Lippe river waters.

Additional data are available for river water in the Netherlands. The following summary is taken from an RIVM report (2005):

Concentrations of TDCP from STP effluents in 2002/2003 (**Table 3.13**) were in the same order of magnitude as concentrations in surface water in 1989 (reported by Hendriks *et al.,* 1994). These data are monitoring data for several phosphate esters in the river Meuse and tributaries (data from Jeuken and Barreveld (2004)) and discharging effluents in comparison with effluents in Friesland (data from Berbee *et al*. (2004)).

Table 3.13 Monitoring data for several phosphate ester in the river Meuse and tributaries and discharging effluents

Location	Date	Max [µg/L]	Min [µg/L]
STP effluents (5) Meuse basin	12/2002-3/2003	0.45	0.15

Andresen *et al.* (2007) monitored for TDCP among other organophosphate compounds and other pollutants in the German Bight (an area heavily influenced by the Elbe estuary plume) in the North Sea (an area which receives outflow from several relatively highly-polluted European rivers). Data were also obtained for Lake Ontario, the most downstream of the Great Lakes, for comparison, but being of low relevance to the EU environment, these data are not discussed here.

Water samples were extracted using toluene, separated, dried and concentrated. Samples were analysed using GC-MS with quadrupole mass spectrometric detection, and equipped with a programmed temperature vaporiser injector. Extractions and analyses were both carried out in duplicate. Substance-specific recovery rates are not presented. A concentration of ~3 ng TDCP/l was measured in the River Elbe (near town of Stade).

TDCP was one of several organophosphates analysed for in a study of three drinking water purification plants, using a range of water treatment processes (Andresen and Bester, 2006). Samples were taken over a five-day period and analysed using GC/MS. Amounts of TDCP were reduced from 13 ng/l in the river Ruhr to 2.0 ng/l in the finished water at site A, 32 to 17 ng/l at site B, ~16 ng/l to ~3 ng/l at site C. Filtration with activated carbon was found to be the most effective treatment method for removal of TDCP and related substances.

In snow samples collected in northern Sweden, TDCP was detected in all samples (Marklund et al, 2005a. Snow samples were taken in March 2003, at a municipal airport, and in the vicinity of a road intersection. Samples were analysed using GC-NPD and GC/MS. Results are presented in **Table 3.14** below.

Table 3.14 TDCP concentrations in snow (Marklund *et al*, 2005a)

	Concentration (ng/kg snow)
Road 1	12
Road 2	230
Road 3	8
Airport 1	5
Airport 2	4
Airport 3	15

Sediments

Analysis of flame retardant compounds in sediments of the river Elbe has been undertaken (Heemken, Kuballa and Stachel, undated). Samples of freshly-deposited sediment were taken at ten sites in January-February 2001, the intention being to obtain a pollution profile along the river. TDCP was one of nine FRs analysed for. Analysis was by GC/MS. TDCP was detected in eight samples (the text implies that two samples were taken at each point though this is not stated explicitly; no FR occurs in more than 20 samples), at a concentration range of 8.9 – 44 μg/kg (mean 20 μg /kg).

Sediments were taken from the rivers Danube, Neckar and Rhine, as part of annual monitoring by the local environmental protection authority. The results are reported as part of a wider study (Kuch *et al*, undated). Details of the sampling regime and analytical methods are not presented. High concentrations in the sediments of the three rivers (up to 1.3 mg/kg dry weight) are noteworthy, since this suggests accumulation.

Sediments were sampled and analysed after a period of flooding of the Elbe (Stachel *et al*, 2005). The samples were taken following the flooding in September 2002 along the Elbe and at the mouths of its major tributaries. Samples were analysed using GC-FPD. Across 37 samples, concentrations of TDCP ranged between <1-13 μg/kg dwt, median 7.9 μg/kg dwt. The results show that only a few weeks after the flood, contaminant concentrations in solid matter were comparable to those prevailing beforehand. Significant sources of contaminant input are believed to include the tributaries Vltava (Moldau), Bilina (both in the Czech Republic), and the Mulde (Germany), as well as industrial and municipal WWTPs located along the Elbe. Ecotoxicological studies with two sediment organisms (*Chironomus riparius* and *Potamopyrgus antipodarum*) were also conducted (see section 3.3.1.1.6).

WWTP and other effluents

Twenty wastewater treatment plants and 4 disposal site effluents were sampled and analysed as part of a wider study (Kuch *et al*, undated). Details of the sampling regime and analytical methods are not presented. Chloroalkylphosphate FRs were predominantly detected in trace concentrations. The concentrations of FRs were lower upstream of the WWTP receiving points than downstream, which clearly indicates that the FRs were being introduced to the river via the WWTP. Concentrations of TDCP in treated effluent were up to 0.9 µg/l. Concentrations in disposal site effluents reached the mg/l range. However, after treatment with active charcoal the substances were no longer detectable by the analytical method used. This suggests that treatment using activated charcoal is suitable for effectively treating highly loaded effluents.

Two WWTPs, in Köln and Düsseldorf were sampled at different steps of the wastewater treatment process between February and March 2003 (NRW, 2003). The samples were analysed for certain chlorinated and non-chlorinated organophosphate esters. The report states that in a previous study of the STP of Düsseldorf, TDCP was eliminated up to 10%. However in this study no removal was apparent. At both WWTPs the efficiency of the cleaning process concerning the flame retardants was comparable so the type of construction of the WWTP does not seem to be relevant for the elimination of these substances. By comparison, non-chlorinated alkylphosphates were eliminated by 57-86% (Köln) and 60-85% (Düsseldorf). Concentrations of up to 1.35 µg/l TDCP were measured in treated effluent. Raw data are not presented. Median and maximum concentrations are shown in **Table 3.1**5.

Table 3.15 Concentrations of TDCP in treated effluent (NRW, 2003)

	Number of samples	Number > detection limit	Detection limit (µg/l)	Maximum value (µg/l)	Median (µg/l)	Elimination
Düsseldorf						
Influent	12	12	0.01	1.35	0.08	
Effluent	12	12	0.01	0.31	0.11	+38%
Köln						
Influent	12	12	0.01	0.18	0.086	
Effluent	12	12	0.01	0.18	0.12	+40%

In a very similar study (Fahlenkamp *et al*, 2004), samples from influent and effluent of two municipal wastewater treatment plants were analysed for organic contaminants. In both the Düsseldorf and Köln WWTPs, TDCP was present at approximately 0.1 µg/l in both influent and effluent.

In another very similar study (Meyer and Bester, 2004), influent and effluent from two unidentified WWTPs in the North Rhine-Westphalia region of Germany were sampled in Spring 2003 and analysed. Samples analysed were 24-hour composite samples. Details of the samples taken are given in **Table 3.16** and the results are summarised in **Table 3.17**.

Table 3.16 STPs sampled (Meyer and Bester, 2004)

	Wastewater volume (m^3/d)	Inhabitant equivalents	Fate of effluent	Sampling locations (see diagrams)
STP A	220,000	1,100,000	Receiving water not identified.	Influent stream, intermediate settling tank, final sedimentation tank, final effluent
STP B	108,959	1,090,000	Effluent passes into river Rhine	Influent, primary settling tank, final sedimentation tank, final effluent

STP A: Influent -> 1st aeration basin -> intermediate settling tank -> 2nd aeration basin -> final sedimentation tank -> Filter -> Effluent

STP B: Influent -> primary settling tank -> aeration basin -> final sedimentation tank -> Filter -> Effluent

Results showed concentrations of TDCP of up to 250 ng/l in influent and up to 310 ng/l in effluent.

Table 3.17 Concentrations of TDCP in various water streams Meyer and Bester, 2004)

	STP A (ng/l)	STP B (ng/l)
Influent	Max 180 Mean 100	Max 250 Mean 110
Intermediate settling tank / primary settling tank	Max 180 Mean 100	Max 120 Mean 62
final sedimentation tank	Max 180 Mean 110	Max 440 Mean 310
Effluent	Max 180 Mean 130	Max 310 Mean 150

There is no evidence of removal of either substance at either WWTP. The day-to-day variability in organophosphates at both WWTPs is described as 'extremely high'.

In a further very similar study, Friedrich *et al.* (2005) report TDCP concentrations in influent and effluent for municipal wastewater treatment plants Düsseldorf-Sud and Köln-Stammheim. Median concentrations suggest very low levels of removal in either treatment plant, with TDCP concentrations of ~0.1 μg/l in both influent and effluent of both treatment plants.

WWTP effluents were sampled at 38 locations in the Ruhr river system (Andresen *et al*, 2004). Samples were taken in September 2002, at a time of low water flow due to low rainfall. Some samples had also been sampled in July 2002 and comparative results are available. Analysis was by GC-MS; TDCP had a recovery rate of 95% and a limit of quantification of 14 ng/l. In STP effluents, concentrations of ~20-~120 ng/l TDCP were analysed.

It is clear that there is no consistent picture of removal, although the range of measured removals is not inconsistent with the SIMPLETREAT prediction of 17.9%. The possible significance of this observation is considered in the Conclusions.

Samples of influent water, effluent water and/or sludge from eleven Swedish WWTPs were analysed (Marklund *et al*., 2005b). It is stated that the sampling locations were selected on the basis of these WWTPs being small municipal plants with negligible industrial inflow; medium sized plants receiving water from large industrial sites; and large plants serving big

cities. However the results are not divided in these contexts. Information about flow and sludge volumes are presented as well as concentration data (for most sites data are available for single samples only). Analysis was by GC-NPD. The data are presented in **Table 3.18** below.

Table 3.18 TDCP in WWTP waters and sludges (Marklund *et al*., 2005b)

STP	Water volume m^3/d	Sludge volume t dw/y	Influent concentration ng/l	Effluent concentration ng/l	Sludge concentration ng/g dw
1	4700	170	250	270	3.3
	4700	170			3.4
2	140900	5800	450	180	49
	140900	5800	310	180	35
3	46100	3500	380	240	220
	46100	3500			230
4	317500	13900	330	130	92
	317500	13900	210	150	75
5 [1]	500	-	240	-	3
6	10300	790	310	340	21
7	14900	770	320	310	11
	14900	770			76
8	-	800			190
	-	800			260
9	-	240			7.3
10	-	14400			40
11	-	1900			41

Note 1: no biological treatment at site 5.

Rodil *et al.* (2005) reported concentrations of TDCP in raw wastewater, primary effluent and tertiary effluent (i.e. treated wastewater) of a WWTP, in a paper that focuses principally on analytical determination method and recovery. Samples were taken in August 2004 and analysed as 24-hour composite samples. TDCP concentrations varied from 0.21 µg/l (raw wastewater), 0.18 µg/l (primary effluent) to 0.13 µg/l (tertiary effluent).

Two WWTPs in the Frankfurt area were sampled in a study reported by Höhne and Püttmann (2006). TDCP was among a number of flame retardants analysed. The maximum influent concentrations were 1735 ng/l TDCP (Niederrad/Griesheim) and 1563 ng/l (Sindlingen); reducing to 394 ng/l and 408 ng/l respectively. Minimum and median concentrations suggest significant variability in levels of TDCP entering the Sindlingen plant as reported concentrations increase significantly in treated effluent (min. <LOD increasing to 96 ng/l; median 89 increasing to 177 ng/l).

Groundwater

Three groundwaters were sampled and analysed as part of a wider study (Kuch *et al*, undated). Two of the groundwaters were sampled from a location of high exposure. Details

of the sampling regime and analytical methods are not presented. Chloroalkylphosphate FRs were predominantly detected in trace concentrations; the limit of quantitation appears to be approximately 0.1 μg/l so it is assumed that TDCP was below this level in the groundwater samples.

Measured levels in Asia

Water

Behaviour of phosphate esters in Japanese waters has been investigated (Fukushima, Kawai and Yamaguchi, 1992. Monitoring for organophosphoric acid triesters has been regular since 1976 in the Yodo river basin, Yamato river and Osaka bay, Japan. River water is "typically polluted" by receiving various kinds of agricultural, domestic and industrial wastewaters with or without treatment – i.e. is likely to represent local post-WWTP levels.

Samples were analysed for various organophosphoric triesters using GC/MS and determined by GC with a flame photometric detector. Maps showing distribution of different levels are presented in the paper. TDCP levels were comparatively low (<0.5 μg/l in all samples analysed). Changes in the levels of TDCP in the Yodo river basin over time were assessed; levels had risen greatly in the 1976 – 1988 period, particularly in the generally more polluted areas. The nature of local industries in the areas surrounding the sampling sites is not set out.

The role of urban runoff in relation to chemical concentration in river waters was investigated (Fukushima *et al,* 1986). Only the abstract had been translated into English for review. River water was sampled on a rainy day, following a dry period. No information is available on the sampling regime or details of the analysis. Several substances were analysed for, including TDCP (identified by the name in English and the chemical structure). The maximum concentration of TDCP (ca 0.55 μg/l) was recorded for the 1600 and 2200 samples; the flow rate of the river is represented as reaching its peak between these times.

Further analysis of the changing load of the various substances (i.e. correcting for rainfall volumes) is performed. While some substances in the study increased in concentration as a result of rainfall, TDCP levels were found to remain constant, or drop slightly. This implies that no extra TDCP was being washed into the river by the rain.

Landfill leachate and Disposal sites

Seven controlled landfill sites and one open landfill in Japan were sampled (Yasuhara *et al,* 1997) for the assessment of leachate. The sites are said to be 'representative' though what they are representative of is not clear. Several different types of substance were analysed for and the methods of preparation, column temperatures etc. are set out in detail. TDCP was determined at a range of 2.8-1890 ng/l. individual results are not given and it is clear that the report is chiefly concerned with the techniques of extraction/determination.

In a follow-up study, nine controlled landfill sites and two open landfill sites in Japan were sampled in 1995 (Yasuhara *et al*, 1999) for the assessment of leachate. Several different types of substance were analysed for and the methods of preparation, column temperatures etc. are set out in detail. TDCP was determined at a level of up to 5500 ng/l (maximum concentration measured at a controlled site). There is no clear difference between open and controlled landfill site findings for TDCP (**Table 3.19**).

Table 3.19 TDCP levels found in Japanese landfill sites (ng/l)

Site no.	Type of rubbish	TOC[1]	Level of TDCP (ng/l)
1	Contains waste plastics. 4 yrs after completion of reclamation	14.5	230
2	Contains waste plastics and rubber. 14 yrs after completion of reclamation	22.5	620
3	Contains waste plastics, rubber and paper. 17 yrs after completion of reclamation	20.1	600
4	Over 70% domestic waste. 12 yrs after completion of reclamation	198	23
5	Contains waste plastics. Under reclamation	85.2	Not detected
6	Contains waste plastics. Under reclamation	40.1	3280
7	Open landfill – no hazardous wastes. Under reclamation	4.43	60
8	Mainly inorganic wastes but contains industrial incombustible rubber waste. 13 yrs after completion of reclamation	2.7	Not detected
9	Contains waste plastics. Open landfill – no hazardous wastes	12.9	880
10	Contains waste plastics. Under reclamation	188	140
11	Mainly waste plastics and rubber. Under reclamation	16.1	5500

Note: 1 Total Organic Carbon

Decomposition of phosphorus compounds has been reported (Kawai *et al,* 1993). Neither article nor abstract are translated from the Japanese. There is reference to TDCPP (another abbreviation used for TDCP) but there is no full chemical name or diagram so it is not clear whether this is the substance that is the subject of this risk assessment. Results are not translated.

Samples were taken from degradation ponds at a sea-based disposal site, as part of a recent study (Kawagoshi *et al*, 2002). The site is divided into three areas of which one takes solid wastes (presumably inert wastes) and two take dredged soils. Degradation of organophosphates was determined in seven different test conditions (presence and absence of sediments, aeration, presence and absence of biota). Initial concentration of TDCP was approximately 80 µg/l. TDCP was degraded under some test conditions but this is likely to be a consequence of adsorption to algae/sediment rather than degradation (this is acknowledged in the report).

Humans and animals

Levels of certain chemicals in fish have been investigated by Okumura (1994). Neither article nor abstract are translated from the Japanese. Results are presented graphically with English annotations and it is possible to see that the maximum measured level of TDCP in fish in the Yamato river was approx 31 ppb in early June and early August of the year of interest (not clear which year), and in the Okawa river approx 20 ppb in early June and late July of the same year.

Measured levels in North America

Water

Occurrence of various chemicals in American surface water has been investigated (Kolpin *et al.,* 2000). Stream water was sampled at 139 sites of varying conditions of geography, hydrogeology, land use, climate and basin size, in 1999-2000. The sites were selected due to being downstream of urban, agricultural or industrialised areas. The sampling and sample preparation procedures are described. Samples were analysed in duplicate. Analysis was by GC/MS. Many different types of contaminant were detected. TDCP was detected in 12.9% of 85 samples analysed, with a maximum concentration of 0.16 micrograms/l and a median detectable concentration of 0.1 μg/l.

Occurrence of phosphate esters in Canadian drinking water has been investigated (Williams and LeBel, 1981). The study considers tris(1,3-dichloropropyl)phosphate but does not confirm the structure so results may not be considered relevant to TDCP. 29 municipalities, selected so as to represent a variety of populations, locations and raw water sources, were sampled and tested for various organophosphate chemical types as named, among which TDCP was detected at a maximum of 23 ng/l (Brantford, August sample). There is some assessment of the sampling by river basin, water source type (lake, river, well or brook). The level of industrialisation associated with the different cities is not set out. Mean for cities sourced by river = 3.7 ng/l, for cities sourced by lake = 0.3 ng/l, for cities sourced by well = 0 ng/l. Drainage basin concentrations range from ≈0 (coasts) to 3.1 ng/l (St. Lawrence River).

Sampling and analysis of samples of raw and treated water from the Great Lakes has been undertaken (LeBel, Williams and Benoit, 1987). There are some concentration results for tris(1,3-dichloropropyl) phosphate (not identified by CAS number). The highest concentration determined is 2.5 ng/l, determined by GC/NPD.

Samples of Great Lakes waters were taken and the samples tested for mutagenicity (Williams *et al.,* 1982). Concentrations are reported for twelve sites (sampling twice in the year): highest concentration is 15.7 (presumably ng/l although units are not made clear). Results are for tris(1,3-dichloropropyl) phosphate.

Phosphate esters in drinking water have been analysed (LeBel, Williams and Benoit, 1981). The main focus of the article is concerned with the analytical method. Concentrations in water sampled at six Ontario water treatment plants are given. The highest measured concentration of TDCP (tris(1,3-dichloropropyl) phosphate, not identified by CAS number) is 1.8 ng/l.

Plants

Pine needles were sampled from nine sites (including one acceptable blank) in the Sierra Nevada foothills, for investigation of organophosphate pesticides (Aston *et al,* 1996); flame retardant substances were also unexpectedly found. Samples were taken in 1993 and 1994 from nearby but different sites. The relative positions of the sampling sites are shown with a crude indication of scale. The age of the trees sampled is not stated, nor is it stated that the same phase of the growing period was met with the different sampling times (there are three sampling times in 1993, separated by one-month and three-month gaps). There are no indications of phytotoxicity.

Needles were analysed on a compartmentalised basis, breaking down the needles into the polar surface compartment, non-polar surface compartment, cuticular wax and remainder of

needle. Sample fractions were analysed using GC and selective ion MS at m/z 383. Percent recovery is very good for TDCP except for the remainder of needle fraction.

The results are shown in **Table 3.20**. Very high levels were determined for two of the sites from the summer 1993 sampling, one of which was also sampled in 1994, showing a much lower level. Aerial deposition, either via dusts or rainfall, is cited as the cause (highest levels found in polar surface component in all cases) attributed to incineration of waste plastic items, from 'nearby point sources'.

Table 3.20 Concentration of TDCP in pine needles

Site	Polar surface concn. (ng/g wet wt.)	Non-polar surface concn. (ng/g wet wt.)	Cuticular wax concn. (ng/g wet wt.)	Remainder needle concn. (ng/g wet wt.)	Total (ng/g wet wt.)
1 (6/93)	1260	25.8	33.1	<LOQ[1]	1319
2 (7/93)	510	17.4	17.5	17.4	562
3 (7/93)	3.73	<LOQ[1]	<LOQ[1]	<LOQ[1]	3.7
4 (7/93)	41.8	<LOQ[1]	2.51	<LOQ[1]	44.3
5 (7/93)	8.20	<LOQ[1]	<LOQ[1]	11.6	19.8
6 (10/93)	9.86	<LOQ[1]	2.74	<LOQ[1]	12.6
1 (5/94)	29.0	8.55	5.63	<LOQ[1]	43.2
7 (5/94)	<LOQ[1]	<LOQ[1]	<LOQ[1]	<LOQ[1]	-
8 (5/94)	2.94	<LOQ[1]	<LOQ[1]	NOT DETECTED	2.9

Note [1] Limit of Quantification, not clearly reported.

Human tissue

TDCP has been identified in human seminal plasma (Hudec *et al,* 1981): however the sampling regime is not clear. Exposure is thought to be due to TDCP use as a flame retardant in Dacron and other consumer use of TDCP-flame retarded products, in addition to exposure via drinking water, and via the environment. Where TDCP was detected it was present at 5-50 ppb.

Occurrence of phosphate esters in human adipose tissue has been investigated (LeBel and Williams, 1986). The analytical method for determination of triaryl/alkyl phosphates in human tissue is referenced in LeBel and Williams, 1983. This paper includes the results of the previous work in addition to new results.

Adipose tissue samples were taken from hospitals in two cities. The fraction of samples in which TDCP was detected is slightly higher in one than the other (one-third of cases against one-fifth of cases, approximately). This may reflect the influence of local industry, though the area's industrialisation is not described. The range of levels detected did not vary significantly between the two sample sites. Results are for tris(1,3-dichloropropyl) phosphate.

Occurrence of phosphate esters in human adipose tissue has been investigated (LeBel, Williams and Berard, 1989). Samples of adipose tissue were taken from hospitals in six municipalities in Ontario. TDCP was not detected (detection limit of 1 ng/g) in four of the sampling sites. Of the two sampling sites at which TDCP was present, at one TDCP was detected in only one of sixteen samples, while at the other the frequency was almost half of the samples tested. A range of <1 – 32 ng/g TDCP applied for the highest detection site. This clearly indicates the influence of local activities, although there is insufficient information

about local industrialisation to draw any meaningful conclusions. Results are for tris(1,3-dichloropropyl) phosphate.

Table 3.21 TDCP concentrations in the environment

Sample type	Location	Sample period	Analytical method	Results	Scale represented	Reliability	Ref.
Surface water	EU: River Rhine in Netherlands	1989	GC/MS	Highest measured 0.055 µg/l	Unknown without further information.	(4) not assignable – no info on GLP etc. Measured values also questionable.	Hendriks *et al,* 1994
River water and treated drinking water	EU: River Ruhr	Not clear	GC/MS	Concentrations in river water 13, 32 and ~16 ng/l Concentrations in treated drinking water 2.0, 17 and ~3.0 ng/l	Unknown without further information.	(4) not assignable.	Andresen and Bester, 2006
WWTP receiving waters	EU: Germany	Not stated	Not stated	Max 0.74 µg/l	Local pre- and post-wastewater treatment.	(4) not assignable.	Kuch *et al*, undated
River waters	EU: River Ruhr and its tributaries including Möhne and Lenne; also Rhine and Lippe	Sept 2002	GC-MS	Ruhr and tributaries: max ~50 ng/l Rhine 13-36 ng/l Lippe 17 ng/l	Unclear but probably regional	(4) not assignable. No validation of storage and analysis	Andresen *et al*, 2004
River/estuarine water	EU: R. Elbe estuary	May-June 2005	GC-MS	3 ng/l	Unclear	(4) not assignable.	Andresen *et al.* (2007)
Snow	EU: Northern Sweden	March 2003	GC-NPD and GC/MS	Near road intersection: 8-230 ng/kg snow Airport: 4-15 ng/kg snow	Unclear	(4) not assignable	Marklund *et al*, 2005a
River sediments	EU: River Elbe	Jan-Feb 2001	GC/MS	Max 44 µg/kg, mean 20 µg/kg	Unknown	(2) valid with restrictions	Heemken, Kuballa and Stachel, undated
River sediments	EU: Rivers Danube, Neckar and Rhine	Not stated	Not stated	Max 1.3 mg/kg dry weight	Unclear	(4) not assignable.	Kuch *et al*, undated
River sediments	EU: River Elbe and tributaries	2002	GC-FPD	<1-13 µg/kg dwt, median 7.9 µg/kg dwt	Presumably local	(4) not assignable	Stachel *et al*, 2005

Sample type	Location	Sample period	Analytical method	Results	Scale represented	Reliability	Ref.
Freshwater sediments	EU: England and Wales	2002 or earlier	LC-MS	Not detected (<10 µg/kg wwt)	Unclear	(2) valid with restrictions	CEFAS, 2002
WWTP and disposal site effluents	EU: Germany			Treated effluent: max 0.9 µg/l Disposal site effluent: in mg/l range	Local (though the sources of TDCP are not made clear, and cannot be linked to specific life cycle stages)	(4) not assignable.	Kuch *et al*, undated
WWTP effluents	EU: Germany	Feb – March 2003		Treated effluent: max 1.35 µg/l	Local (though the sources of TDCP are not made clear, and cannot be linked to specific life cycle stages)	(4) not assignable.	NRW, 2003
WWTP effluents	EU: North Rhine-Westphalia	Spring 2003		Treated effluent: up to 310 ng/l	Local (though the sources of TDCP are not made clear, and cannot be linked to specific life cycle stages)	(4) not assignable.	Meyer and Bester, 2004
WWTP effluents	EU: WWTPs in Ruhr river system	Sept 2002	GC-MS	Treated effluent: ~20-~120 ng/l	Local (though the sources of TDCP are not made clear, and cannot be linked to specific life cycle stages)	(4) not assignable.	Andresen *et al*, 2004
WWTP effluents and sludges	EU: Swedish WWTPs	2003	GC-NPD	210 - 450 ng/l measured in influent wastewater 130 - 340 ng/l measured in treated wastewater 3 – 260 ng/g dw measured in sludge	Local (though the sources of TDCP are not made clear, and cannot be linked to specific life cycle stages)	(4) not assignable.	Marklund *et al.*, 2005b
WWTP effluents	EU: Germany	Not clear	Not clear	~0.1 µg/l in both influent and effluent of both treatment plants	Unknown	(4) not assignable	Friedrich *et al*. (2005)
WWTP effluents	Not clear	2004	LC-ESI-MS/MS	0.21 µg/l (raw wastewater), 0.18 µg/l (primary effluent) 0.13 µg/l	Unknown	(4) not assignable	Rodil *et al.* (2005)

Sample type	Location	Sample period	Analytical method	Results	Scale represented	Reliability	Ref.
				(tertiary effluent)			
WWTP effluents	EU: Germany (Frankfurt area)	Not clear	Not clear	Niederrad/Griesheim: Max 1735 ng/l (influent) Max 394 ng/l (effluent) Sindlingen Max 1563 ng/l (influent) Max 408 ng/l (effluent)	Unknown	(4) not assignable	Höhne and Püttmann (2006)
Landfill leachate	EU: UK (Environment Agency Thames, Anglian, Southern and Wales Regions)	2005	Not stated	18 sites with analysis for TDCP: all results <LOD (10 µg/l)	Local	(2) valid with restrictions	Pers. comm., 3rd August 2005
Ground water	EU: UK	2003-2004		20 ng/l – 240 ng/l	Unknown	(4) not assignable. Data acceptable	Environment Agency, pers. comm. 22nd December 2005
Ground waters	EU: Germany			Trace concentrations		(4) not assignable.	Kuch *et al*, undated
River water	Asia: Various rivers, Japan	1976-90	GC/MS	<0.5 µg/l	Probably local for private use.	(2) valid with restrictions – no info on GLP etc.	Fukushima, Kawai and Yamaguchi, 1992
River waters	Asia: Yodo river basin	Unknown	Unknown	Max 0.55 µg/l	unknown	(4) not assignable – important information not translated	Fukushima et al, 1986
Landfill site	Asia: Various sites, Japan	Unknown	GC/MS	2.8-1890 ng/l	Local for disposal stage (some sites specialise in plastic/rubber waste)	(2) valid with restrictions – no info on GLP etc.	Yasuhara *et al,* 1997
Landfill site	Asia: Various sites, Japan	1995	GC/FPD and GC/MS	Max 5500 ng/l	Local for disposal stage (some sites specialise in plastic/rubber waste)	(2) valid with restrictions – no info on GLP etc.	Yasuhara *et al,* 1999

Sample type	Location	Sample period	Analytical method	Results	Scale represented	Reliability	Ref.
Degradation ponds at sea-based disposal site	Asia: Japan			approximately 80 µg/l	Presumably represents local environment for disposal	(4) not assignable.	Kawagoshi *et al*, 2002
Unknown	Asia: Unknown (Japan)	Unknown	Unknown	Unknown	Unknown	(4) not assignable. Unacceptable without translation. Not clear which isomer	Kawai *et al,* 1993
River water	Asia: Osaka region, Japan	Unknown	Unknown	Max 31 µg/l	Unknown	(4) not assignable – important information not translated	Okumura 1994
River waters	North America: Various sites in USA	1999-2000	GC-MS	Max. 0.16 µg/l	Local urban/mixed sites	(2) valid with restrictions	Kolpin et al, 2000
Various surface waters	North America: Various sites, Canada	1979	GC/MS	Max. 23 ng/l	Unknown without further information	(2) valid with restrictions – no info on GLP etc.	Williams and LeBel, 1981
Surface water	North America: The Great Lakes		GC/NPD	2.5 ng/l	Probably regional	(2) valid with restrictions	LeBel, Williams and Benoit, 1987
Surface water	North America: Great Lakes	1980	See ref M10	Highest concentration 15.7 ng/l	Probably regional	(4) not assignable – no info on GLP etc. Results are not made clear	Williams *et al,* 1982
WWTP water	North America: Ontario	1978	GC, MS	Highest concentration 1.8 ng/l	Probably regional	(2) valid with restrictions – no info on GLP etc.	LeBel, Williams and Benoit, 19810
Pine needles	North America: Sierra Nevada, California	1993-94	GC/MS	Max 1319 ng/g wet wt.	Suggestion is that high levels due to local incineration of products – but lower levels due to	(2) valid with restrictions – no info on GLP etc.	Aston *et al,* 1996

Sample type	Location	Sample period	Analytical method	Results	Scale represented	Reliability	Ref.
					regional aerial deposition		
Human seminal plasma	North America			5 – 50 ppb	Represents exposure as a result of consumer use, in addition to exposure via the environment	(4) not assignable – no info on GLP etc. Results are not made clear	Hudec *et al*, 1981
Human adipose tissue	North America: Ontario		GC-NPD, GC-EIMS	Overall, 0.5 – 257.1 ng/g	Need more information	(2) valid with restrictions – no info on GLP etc.	LeBel and Williams, 1986
Human adipose tissue	North America: Ontario		GC-NPD, GC-EIMS	Range at highest detection sampling site, ND – 32 ng/g		(4) not assignable	LeBel, Williams and Berard, 1989

Comparison between predicted and measured levels

The existence of EU measurements of comparable magnitude to the modelled $PEC_{regional}$ value of 0.022 μg/l for water suggests that the predicted release rates are not unreasonable, since the predicted concentrations are within an order of magnitude of measured values.

UK monitoring data show that measured levels in freshwater sediments are less than 10 ng/g wwt (equivalent to 10 μg/kg wwt). The EUSES predicted concentrations at regional scale and many local scale endpoints are in agreement with this finding, though several predicted local sediment concentrations are higher than this limit of detection.

Terrestrial compartment

Calculation of PEC_{local}

The most significant contribution to $PEC_{local, soil}$ comes from spreading of WWTP sludge onto agricultural land. The PECs for TDCP are calculated using the methods given in the Technical Guidance Document, except where site-specific assessment is appropriate and suitable acceptable data have been provided (more information is given in the Confidential Annex). Where a default local assessment applies, the usual models, equations and assumptions apply.

Calculation of PEC_{local} for production

PEC_{local} for production is based on site specific, confidential details of effluent concentration and wastewater treatment plant size and function. Calculated PECs are summarised in **Table 3.22**.

Table 3.22 PECsoil for production

	Agric. soil 30 day average (mg/kg wet w t.)	Agric. soil 180 day average (mg/kg wet wt.)	Grassland 180 days average (mg/kg wet wt.)
Producer 1	1.02E-03	1.02E-03	1.04E-03
Producer 2	9.95E-03	9.92E-03	4.31E-03

Calculation of PEC_{local} for formulation

Formulation is not a relevant life cycle stage for TDCP.

Calculation of PEC_{local} for industrial/professional use

PEC_{local} values for industrial and professional use are calculated for all life cycle stages. Calculated PECs are summarised in **Table 3.23**.

Table 3.23 PECsoil for industrial and professional use

	Agric. soil 30 day average (mg/kg wet w t.)	Agric. soil 180 day average (mg/kg wet wt.)	Grassland 180 days average (mg/kg wet wt.)
A1a: Flexible foam – automotive - foaming large site	1.57E-03	1.57E-03	1.23E-03
A1b: Flexible foam – automotive - foaming	1.07E-03	1.07E-03	1.03E-03
A2: Foam cutting	1.25E-03	1.25E-03	1.10E-03
B1: Flexible foam - furniture – foaming	1.56E-03	1.56E-03	1.22E-03
B2: Foam cutting	1.10E-03	1.10E-03	1.04E-03
C1a: CONFIDENTIAL[1]	*0.0204*	*0.0207*	*0.0374*
C1b: CONFIDENTIAL[1]	*0.537*	*0.536*	*0.228*
C2: CONFIDENTIAL[1]	*3.09*	*3.08*	*1.14*
D1: CONFIDENTIAL	9.97E-04	9.97E-04	9.97E-04
D2: CONFIDENTIAL	2.03E-03	2.05E-03	2.94E-03
E1a: CONFIDENTIAL	0.043	0.043	0.043
E1b: CONFIDENTIAL	0.0631	0.0629	0.0242
F1: CONFIDENTIAL	0.0474	0.0473	0.0182
G1: CONFIDENTIAL	7.54E-03	7.51E-03	3.46E-03
I1: Flexible foam - Furniture, seating, mattresses - re-bonding of scrap	1.01E-03	1.01E-03	1.02E-03
J1: Loose Crumb	1.00E-03	1.00E-03	1.01E-03

Note 1 The industry has confirmed that the confidential application C of TDCP (life cycle stages C1a and b and C2) is no longer applicable in Europe and supply has ceased. While risk characterisation has been performed for these life cycle stages, it should be recognised that the risks are no longer believed to be relevant.

Calculation of PEC_{local} for private use

Not applicable. In-service loss and waste remaining in the environment are characterised on a regional scale.

Calculation of PEC_{local} for disposal

Not included in the present assessment, though preliminary research suggests that local scale exposure is possible due to WWTP treatment of landfill leachate. This is covered by discharge consents and is not a high priority in this risk assessment at this time.

Measured levels

No data are available for review.

Comparison between predicted and measured levels

No data are available for review.

Atmosphere

Given the low levels of releases, the low volatility and moderate adsorption coefficient of TDCP, together with its short predicted atmospheric half-life for degradation by hydroxyl radicals, it is not expected that exposure via the atmosphere will be significant.

The concentrations of TDCP in the atmosphere have been estimated using EUSES 2.0.3. The predicted local and regional atmospheric concentrations are shown in **Table 3.24**.

Table 3.24 Estimated air concentrations of TDCP

Scenario	Air concentrations (C_{local}) (mg/m³)		$PEC_{local(air),\ ann}$ (mg/m³)
	Emission episode	Annual average	
Producer 1	2.56E-07	6.31E-08	7.75E-08
Producer 2	1.43E-10	3.44E-11	1.44E-08
A1a: Flexible foam - automotive - foaming large site	5.00E-08	4.11E-08	5.55E-08
A1b: Flexible foam - automotive – foaming	6.26E-09	5.14E-09	1.95E-08
A2: Foam cutting	2.22E-08	1.83E-08	3.27E-08
B1: Flexible foam - furniture – foaming	4.99E-08	2.28E-08	3.72E-08
B2: Foam cutting	9.26E-09	7.61E-09	2.20E-08
C1a: CONFIDENTIAL[1]	*2.32E-04*	*5.71E-05*	*5.71E-05*
C1b: CONFIDENTIAL[1]	*2.32E-04*	*5.71E-05*	*5.71E-05*
C2: CONFIDENTIAL[1]	*3.46E-07*	*1.01E-07*	*1.15E-07*
D1: CONFIDENTIAL	0	0	1.44E-08
D2: CONFIDENTIAL	6.32E-06	3.05E-06	3.06E-06
E1a: CONFIDENTIAL	5.56E-06	2.28E-07	2.43E-07
E1b: CONFIDENTIAL	5.56E-06	3.81E-07	3.95E-07
F1: CONFIDENTIAL	4.99E-10	2.28E-10	1.46E-08
G1: CONFIDENTIAL	3.06E-07	8.38E-08	9.82E-08
I1: Flexible foam - Furniture, seating, mattresses - re-bonding of scrap	4.00E-08	3.28E-08	4.72E-08
J1: Loose Crumb	2.09E-08	1.41E-08	2.85E-08

Note 1 The industry has confirmed that the confidential application C of TDCP (life cycle stages C1a and b and C2) is no longer applicable in Europe and supply has ceased. While risk characterisation has been performed for these life cycle stages, it should be recognised that the risks are no longer believed to be relevant.

Some monitoring data for indoor air and environments have been obtained and these are presented in section 3.1.6.1. These are informative in terms of context for the models of release via volatilisation, but cannot be directly compared with predicted environmental concentrations from the risk assessment.

Measured levels reported in the open literature

The following measured data relate to indoor environments. All available data are summarised in **Table 3.29**.

Measured levels in Europe

Indoor environments

In a study conducted on behalf of the Swiss Federal Office of Public Health, air samples were analysed for FR content (Bürgi, 2002). Samples were taken in eleven locations: electronic appliance showrooms, open-plan offices, car interiors and a theatre. Air samples of approximately 2 m^3 were taken using polyurethane foam adsorbents, which were later extracted and analysed using GC-MS. TDCP was below the limit of detection (which is not stated in the translated excerpts) in all samples.

Settled dusts were collected from 15 environments including workplaces, domestic and public buildings, as part of a recent study (Marklund *et al*, 2003). (Indoor air has been sampled in similar environments (Marklund *et al.*, 2005c; see below for discussion and results)). Dust was collected from vacuum cleaner dust bags and also collection by hand in some cases. Wipe sampling was also used to look at surfaces. Dust samples were stored in glass jars in freezers prior to analysis. The samples were extracted using DCM with ultrasonication and analysis was by GC-NPD. TDCP was detected at the concentrations shown in **Table 3.25.**

Table 3.25 TDCP concentrations in settled dusts (Marklund *et al*, 2003)

Sampling location	**TDCP concentration** (mg/kg dust or ng/m^2 for computer screen and computer cover)
Home 1a	0.39
Home 2	1.1
Day care centre	1.8
Hospital wardsa	2.1
Hospital officeb	0.56
Radio shopb	0.59
Textile shop	0.20
Hotela	0.91
Prisonb	53
University lobby	5.7
Officeb	67
Librarya	0.84
Aircraftb	0.86
Cinemab	7.0
Public dance hallb	1.1
Computer screen	290
Computer cover	170

Notes a Average of three replicates.
b Average of two parallel samples.

The highest levels of TDCP were detected in office, prison, cinema and university lobby (all above 5 mg/kg dust). Office and lobby environments will be furnished with upholstered furniture and this is the most likely source. In the university lobby the upholstered furniture itself had actually been vacuumed. It has been indicated that foam mattresses and mattress coverings in prisons are heavily flame retarded due to the high fire and arson risks, which might explain the high levels detected in this environment (pers. comm., 27th July 2005). TDCP was found at significant concentration on the surface of computer screen/casing. It is unclear how this could have arisen as TDCP is not used in such materials; it could be due to adsorption.

It is unclear why the levels determined in public/occupational environments are so much higher than domestic environments, though the frequency of vacuuming may be a factor. The possible roles of variations in total dust load, dust type (e.g. composition, particle size) are mentioned in the report but no conclusions are drawn regarding the samples analysed. Overall, these findings support those of previous reports in the indication that TDCP can be detected in environments of use, which naturally leads to the conclusion that there is release in service.

The report also cites findings from previous work, including detection of TDCP in indoor atmospheres of buildings in Sweden and Japan at concentrations in the ng/m^3 range (Carlson *et al*, 1997 and Otake *et al*, 2001).

Settled and suspended dusts were collected in a recent study (Nagorka and Ullrich, 2003). Analysis was by GC-NPD and GC-MS. This report concentrates primarily on development of the analytical method. TDCP results in various samples are shown in **Table 3.26**.

Table 3.26 TDCP concentrations in dusts from various locations (reported in Nagorka and Ullrich, 2003)

Sample	Description	Results
Indoor air	Two rooms	<0.5 ng/m3 3 ng/m3 (standard deviation 16%)
Suspended dust	Two rooms	<0.5 ng/m3 3 ng/m^3
Settled dust	Two rooms	<0.5 mg/kg dust 7.68 mg/kg dust
	Three rooms	~0 mg/kg dust ~35 mg/kg dust ~5 mg/kg dust
	Results from a year-long collection Maximum concentration 95%-ile Arithmetic mean Geometric mean	 18.3 mg/kg dust 12.4 mg/kg dust 4.56 mg/kg dust 2.39 mg/kg dust

The report also cites findings from several other studies of house dusts and dusts from other buildings (including a kindergarten, and buildings with organophosphate FR building materials), in which TDCP was not detected.

Indoor air was sampled at twelve locations around Zurich: car interiors, a theatre, two furniture stores, three offices and three electronics stores (Hartmann *et al*, 2004). A single sample per site was taken via polyurethane foam plugs, with a sampling rate of 4 l/minute over a sampling period of 8 hours. Some overnight samples (6 or 14 hours) were taken. The precise location of air intake was chosen to be in the 'breathing zone' of workers or consumers in those locations. Samples were analysed by GC/MS, though a method recovery was not performed for TDCP (no reason is given). The limits of detection and quantification are 0.11 and 1.1 ng/m^3 respectively for TDCP. TDCP was not detected in any of the samples.

Indoor air has been sampled in various indoor environments (Marklund *et al.*, 2005c). Samples were collected using solid phase extraction tubes at a height chosen to represent the breathing zone of people working in the room. Analysis was by GC-NPD. The results are presented in **Table 3.27**.

Table 3.27 TDCP concentrations in indoor air (Marklund *et al*., 2005c)

	TDCP (ng/m3)
Home 1	<0.5
Home 2	<0.5
Day care centre	59
Hospital ward	150
Radio shop	<0.4
Textile shop	<0.2
Hotel	<0.6
Prison	6.0
University lobby	1.7
Office	35
Library	<0.7
Public dance hall	<0.2
Furniture store	0.8
Plastics Factory 1	0.4
Plastics Factory 2	<0.5
Bowling alley	<0.4
Laboratory	<0.3
Blank (n = 3)	<0.4

Another study investigated air concentrations of TDCP and other flame retardants in automobile interiors (Wensing *et al.*, 2004). Eight new vehicles were tested at approximately 20°C and 65°C, while flushing the vehicles with 0.6 m^3/h ultrapure nitrogen at 23°C and 50% relative humidity. A nine-month-old vehicle was also tested after being left outdoors at a temperature of 26°C (internal temperature 48°C). Samples were also taken from one new and one old car during a journey.

Samples were collected using the adsorbent WAD-2 which was later extracted and analysed using GC-MS. Results for TDCP are summarised in **Table 3.28**. As expected, measured air

concentrations of both substances were higher in the heated vehicles than at 20°C. However, during a journey, levels were found to drop below detection levels after twelve minutes.

Table 3.28 Summary results of Wensing *et al.* (2004)

Vehicle	Old		New (all vehicles)		New (single vehicle)			
Temp (°C)	481	202	201	651	651	652	502	402
TDCP (µg/m3)	< 0.1	< 0.2	< 0.01 – 0.20	0.07 – 8.64	8.64	0.47	< 0.53	< 0.34

[1]Stationary

[2] Measurement when travelling; the temperature range reflects the different parts of the vehicle in which the foam is used

Staaf and Östmann (2005) reported concentrations of TDCP among various organophosphate compounds in 29 indoor environments. TDCP concentrations were not detected (<1 ng/m^3) in ten private homes; not detected – 5 ng/m^3 in seven transport vehicles; not detected in three offices; not detected – 7 ng/m^3 in three workshops; not detected in four shops and not detected in three healthcare facilities.

Public domain and private use products

Various products suspected to be flame retarded were analysed (Sellstrom and Jansson, 1987). Of 104, TDCP was found in 11 samples. Five sample types are listed: sound absorbing materials (six of seven samples), shock absorbing materials (one of eight samples), mattresses (one of twelve samples), bus liners (two of eleven samples) and car liners (one of six samples). The material types are stated as mainly polyurethane products. The products were purchased in or near Stockholm. The contents of vacuum cleaner bags, originating from two houses of different ages, were also analysed. The results from these samples are not clear. Levels detected in the products are not stated, but the level of detection is 10 pg.

Levels in human blood samples were also analysed. The analytical method was not fully reviewed. The detection limit was 600 pg/ml. None of 37 blood samples exceeded this concentration.

This paper is useful for the use pattern only, although the blood analysis data may be useful for the assessment of human exposure.

Measured levels in Asia

Air

The occurrence of phosphate esters in ambient air has been investigated (Haraguchi, Yamashita and Shigemori, 1985). Neither article nor abstract are translated from the Japanese. There is reference to TDCPP (another abbreviation used for TDCP) but there is no full chemical name or diagram so it is not clear whether this is the substance is the isomer of TDCP that is the subject of this risk assessment.

Table 3.29 TDCP concentrations in the environment

Sample type	Location	Sample period	Analytical method	Results	Scale represented	Reliability	Ref.
Indoor environments	EU: Indoor air			<LOD	N/A	(4) not assignable.	Bürgi, 2002
Settled dust	EU: Workplaces, domestic and public buildings		GC-NPD	Levels above 5 mg/kg dust in several locations	Presumably represents local environment for in-service loss	(4) not assignable.	Marklund *et al*, 2003
Settled and suspended dusts			GC-NPD and GC-MS	Indoor air: <0.5-3 ng/m^3 Suspended dust: <0.5-3 ng/m^3 dust Settled dust: ~0 – 35 mg/kd dust; 95%ile from year-long collection = 12.4 mg/kd dust	Presumably represents local environment for in-service loss	(4) not assignable.	Nagorka and Ullrich, 2003
Indoor air	Europe: Zurich		GC/MS	Vehicles: ND (<0.11 ng/m^3) Buildings: ND (<0.11 ng/m^3)	Buildings presumably represent local environment for in-service loss	(4) not assignable.	Hartmann *et al*, 2004
Indoor air	Europe: Sweden		GC-NPD	<0.2 – 150 ng/m^3 Concentrations above 10 ng/m^3 seen in Day Care Centre, Hospital Ward and Office (59, 150 and 35 ng/m^3 respectively)	Buildings presumably represent local environment for in-service loss	(4) not assignable.	Marklund *et al*, 2005c
Indoor air	Europe: Sweden		GC-NPD	Not detected-7 ng/m^3 in a range of indoor environments. Only concentrations above limit of detection (1 ng/m^3) seen in one transport vehicle and one workshop.	Buildings presumably represent local environment for in-service loss	(4) not assignable.	Staaf and Östmann (2005)
Various products	EU: Purchased in	Not stated.	NCIMS, GC/ECD	Unknown	N/A	(2) valid with restrictions – not GLP. Acceptable as an indication of some	Sellstrom and Jansson,

Sample type	Location	Sample period	Analytical method	Results	Scale represented	Reliability	Ref.
	Stockholm					applications.	1987
Air	Asia: Unknown	Unknown	Unknown	Unknown	Unknown	(4) not assignable – important information not translated	Haraguchi, Yamashita and Shigemori, 1985

Secondary poisoning

The concentrations of contaminant in food (fish or worms) of fish- or worm-eating predators ($PEC_{oral,\ predator,\ fish}$ and $PEC_{oral,\ predator,\ earthworm}$) are calculated in accordance with the TGD.

Table 3.30 below sets out the values of $PEC_{oral,\ predator}$ for fish and earthworm predators for each life cycle stage. The regional background contribution to the value is already accounted for and is not evaluated separately. The regional background level does not in itself constitute a risk, and for most life cycle stages its contribution to local PEC is not significant.

Table 3.30 PEC values for secondary poisoning

	$PEC_{oral,\ predator,\ fish}$ [mg.kg-1]	**$PEC_{oral,\ predator,\ earthworm}$ [mg.kg-1]**
Producer 1	2.66E-03	2.02E-03
Producer 2	1.07E-03	0.01
A1a: Flexible foam - automotive – foaming large site	1.14E-03	2.51E-03
A1b: Flexible foam - automotive – foaming	1.02E-03	2.06E-03
A2: Foam cutting	1.06E-03	2.22E-03
B1: Flexible foam - furniture - foaming	1.08E-03	2.50E-03
B2: Foam cutting	1.03E-03	2.09E-03
C1a: CONFIDENTIAL	2.47E-03	0.0197
C1b: CONFIDENTIAL	0.0388	0.483
C2: CONFIDENTIAL	0.267	2.77
D1: CONFIDENTIAL	0.122	0.86
D2: CONFIDENTIAL	1.00E-03	2.94E-03
E1a: CONFIDENTIAL	1.08E-03	0.0398
E1b: CONFIDENTIAL	2.26E-03	0.0577
F1: CONFIDENTIAL	7.31E-03	0.0436
G1: CONFIDENTIAL	1.53E-03	7.86E-03
I1: Flexible foam - Furniture, seating, mattresses - re-bonding of scrap	1.00E-03	2.01E-03
J1: Loose Crumb	1.00E-03	2.00E-03

Calculation of $PEC_{regional}$ and $PEC_{continental}$

$PEC_{regional(water)}$ = 2.24E-05 mg/l from the EUSES v2.03 model.

$PEC_{regional(freshwater\ sediment)}$ = 1.38E-03 mg/kg wwt from the EUSES v2.03 model.

$PEC_{regional(soil)}$ = 1.22E-03 mg/kg wwt from the EUSES v2.03 model.

$PEC_{continental(water)}$ = 2.26E-06 mg/l from the EUSES v2.03 model.

$PEC_{continental(freshwater\ sediment)}$ = 1.39E-04 mg/kg wwt from the EUSES v2.03 model.

$PEC_{continental(soil)}$ = 5.56E-05 mg/kg wwt from the EUSES v2.03 model

MARINE EXPOSURE ASSESSMENT

General Discussion

The marine PECs for TDCP are calculated using the methods given in the Technical Guidance Document.

TDCP does not contain any ionisable functional groups, therefore the partition coefficients derived for the freshwater assessment can be used without adjustment.

Degradation

TDCP is not significantly biodegradable on the basis of freshwater tests. It is considered to be persistent in the marine environment.

Calculation of Predicted Environmental Concentrations (PEC)

For the local assessment it is assumed that industrial effluents are not treated in a municipal biological STP and a dilution factor of 100 can be assumed for discharges to coastal regions.

Values of $PEC_{regional(seawater)}$, $C_{local\ seawater}$, $PEC_{local(seawater)}$ and $PEC_{local,sed}$ are evaluated in accordance with the revised TGD.

Calculation of PEC_{local} for production

PEC_{local} for production is based on site specific, confidential details of effluent concentration and wastewater treatment plant size and function. Calculated PECs are summarised in **Table 3.31**.

Table 3.31 Marine PECs for production

	$PEC_{sea\ water}$	$PEC_{marine\ sediment}$ [mg.kgwwt-1]
Producer 1	3.00E-04	0.0119
Producer 2	1.66E-05	6.57E-04

Calculation of PEClocal for formulation

Formulation is not a relevant life cycle stage for TDCP.

Calculation of PEClocal for industrial/professional use

PEC_{local} values for industrial and professional use are calculated for all life cycle stages. Calculated PECs are summarised in **Table 3.32**.

Table 3.32 Marine PECs for industrial and professional use

	$PEC_{sea\ water}$ [mg.l-1]	**$PEC_{marine\ sediment}$ [mg.kgwwt-1]**
A1a: Flexible foam - automotive - foaming large site	3.13E-06	1.24E-04
A1b: Flexible foam - automotive - foaming	2.34E-06	9.26E-05
A2: Foam cutting	2.63E-06	1.04E-04
B1: Flexible foam - furniture - foaming	3.13E-06	1.23E-04
B2: Foam cutting	2.40E-06	9.47E-05
C1a: CONFIDENTIAL[1]	*3.44E-05*	*1.36E-03*
C1b: CONFIDENTIAL[1]	*8.33E-04*	*0.0329*
C2: CONFIDENTIAL[1]	*4.97E-03*	*0.196*
D1: CONFIDENTIAL	2.23E-06	8.81E-05
D2: CONFIDENTIAL	2.23E-06	8.81E-05
E1a: CONFIDENTIAL	1.02E-04	4.03E-03
E1b: CONFIDENTIAL	1.02E-04	4.03E-03
F1: CONFIDENTIAL	7.69E-05	3.04E-03
G1: CONFIDENTIAL	1.27E-05	5.02E-04
I1: Flexible foam - Furniture, seating, mattresses - re-bonding of scrap	2.23E-06	8.81E-05
J1: Loose Crumb	2.23E-06	8.81E-05

Note 1 The industry has confirmed that the confidential application C of TDCP (life cycle stages C1a and b and C2) is no longer applicable in Europe and supply has ceased. While risk characterisation has been performed for these life cycle stages, it should be recognised that the risks are no longer believed to be relevant.

Calculation of PEClocal for private use

Not applicable. In-service loss and waste remaining in the environment are characterised on a regional scale.

Calculation of PEC_{local} for disposal

Not included in the present assessment, though preliminary research suggests that local scale exposure is possible due to WWTP treatment of landfill leachate. This is covered by consents and is not a high priority in this risk assessment at this time.

Measured levels

Andresen *et al.* (2007) monitored for TDCP among other organophosphate compounds and other pollutants in the German Bight in the North Sea (an area which receives outflow from

several relatively highly-polluted European rivers). The German Bight is an area heavily influenced by the Elbe estuary plume. Seawater samples were taken in May-June 2005 in various locations in the North Sea, at a depth of 5 m.

Water samples were extracted using toluene, separated, dried and concentrated. Samples were analysed using GC-MS with quadrupole mass spectrometric detection, and equipped with a programmed temperature vaporiser injector. Extractions and analyses were both carried out in duplicate. Substance-specific recovery rates are not presented.

At the mouth of the River Elbe a concentration of ~3.5 ng TDCP/l was measured. In the Bight, concentrations of ~0.6 to 3 ng/l were measured, with lowest concentrations seen in waters furthest offshore.

Comparison between predicted and measured levels

The available data most likely relate to the regional scale, though the data relating to the river mouth and estuary could be considered local if the River Elbe is a receiving water for industrial sites where relevant life cycle stages take place (it is not known whether this is the case). Local PECs range between 2E-06 to 0.005 mg/l. The predicted regional PEC for marine water is 2.23E-06 mg/l (equivalent to ~2 ng/l). The measured data, derived from a relatively limited number of samples, range from ~0.6 to 3.5 ng/l. The measured data are therefore well in line with the modelled regional concentration and lower range local concentrations.

Secondary poisoning

The concentrations of contaminant in the marine food chain are calculated in accordance with the TGD.

Table 3.33 sets out the values of $PEC_{oral,\ predator}$ for marine predators for each life cycle stage. The regional background contribution to the value is already accounted for and is not evaluated separately. The regional background level does not in itself constitute a risk, and for most life cycle stages its contribution to local PEC is not significant.

Measured levels

Marine predators

In a study conducted on behalf of DEFRA (CEFAS, 2002), various samples were collected from around England and Wales during or prior to 2002. Porpoise (25 samples) and cormorant (28 liver samples) samples were analysed using LC-MS for selected chemicals including TDCP (lower limit of quantitation 10 ng/g ww for all matrices). TDCP was not detected in any samples.

Table 3.33 PECs for marine secondary poisoning

	$PEC_{oral,\ predator,\ fish\ (marine)}$ [mg.kgwwt-1]	$PEC_{oral\ marine\ top\ predator}$ [mg.kgwwt-1]
Producer 1	1.75E-03	4.31E-04
Producer 2	1.79E-04	1.16E-04
A1a: Flexible foam - automotive – foaming large site	1.17E-04	1.04E-04
A1b: Flexible foam - automotive – foaming	1.03E-04	1.01E-04
A2: Foam cutting	1.08E-04	1.02E-04
B1: Flexible foam - furniture - foaming	1.10E-04	1.02E-04
B2: Foam cutting	1.04E-04	1.01E-04
C1a: CONFIDENTIAL[1]	*2.79E-04*	*1.36E-04*
C1b: CONFIDENTIAL[1]	*4.71E-03*	*1.02E-03*
C2: CONFIDENTIAL[1]	*0.0326*	*6.59E-03*
D1: CONFIDENTIAL	1.00E-04	1.00E-04
D2: CONFIDENTIAL	1.00E-04	1.00E-04
E1a: CONFIDENTIAL	1.93E-04	1.19E-04
E1b: CONFIDENTIAL	2.54E-04	1.31E-04
F1: CONFIDENTIAL	8.69E-04	2.54E-04
G1: CONFIDENTIAL	1.65E-04	1.13E-04
I1: Flexible foam - Furniture, seating, mattresses - re-bonding of scrap	1.00E-04	1.00E-04
J1: Loose Crumb	1.00E-04	1.00E-04

Note 1 The industry has confirmed that the confidential application C of TDCP (life cycle stages C1a and b and C2) is no longer applicable in Europe and supply has ceased. While risk characterisation has been performed for these life cycle stages, it should be recognised that the risks are no longer believed to be relevant.

Comparison between predicted and measured levels

UK monitoring data show that measured levels in marine predators (cormorants and porpoise) are less than 10 ng/g wwt (equivalent to 10 μg/kg wwt). The EUSES predicted concentrations are in agreement with this finding.

Calculation of $PEC_{regional}$ and $PEC_{continental}$

$PEC_{regional(sea\ water)}$ = 2.23E-06 mg/l from the EUSES v2.03 model

$PEC_{regional\ (marine\ sediment)}$ = 1.15E-04 mg/kg wwt from the EUSES v2.03 model.

$PEC_{continental(sea\ water)}$ = 6.69E-08 mg/l from the EUSES v2.03 model.

$PEC_{continental\ (marine\ sediment)}$ = 3.45E-06 mg/kg wwt from the EUSES v2.03 model.

EFFECTS ASSESSMENT: HAZARD IDENTIFICATION AND DOSE (CONCENTRATION) - RESPONSE (EFFECT ASSESSMENT)

The following Sections review the available toxicity data for TDCP with aquatic and terrestrial organisms. A reliability assessment is given for each study (this appears in the summary Tables within each Section). The assessment is based on the Klimisch system, which includes the following categories:

1 **Reliable without restriction**. "studies or data…generated according to generally valid and/or internationally accepted testing guidelines (preferably according to GLP) or in which the test parameters documented are based on a specific (national) testing guideline….or in which all parameters described are closely related/comparable to a guideline method."

2 **Reliable with restrictions**. "studies or data….(mostly not performed according to GLP), in which the test parameters documented do not totally comply with the specific testing guidelines, but are sufficient to accept the data or in which investigations are described which cannot be subsumed under a testing guideline, but which are nevertheless well documented and scientifically acceptable."

3 **Not reliable**. "studies or data….in which there were interferences between the measuring system and the test substance or in which organisms/test systems were used which are not relevant in relation to the exposure (e.g., unphysiologic pathways of application) or which were carried out or generated according to a method which is not acceptable, the documentation of which is not sufficient for assessment and which is not convincing for an expert judgement."

4 **Not assignable.** "studies or data….which do not give sufficient experimental details and which are only listed in short abstracts or secondary literature (books, reviews, etc.)."

In terms of the risk assessment, toxicity data assigned a reliability assessment of 1 or 2 will be considered in preference to the other toxicity data when deriving the PNEC.

The extent to which TDCP impurities could influence the toxicity of test media has been assessed. None of the known impurities are considered to have properties that would have significantly influenced the toxicity of the TDCP samples used in the tests reported below.

Aquatic compartment (including sediment)

Reports of ecotoxicity tests carried out with TDCP on fish (acute), aquatic invertebrates (acute and chronic), and algae (acute/chronic) have been reviewed.

Toxicity test results

The contents of the test reports are summarised below and in **Table 3.34**.

Table 3.34 Summary of aquatic toxicity test results for TDCP

Test species	Test protocol	Year test completed	Endpoint and exposure period	Result (mg/l)[1]	Reliability assessment	Comments	Study reference
Toxicity to fish							
Rainbow trout (*Oncorhynchus mykiss*)	OECD 203	1989	96-h NOEC 24-h LC_{50} 48-h LC_{50} 72-h LC_{50} 96-h LC_{50}	<0.6 (N) 1.4 (N) 1.4 (N) 1.4 (N) 1.4 (N)	(3) invalid	Static test. The test was not supported by analysis of exposure concentrations. This factor is considered important in view of comments relating to Ref. Sasaki, K., Takeda, M. and Uchiyama, M. (1981).	Jenkins, (1990)
Rainbow trout (*Oncorhynchus mykiss*)	OECD 203	1993	96-h NOEC 24-h LC_{50} 48-h LC_{50} 72-h LC_{50} 96-h LC_{50}	0.56 (N) 1.8 (N) 1.5 (N) 1.3 (N) 1.1 (N)	(2) valid with restrictions	Semi-static test. The test was not supported by analysis of exposure concentrations but in other respects was considered to be acceptable. The result is below the reported water solubility of TDCP of 18 mg/l.	Sewell, (1993a)
Killifish (*Oryzias latipes*)	Method not specified	1981	96-h LC_{50}	3.6 (N)	(3) invalid	Static test. An associated stability study showed that at an initial concentration of approximately 1 mg/l declined by 50% over 96 hours in the presence of fish. However the test media were not renewed, the exposure concentrations were not analysed and the results are expressed relative to nominal.	Sasaki, Takeda, and Uchiyama (1981)
Killifish (*Oryzias latipes*)	Japanese Industrial Standard (JIS K 0102-1986-71)	1992	48-h LC_{50}	3.7 (N)	(4) not assignable	Only a summary report was available for review. The test was not supported by analysis of exposure concentrations. This factor is considered important in view of comments relating to Ref. Sasaki, K., Takeda, M. and Uchiyama, M. (1981). A 48-h LC_{50} value has only been determined.	CITI (1992)
Goldfish (*Carassius auratus*)	Method not specified	1981	96-h LC_{50}	5.1 (N)	(3) invalid	Static test. An associated stability study showed that at an initial concentration of approximately 1 mg/l declined by 50% over 96 hours in the presence of fish. However the test media were not renewed, the exposure concentrations were not analysed and the results are expressed relative to nominal.	Sasaki, Takeda, and Uchiyama, (1981)
Goldfish (*Carassius auratus*)	Method not specified	1979	3-h LC_{50}	<30	(4) not assignable	Secondary literature cited by US EPA in Flame Retardant Alternatives – Tris(1,3-dichloro-2-propyl) phosphate – Hazard Review (http://www.epa.gov/dfe/pubs/flameret/altrep-v2/altrept-v2-section3a.pdf)	Ahrens, VD; Maylin, GA; Henion, JD; et al. (1979)
Goldfish (*Carassius auratus*)	Method not specified	1977	24-h LC_{50}	1-5	(4) not assignable	Secondary literature cited by US EPA in Flame Retardant Alternatives – Tris(1,3-dichloro-2-propyl) phosphate – Hazard Review (http://www.epa.gov/dfe/pubs/flameret/altrep-v2/altrept-v2-section3a.pdf)	Eldefrawi, AT; Mansour, NA; Brattsten, LB; et al. (1977)

Test species	Test protocol	Year test completed	Endpoint and exposure period	Result (mg/l)[1]	Reliability assessment	Comments	Study reference
Fish - acute QSAR (Esters)	ECOSAR (version 0.99g)		96-h LC_{50}	8.1		The estimated values are of the same order as the measured values. The estimates were obtained using measured physicochemical data as inputs to the model.	
Fish – acute QSAR (Phosphate esters)	ECOSAR (version 0.99g)		96-h LC_{50}	4.5			
Fish – chronic QSAR (Esters)	ECOSAR (version 0.99g)		NOEC	1.0			
Toxicity to aquatic invertebrates							
Cladoceran (*Daphnia magna*)	OECD 202; U.S. EPA Series 850 – Ecological Effects Test Guidelines OPPTS Number 850.1010	1999	48-h NOEC 24-h EC_{50} 48-h EC_{50}	1.6 >5.1 3.8	(1) valid without restriction	Fulfils all the reliability criteria. Results are expressed relative to mean measured concentrations. The study was subject to GLP.	Drottar, Kendall, and Krueger (1999)
Cladoceran (*Daphnia magna*)	OECD 202	1993	48-h NOEC 24-h EC_{50} 48-h EC_{50}	1.8 (N) 5.5 (N) 4.6 (N)	(3) invalid	The test was not supported by analysis of exposure concentrations. The pattern of mortality did not change much beyond the first day and consequently there are questions over the stability of the exposure concentrations. However the result is similar to that obtained in a fully valid test.	Sewell, (1993b)
Invertebrate - acute QSAR (Esters)	ECOSAR (version 0.99g)		48-h LC_{50}	9.9		The estimated value is of the same order as the measured values. The estimates were obtained using measured physicochemical data as inputs to the model.	
Cladoceran (*Daphnia magna*)	211 (Semi-static)	2004	21-day LOEC repro 21-day NOEC repro	1.0 (N) 0.5 (N)	(1) valid without restriction	Fulfils all the reliability criteria. Test concentrations were stable and within +/-20% of nominal. Results are therefore expressed relative to nominal concentrations. The study was subject to GLP.	Thomas et al (2004)
Invertebrate – longer term repro QSAR (Neutral organics)	ECOSAR (version 0.99h)		16-d EC_{50} (reproduction)	1.1		A recommended valid QSAR method is not readily available for the endpoint of chronic invertebrate. The method used, while the most appropriate from ECOSAR for this substance, is not recommended by ECOSAR for this type of compound and the QSAR is not well validated.	

Test species	Test protocol	Year test completed	Endpoint and exposure period	Result (mg/l)[1]	Reliability assessment	Comments	Study reference
						The estimate was obtained using measured physicochemical data as inputs to the model.	
Toxicity to algae							
Freshwater alga (*Pseudokirchneriella subcapitata*)	OECD 201; EEC Dir 92/69/EEC, Method C3	2004	72-h ErC_{10} (growth rate) 72-h EbC_{10} (biomass) 72-h ErC_{50} (growth rate) 72-h EbC_{50} (biomass) NOEC	2.3 (M) 1.2 (M) 4.6 (M) 2.8 (M) ≥1.2 (M)	(1) valid without restriction	Fulfils all the reliability criteria. Results are expressed relative to mean measured concentrations. The study was subject to GLP.	Desjardins (2004)
Freshwater alga (*Selenastrum capricornutum)* Note: now known as *Pseudokirchneriella subcapitata*	OECD 201; EEC DOC 89/88/XI, Directive 79/831, Annex V-C3	1991/2000	96-h NOEC 96-h ErC_{50} (growth rate) 96-h EbC_{50} (biomass)	6 (N) 39 (N) 12 (N)	(3) invalid	The test was not supported by chemical analysis and test media were prepared by dilution of a concentrated (1086 mg/l) stock suspension in which the water solubility of the substance (18 mg/l) was substantially exceeded. Consequently there is significant uncertainty over the agreement between nominal and actual exposure concentrations.	Kroon and van Ginkel (1992).
Freshwater alga (*Scenedesmus subspicatus*)	OECD 201 (Limit test)	1994	72-h NOEC 72-h ErC50 (growth rate) 72-h EbC50 (biomass)	≥10 (N) >10 (N) >10 (N)	(2) valid with restrictions	The test was not supported by chemical analysis and was not subject to GLP.	Sewell, (1994)
Algae QSAR (Esters)	ECOSAR (version 0.99g)		96-h EC_{50} 96-h NOEC	0.69 0.55		The estimated values are lower than the measured values. The estimates were obtained using measured physicochemical data as inputs to the model.	
Toxicity to micro-organisms							
Activated sludge	OECD 209	1990	IC_{50}	>10000 (N)	(2) valid with restrictions	Concentrations of 1 mg/l, 10 mg/l, 100 mg/l, 1000 mg/l, 10 g/l were used, and no inhibition of the respiration of activated sludge was observed.	Jenkins, (1990b)

Test species	Test protocol	Year test completed	Endpoint and exposure period	Result (mg/l)[1]	Reliability assessment	Comments	Study reference
Toxicity to sediment dwelling organisms							
Chironomus riparius (midge)	28-day test based on OECD guideline 218 (February 2001)	2006	Day 0-3 NOEC development 28-day NOEC development 28-day LOEC development 28-day EC50 emergence	8.8 mg/kg dwt (3-day geometric mean measured) (13 mg/kg dwt (nominal)) 3.9 mg/kg dwt (28-day time-weighted geometric mean measured) (13 mg/kg dwt (nominal)) 8.5 mg/kg dwt (28-day time-weighted geometric mean measured) (25 mg/kg dwt (nominal)) 16 mg/kg dwt (28-day time-weighted geometric mean measured) (34 mg/kg dwt (nominal))	(1) valid without restriction	Fulfils all the reliability criteria. The study was subject to GLP. The test results are quoted in the test report relative to nominal and initial measured concentrations. For the purposes of deriving a PNEC for risk assessment the NOEC for effects on development has subsequently been recalculated on the basis of geometric mean measured concentrations for the first three days of the test. The 3-day related value is considered to cover the most susceptible first instar phase of the life-cycle of the test organisms and a period of the test when the exposure concentrations were reasonably close to the target values (see section 3.3.1.1.6 for full discussion and justification). The NOEC for effects on development and the EC50 for effects on adult emergence have also been calculated relative to 28-day time-weighted geometric mean measured concentrations. These values are included within the table along with the corresponding nominal values for comparative purposes only. The test sediment contained 5.3% total organic carbon.	Wildlife International, Ltd. (2006a).
Hyallela azteca (amphipod)	ASTM E 1706-00 OPPTS 850.1735	2006	28-day EC50 survival 28-day NOEC survival/repro 28-day LOEC	>71 mg/kg dwt (M) 71 mg/kg dwt (M)	(1) valid without restriction	Fulfils all the reliability criteria. Results are expressed relative to mean measured concentrations. The study was subject to GLP. Test sediment contained 5.7% total organic carbon.	Wildlife International, Ltd. (2006b)

Test species	Test protocol	Year test completed	Endpoint and exposure period	Result (mg/l)[1]	Reliability assessment	Comments	Study reference
			survival/repro	>71 mg/kg dwt (M)			
Lumbriculus variegatus (oligochaete)	ASTM E 1706-00 OPPTS 850.1735	2006	28-day EC50 survival 28-day NOEC survival/repro 28-day LOEC survival/repro	>60 mg/kg dwt (M) 60 mg/kg dwt (M) >60 mg/kg dwt (M)	(1) valid without restriction	Fulfils all the reliability criteria. Results are expressed relative to mean measured concentrations. The study was subject to GLP. Test sediment contained 5.7% total organic carbon.	Wildlife International, Ltd. (2006c)
Chironomus riparius and *Potamopyrgus antipodarum*					(3) invalid	Very little information available and it is impossible to interpret the results in the context of TDCP.	Stachel *et al*, 2005

Note: [1] 'N' denotes result expressed as nominal concentration, 'M' denotes result expressed as mean measured concentration

Fish

Acute toxicity

Study data

Reports have been provided for five acute fish tests – two carried out with *Oncorhynchus mykiss* (Rainbow trout), two with *Oryzias latipes* (Killifish) and one with *Carassius auratus* (Goldfish).

None of the fish tests were supported by analysis of exposure concentrations. However the results of a test media stability study reported by Sasaki et al (1981) showed an initial aqueous exposure concentration of approximately 1 mg/l to decline by 50% over 96 hours when fish were present in the medium. The cause of the decline in concentration was reported to be adsorption by the fish; biodegradation or hydrolysis were considered to be unlikely causes since the properties of the substance indicate that they would not occur to a significant extent within the time period of the experiment. In the absence of exposure analysis the studies in which the media were renewed at intervals during the test were considered to provide the most constant exposure concentrations and the most reliable data.

Only one of the tests was considered acceptable for the purposes of determining a PNEC. The test with *O. mykiss* gave a 96-h LC_{50} value of 1.1 mg/l. The other four tests did however give comparable 96-h LC_{50} values of 1.4 mg/l for *O. mykiss,* 3.6 and 3.7 (48-h LC_{50}) for *O. latipes* and 5.1 mg/l for *C. auratus.*

Two further fish toxicity studies have also been identified by the US EPA in a hazard review of TDCP. The studies did not evaluate toxicity using a range of concentrations in water and cannot be used to derive definitive LC_{50} values. They have therefore not been subject to further review for this risk assessment. Ahrens *et al*. (1979) tested the toxicity of TDCP released from treated fabric to goldfish (*C. auratus*). Laundered or unlaundered sections of fabric were placed in tanks with six goldfish. Fish in the tank with the unlaundered section became sluggish and all died within 3 hours. The concentration of the substance in the test water reached 30 mg/l. Fish exposed for 96 hours to the laundered section did not exhibit signs of toxicity. In another study by Eldefrawi et al., (1977), TDCP in water at 1 mg/l was not toxic to goldfish after 168 hours, but 5 mg/l of TDCP killed all (6/6) goldfish within 24 hours. This result suggests that the 24-h LC_{50} would lie between 1 and 5 mg/l.

QSAR estimated acute toxicity

Estimated values of 8.1 and 4.5 mg/l have been derived for acute (96-hour LC_{50}) fish toxicity using ECOSAR QSARs applicable to esters and phosphate esters respectively. The values are consistent with those obtained in the reported studies.

Long-term toxicity

Study data

No data are available for review

QSAR estimated chronic toxicity

An estimated value of 1.0 mg/l has been derived for chronic fish toxicity using an ECOSAR QSAR applicable to esters.

Aquatic invertebrates

Acute toxicity

Study data

Reports have been provided for two acute invertebrate tests with *Daphnia magna*. One test (Drottar, K.R., Kendall, T.Z. and Krueger, H.O. (1999)) fulfilled all the criteria for acceptability for determining a PNEC and gave a 48-h EC_{50} value of 3.8 mg/l. The other study did not include analysis of exposure concentrations but gave a comparable 48-h EC_{50} value of 4.6 mg/l.

QSAR estimated acute toxicity

An estimated value of 9.9 mg/l has been derived for acute (48-hour LC_{50}) toxicity to invertebrates using an ECOSAR QSAR applicable to esters. The value is consistent with those obtained in the reported studies.

Long-term toxicity

A report has been provided for a chronic reproduction test with the freshwater invertebrate *Daphnia magna* (Thomas, P., van der Togt, B. and B. Kluskens. (2004)). The test fulfilled all the criteria for acceptability for determining a PNEC and gave a 21-day LOEC for reproduction of 1.0 mg/l and a NOEC of 0.5 mg/l.

QSAR estimated chronic toxicity

An estimated value of 1.1 mg/l has been derived for long-term reproductive effects in invertebrates using an ECOSAR QSAR applicable to neutral organics, though this value may not be of high reliability (method not recommended by ECOSAR for this type of compound, and the QSAR is not well validated).

Algae

Acute toxicity

Study data

Reports have been provided for three algal growth inhibition tests – two with *Pseudokirchneriella subcapitata* (also referred to as *Raphidocelis subcapitata* and *Selenastrum capricornutum*) and one with *Scenedesmus subspicatus*. One of the tests with *P. subcapitata* fulfilled all the reliability criteria. The test gave 72 or 96-h EC_{50} values of $\geq$2.8 mg/l (for both biomass and growth rate) a 72-h ErC_{10} of 2.3 mg/l and a NOEC of ≥1.2 mg/l. The results are below the reported water solubility value for TDCP of 18 mg/l.

Neither of the other tests fulfilled all the reliability criteria for obtaining data suitable for deriving a PNEC, and one was considered invalid due to significant inadequacies.

QSAR estimated toxicity

Estimated 96-hour EC_{50} and NOEC values of 1.8 and 1.4 mg/l have been derived for algae using an ECOSAR QSAR applicable to esters. The estimated values are lower than those obtained in the reported studies.

Micro-organisms

A study was performed in compliance with OECD 209 (Jenkins, 1990b), using sludge obtained from Oakley sewage works, which treats primarily domestic waste. Concentrations of 1 mg/l, 10 mg/l, 100 mg/l, 1000 mg/l, 10 g/l were used, and no inhibition of the respiration of activated sludge was observed.

Amphibians

No amphibian effects data were available for review.

Sediment-dwelling organisms

Results of chronic toxicity studies carried out with three species of sediment-dwelling organisms are available:

A prolonged toxicity study with *Chironomus riparius* using spiked sediment (Wildlife International Project 583A-104), performed in April/May 2005.

A prolonged toxicity study with *Lumbriculus variegatus* using spiked sediment (Wildlife International Project 583A-106), performed in June/July 2005

A prolonged toxicity study with *Hyalella azteca* using spiked sediment (Wildlife International Project 583A-103), performed in November/December 2005

The studies were carried out using procedures that are considered to be current best practice and have been assigned reliability 1.

In all three tests, sufficient food for the duration of the test was delivered to the sediment at the start of the studies. The results of the studies are all considered to be valid without restrictions and are reported in **Table 3.34**. However there were differences in the methods and results that required consideration when deciding on the most appropriate NOEC to use as the basis for deriving a PNEC for sediment.

Test methods

The methods employed in the studies were different in a number of ways:

The chironomid study was run as a static test with partial renewal of the overlying water three times per week. The overlying water was aerated constantly. In contrast, the studies with *L. variegatus* and *H. azteca* were run under a flow-through regime with two partial renewals of overlying water per day but with no aeration.

The exposure chambers used in the *C. riparius* study had a volume of 2 L, with a 2 cm layer of sediment. In the other two studies the chambers had a 300 ml volume and a 3.6 cm layer of sediment. These differences resulted in a ratio of surface area of glass to sediment that was more than three times greater in the *Chironomus* study than in the *Lumbriculus* and *Hyalella* studies. It is possible that the larger ratio of sample volume to test compartment volume in the *Chironomus* test led to a higher variability between the analytical measurements, especially since one sample was taken from a compartment at each time point.
The three studies had a different type of food supplied to the organisms. Fish flake food was used in the *C. riparius* study, salmon starter was used in the *L. variegatus* study and a mixture of yeast, ceriophyllum, and trout chow (YCT) was used in the *H. azteca* study. Sufficient food for the test duration was delivered at the start of the studies.

Analytical sampling and results

There were differences in the sampling and analysis methods that were used to determine exposure concentrations of TDCP in the three studies:

The analytical sampling schedule employed in the *C. riparius* study was that proposed in paragraph 38 of OECD guideline 218. Samples of overlying water, pore water and sediment were taken from the highest and lowest TDCP test concentrations at the start, on day 7 and at the end of the study. The analytical results showed an apparent dramatic decline in measured sediment concentrations from 11 and 260 mg TDCP/kg respectively at the start of the study in the lowest and highest treatments to concentrations that were below the Limit of Quantitation (LoQ, 3 mg/kg) at the end of the study. Significant concentration losses were also observed in the pore water and overlying water samples.
The sampling schedule employed in the *Lumbriculus variegatus* study was modified in the light of the *C. riparius* analytical results in order to obtain a better understanding of the stability of TDCP exposure concentrations in all treatments. All test concentrations were sampled at the start, on day 7 and at the end of the study (day 28). The resulting measured sediment concentrations showed only a limited decline over the duration of the study.
The sampling schedule was further modified for the study with *Hyalella azteca*. Samples from all test concentrations were taken at the start, on days 7, 14 and 21 and again at the end of the study (day 28). In addition, in an attempt to explain the decline in exposure concentration observed in the *C. riparius* study, a more rigorous sample extraction procedure was employed. Thus each sample was extracted three times. The first extraction was with acetone, then hexane, followed by a 50:50 mixture of acetone and hexane. The analytical results showed that, despite variability between time points and a slight decline over time, measured TDCP concentrations in sediment remained similar over a 28-day exposure period. This suggested that TDCP was not binding irreversibly to sediment, and that what was extracted on Day 0 could therefore also be extracted on Day 28. The results also showed that the additional extractions provided negligible changes in TDCP concentrations in sediment and that acetone extraction alone (as employed in the *C. riparius* and *L. variegatus* studies) would recover the TDCP in the sample.

The results of analysing exposure concentrations in the three tests suggested that there were significant declines in concentration in the *C. riparius* test that were not apparent to the same degree in the *L. variegatus* and *H. azteca* tests. The decline could be attributed in part to renewal of the overlying water, but a mass balance calculation showed this potential route of loss to be insufficient to account for all of the concentration decline. The loss could not be accounted for by the rigour of the sediment extraction procedure because the use of a more rigorous extraction regime in the *Hyalella azteca* test resulted in only a small increase in recovery. Differences in degradation or adsorption/desorption behaviour in the three test systems can also be discounted as a loss route because results of other studies have shown:

> No mineralisation and no degradation of TDCP over the 122 day period of an aerobic soil degradation test in soil (see section 3.1.3.1.3).
> TDCP added to anaerobic digested sludge was not biodegraded completely in sediment after 60 days (see section 3.1.3.1.2).
> The adsorption/desorption behaviour of TDCP to be consistent with what would be expected on the basis of its K_{ow} value (see section 3.1.3.2.1).

The explanation for the apparent significant differences in the patterns of exposure concentration stability that were observed in the *C. riparius* test compared to the *L. variegatus* and *H. azteca* tests therefore remains unclear. In the absence of any other reasonable explanation, it is considered that the most likely cause of the apparent loss was adsorption to very fine particulates, which would have been removed during renewal of the overlying water. There was a different exposure regime used in this study compared to the *L. variegatus* and *H. azteca* tests. This is a proposal only; however, it should be noted that the study has been conducted to the best available standards and the losses from the sediment solid phase have been accounted for by the use of a geometric mean of the sediment concentrations.

Expression of no observed effect concentrations (NOECs)

In view of the apparent differences in the pattern of measured TDCP sediment concentrations in the studies it is necessary to use a different approaches to expressing the results of the *C. riparius* test compared to the *L. variegates* and *H. azteca* tests.

Chironomus riparius

The rate of *C. riparius* metamorphosis from egg, through the four larval instars and pupa stages, to adult is temperature-dependent. At 20ºC, egg hatching takes approximately 5 days and the larval stages develop through their four instars over approximately 25 days. The pupa stage lasts, on average, about 10 days before emergence of the adult. A 28-day test period is sufficient for *C. riparius* to complete its development when the test is started with eggs (Taylor et al., 1991).

Watts and Pascoe (1998), McCahon and Pascoe (1991) and Ristola (2000) have reported that the first larval instar life-stage, lasting approximately three days post hatch, is the most susceptible to the effects of toxicants. The possibility of 1st instar larvae being even more susceptible to the effects of toxicants than the population as a whole has been highlighted by Ristola (2000). Increased levels of 1st instar larval mortality arising from the effects of toxic substances may be compensated for by lower levels of mortality in subsequent life-stage because of density-dependent factors such as food availability.

In view of the above and the fact that the early days of the test also corresponded with the period when there was likely to have been closest agreement between target and actual

exposure concentrations, the NOEC for the test has been expressed relative to estimated geometric mean concentrations over the first 3 days. This provided a NOEC that, because of the known susceptibility of the 1st instar larvae, was likely to be close to and probably no higher than the NOEC for the whole life-cycle. This practice represented a divergence from the OECD guideline but was considered to provide a NOEC that, given the uncertainties in the exposure concentrations during the test, was based on the most reasonable interpretation of the data relative to the properties of the test substance.

The estimation of geometric mean concentrations over the first 3 days required extrapolating from the patterns of loss apparent in measured concentrations in the lowest (13 mg/kg nominal) and highest (200 mg/kg nominal) treatments between day 0 and day 7 of the test assuming a logarithmic decline in concentration over this period. For the 10.6 and 268 mg/kg day 0 measured concentrations the corresponding estimated day 3 concentrations are 7.3 and 190 mg/kg. From these were calculated geometric mean concentrations for the day 0 to day 3 period of the test of 8.8 and 226 mg/kg. If necessary, for example to calculate an EC_{50}, the concentrations corresponding to the intermediate treatments could be determined by interpolation but in this test it was not necessary to do this because the NOEC corresponded to the lowest treatment.

For comparative purposes only the NOEC was also determined relative to time-weighted geometric mean measured concentrations over the 28-day test period. These were calculated from measured concentrations on days 0, 7 and 28 of the test using methods described in OECD guidance (OECD, 2000).

The resulting NOEC determined from the geometric mean concentrations over the first 3 days is 8.8 mg/kg dw (equivalent to 8.3 mg/kg dw and 1.8 mg/kg wwt in a standard sediment containing 5% organic matter) based on the 3-day geometric mean concentration. For comparative purposes, the value based on the geometric mean measured concentrations over the 28 day test period is 3.9 mg/kg dw (equivalent to 3.68 mg/kg dw and 0.8 mg/kg wwt in a standard sediment containing 5% organic matter).

Lumbriculus variegatus and Hyalella azteca

The results of the L. variegatus and H. azteca studies have been expressed relative to time-weighted geometric mean measured concentrations over the duration of the test. This approach is recommended by the OECD (OECD, 2000). The measured concentrations determined in these two studies were similar in terms of decline over time and variability and were therefore considered appropriate and reliable for such a method of expression.

NOECs for survival and reproduction of 60 and 71 mg/kg dw (equivalent to 53 and 62 mg/kg dw and 11 and 13 mg/kg wwt in a standard sediment containing 5% organic matter) have been determined for L. variegatus and H. azteca respectively.

Other sediment studies

Some ecotoxicology work was performed as part of a river sediment monitoring study (Stachel *et al*, 2005; see also section 3.1.4.2.2). The sampled sediments contained a range of chemical contaminants and were tested on *Chironomus riparius* and *Potamopyrgus antipodarum*. Oestrogenic effects were seen in *Potamopyrgus antipodarum* and this was linked to the detection of some known endocrine disrupters in the test sediments; there is no basis to believe that these findings could have been linked to TDCP. The authors noted that the increased total organic carbon in the sediment (caused by the Elbe flood event, under

study in the paper) could in itself have mitigated effects that might otherwise have been detectable. Furthermore the study itself was not well described. It is concluded that this study is invalid for the purposes of risk assessment of TDCP.

Calculation of Predicted No Effect Concentration (PNEC)

Not all of the test data included in the summary table are considered acceptable for determining PNEC values. Only the acceptable data have been used for the purposes of determining a $PNEC_{aquatic}$.

Test data

The lowest values available for the preferred end points are as follows:

Acute toxicity to fish	96-hr LC_{50}	= 1.1 mg/l
Acute toxicity to invertebrates	48-hr EC_{50}	= 3.8 mg/l
Acute toxicity to algae	72 hrE_rC_{50}	= 4.6 mg/l
Chronic toxicity to invertebrates (21-day repro test)	21-day NOEC	= 0.5 mg/l
Chronic toxicity to algae	72-hr E_rC_{10}	= 2.3 mg/ l (NOEC ≥1.2 mg/l)
Toxicity to WWTP micro-organisms	EC_{50}	= >10000 mg/l

QSAR estimates

Acute toxicity to fish	96-hr LC_{50}	= 4.5 – 8.1 mg/l
Chronic toxicity to fish	NOEC	= 1.0 mg/l
Acute toxicity to invertebrates	48-hr LC_{50}	= 9.9 mg/l
Chronic toxicity to invertebrates	16-d EC_{50}	= 1.1 mg/l
Acute toxicity to algae	96-hr EC_{50}	= 0.69 mg/l
Chronic toxicity to algae	96-hr NOEC	= 0.55 mg/l

$PNEC_{aquatic}$

Fish were marginally more susceptible to TDCP in the acute tests than the invertebrate, *Daphnia magna*, and the two species of algae. Given the similarity in acute susceptibility of the three taxa, further testing to determine a threshold concentration for chronic effects in fish could not be justified on animal welfare grounds.

A NOEC of 0.5 mg/l and an E_rC_{10} value of 2.3 mg/l were determined respectively in the chronic test with *Daphnia magna* and in the growth inhibition test with the alga *Pseudokirchneriella subcapitata*. A $PNEC_{aquatic}$ of 0.01 mg/l has been derived from the invertebrate chronic data by dividing the NOEC for *Daphnia magna* reproduction of 0.5 mg/l by an assessment factor of 50.

The basic guidance from the TGD is not entirely clear as to whether the EC_{10} or NOEC from the algal study should be used as the main result, in the context of PNEC derivation. In this case, due to the shallow dose-response relationship seen in the study with *P. subcapitata*, it is considered appropriate to use E_rC_{10} as the primary result. The *Daphnia* result is more sensitive than either the algal ErC_{10} or NOEC, so this is not a significant issue for TDCP.

$PNEC_{Micro\text{-}organisms}$

Based on the available data, a PNEC for microbial inhibition can only be a limit value. Although it is usual to dose the substance in an activated sludge respiration inhibition test above the water solubility, as was done in the study reported (Jenkins, 1990b), a more realistic limiting concentration is 1000 mg/l. Using this value as the NOEC, then PNEC ≥ 100 mg/l, using an assessment factor of 10.

$PNEC_{sediment}$

In 28-day tests with three species of sediment-dwelling invertebrates – the midge, *Chironomus riparius*, the oligochaete, *Lumbriculus variegatus* and the amphipod, *Hyallela azteca*, it was found that *C. riparius* was most susceptible to the effects of TDCP. A NOEC of 8.8 mg/kg dwt was obtained for this species in sediment containing 5.3% total organic carbon. The NOEC was based on the geometric mean exposure concentrations over the first 3 days of the test and is equivalent to a NOEC of 8.3 mg/kg dwt or 1.8 mg/kg wwt in a standard test system.

Applying an assessment factor of 10 to the NOEC expressed relative to a standard test system gives a $PNEC_{sediment}$ of 0.18 mg/kg wwt. This value is used for the purposes of risk characterisation. It is supported by the $PNEC_{sediment}$ of 0.395 mg/kg wwt obtained using the equilibrium partitioning approach, discussed below.

However, for the purposes of comparison, an alternative PNEC is derived from the NOEC based on day 0-28 geometric mean measured results.

An alternative $PNEC_{sediment}$ of 0.08 mg/kg wwt can be derived from the sediment test data by dividing the NOEC of 0.8 mg/kg wwt (3.68 mg/kg dwt) for effects on *C. riparius* by an assessment factor of 10.

This suggests that, using this alternative analysis of the test results, the risks to fresh water sediment could be up to 2.25 times greater than the values presented in the report. This is commented upon in the Conclusions to the risk assessment.

Comparison with $PNEC_{sediment}$ derived from the $PNEC_{aquatic}$ by equilibrium partitioning

According to the Technical Guidance Document, $PNEC_{sediment}$ can be calculated from the $PNEC_{water}$ by the equilibrium partitioning method. For comparative purposes only the resulting $PNEC_{sediment}$ for TDCP calculated by EUSES is 0.395 mg/kg wwt. This is very similar to the result derived from measured data.

In earlier drafts of this risk assessment, an additional factor of 10 was applied to risk characterisation based on equilibrium partitioning, based on the perceived high adsorption indicated by the HPLC K_{oc} result. This has now been superseded in a new understanding of adsorption behaviour based on a new and reliable study. TDCP has a log K_{ow} value of 3.69 and a K_{oc} value of 1780, therefore there is no need to apply an additional factor of 10.

Terrestrial compartment

Toxicity test results

Tests have been conducted with soil invertebrates (acute and chronic), plants (seedling emergence and growth test) and soil micro-organisms (nitrogen transformation) for TDCP, and are summarised in **Table 3.35**.

Table 3.35 Summary of terrestrial toxicity test results for TDCP

Test species	Test protocol	Year test completed	Endpoint and exposure period	Result (mg/kg dry weight)[1]	Reliability assessment	Comments	Study reference
Toxicity to earthworms							
Earthworms (*Eisenia foetida*)	OECD 207	1996	14-day NOEC 7-day LC_{50} 14-day LC_{50}	100 (N) 230 (N) 130 (N)	(2) valid with restrictions	The test was not supported by GLP. The test is of an overall acceptable standard although there are inadequacies in some elements. Values require correction for organic matter content (10%) prior to use for risk assessment.	Wetton (1996)
Earthworms (*Eisenia foetida*)	OECD draft guideline (January 2000)	2004	28-day LC_{50} (adult mortality) 28-day NOEC (biomass) 28-day LOEC (biomass) 57-day NOEC (repro) 57-day LOEC (repro) 57-day EC_{50} (repro)	>100 (N) 100 (N) >100 (N) 9.6 (N) 13 (N) 67 (N)	(1) valid without restriction	Fulfils all the reliability criteria. Results are expressed relative to mean measured concentrations. The study was subject to GLP. Values require correction for organic matter content (10%) prior to use for risk assessment.	Servajean (2004a)
Toxicity to plants							
Wheat (*Triticum aestivum*) Red clover (*Trifolium pratense*) Mustard (*Sinapis alba*)	OECD Guideline 208	2004	NOEC (emergence) NOEC (plant growth; wet weight) NOEC (plant growth; dry weight) NOEC (emergence) NOEC (plant growth; wet weight) NOEC (plant growth; dry weight) NOEC (emergence) NOEC (plant growth; wet weight) NOEC (plant growth; dry weight)	$\geq$202 (N) 31.5 (N) 25.1 (N) $\geq$202 (N) 28.7 (N) 85.3 19.3 38.7 $\geq$202	(1) valid without restriction	Fulfils all the reliability criteria. Results are expressed relative to mean measured concentrations. The study was subject to GLP. Organic matter content in the test soil was 1.4%.	Servajean (2004b)
Toxicity to soil micro-organisms							

Test species	Test protocol	Year test completed	Endpoint and exposure period	Result (mg/kg dry weight)[1]	Reliability assessment	Comments	Study reference
Nitrifying micro-organisms in sandy loam soil	OECD Guideline 216	2005	NOEC (micro-organism activity based on nitrate concentration); 28 days	≥128 mg/ kg wet weight = 145 mg/ kg dry weight	(1) valid without restriction	Fulfils all the reliability criteria. The study was subject to GLP. Organic matter content in the test soil was 1%.	van Ginkel (2005b)

Note: [1] 'N' denotes result expressed as nominal concentration

Earthworm

Acute toxicity

A report has been provided (Wetton, 1996) for one acute test with the earthworm *Eisenia foetida*. The test fulfilled the criteria for acceptability for determining a PNEC. A 14-day LC_{50} of 130 mg/kg dry weight was determined in the test.

The organic matter content of the soil used in the test was approximately 10% (sphagnum moss peat 10% w/w dry weight of test soil). Therefore the results need to be corrected to obtain a result relevant for natural soils, containing a TGD default of 3.4% organic matter. A correction factor of 0.34 (3.4/10) is therefore applied, giving standardised results of:

14-day $NOEC_{standardised}$ = 34.0 mg/kg dry weight
14-day $LC_{50standardised}$ = 44.2 mg/kg dry weight.

Long-term toxicity

A report has been provided (Servajean, 2004a) for a chronic test with the earthworm *Eisenia foetida*. The test fulfilled the criteria for acceptability for determining a PNEC. A 57-day NOEC of 9.6 mg/kg dry weight was determined for effects on reproduction. A 28-day LC_{50} of >100 mg/kg dry weight was also determined in the test.

The organic matter content of the soil used in the test was approximately 10% (sphagnum moss peat 10% w/w dry weight of test soil). Therefore the results need to be corrected to obtain a result relevant for natural soils, containing a TGD default of 3.4% organic matter. A correction factor of 0.34 (3.4/10) is therefore applied, giving standardised results of:

57-day $NOEC_{standardised}$ = 3.3 mg/kg dry weight
24-day $LC_{50standardised}$ = >34 mg/kg dry weight.

Terrestrial plants

Acute toxicity

No data are available for review.

Long-term toxicity

A report has been provided (Servajean, 2004b) describing tests to determine the side effects of TDCP on the growth of three plant species – Wheat (*Triticum aestivum*), Mustard (*Sinapis alba*) and Red clover (*Trifolium pratense*). The end points are emergence and plant growth, expressed on a wet weight and dry weight basis. It is assumed that this means the weight of the whole plant including the root, though this is not stated explicitly in the report. The plants were harvested 19 days after 50% of the controls had emerged.

The tests fulfilled the criteria for acceptability for determining a PNEC. The lowest NOEC (19.3 mg/kg dry weight) was determined for seedling emergence in Mustard.

In this case, correction for organic matter content in the test (1.4%) would give a more favourable result and therefore this correction has not been made.

NOEC = 19.3 mg/kg dry weight

Terrestrial micro-organisms

Inhibition by TDCP of nitrogen transformation by soil micro-organisms was examined in a study conducted voluntarily by industry (van Ginkel, 2005b). A 28-day NOEC of ≥128 mg/kg wet weight (no inhibition at the highest concentration tested) was determined in the test.

In this case, correction for organic matter content in the test (1%) would give a more favourable result and therefore this correction has not been made.

The study gave an unexpected result with increased micro-organism activity at the highest concentrations of TDCP. The study authors suggested that this observation could be attributed to possible beneficial effects of TDCP on micro-organisms mineralising nitrogenous organic compounds and/or the nitrifying micro-organisms, possibly caused by the test substance providing a source of phosphorus. This observation was not interpreted as an effect in the context of the study.

Calculation of Predicted No Effect Concentration (PNEC)

The lowest toxicity values (corrected, where appropriate, for standardised organic matter content of 3.4% and a conversion factor of 1.13 for transforming results from a dry weight to wet weight basis) are as follows:

Acute toxicity to earthworms (*Eisenia foetida*)	14 d LC_{50}	= 26 mg/kg dw	= 23 mg/kg ww
Chronic toxicity to earthworms (*Eisenia foetida*)	57 d $NOEC_{repro}$	= 3.3 mg/kg dw	= 2.9 mg/kg ww
Toxicity to plants (Mustard, *Sinapis alba*)	$NOEC_{emergence}$	= 19 mg/kg dw	= 17 mg/kg ww
Toxicity to soil micro-organisms (nitrifying micro-organisms in sandy loam soil)	28 d NOEC		**≥** 128 mg/kg ww

The availability of a data set that includes acceptable results from three long-term tests with species from at least three trophic levels, means that it is possible to derive a $PNEC_{soil}$ from the test data by applying an assessment factor of 10 to the lowest chronic NOEC. The resultant $PNEC_{soil}$ is 3.3/10 = 0.33 mg/kg soil dry weight, equivalent to 0.29 mg/kg soil wet weight.

The PNEC derived by the equilibrium partitioning method from the PNEC for aquatic organisms is 0.32 mg/kg soil wet weight, which is very similar to that derived from measured data.

Atmosphere

No data are available on the toxicity of TDCP to plants or other organisms exposed via air. Based on its structure, TDCP is not expected to have ozone depleting effects and the low level of exposure makes other effects unlikely. The evidence from the open literature indicates that TDCP, found in needles of pine trees (*Pinus ponderosa*), and thought to have been transported by aerial deposition processes, did not exert phytotoxic effects (Aston *et al*, 1996). The possibility of TDCP contributing to atmospheric effects such as global warming, ozone depletion and acid rain is likely to be very small.

Secondary poisoning

Effect data

The most relevant data for derivation of the PNEC for secondary poisoning for TDCP are from a two-year carcinogenicity study in the rat. The lowest dose tested resulted in effects and hence no dose-based NOAEL is available. The LOAEL is 5 mg/kg bw/day and is based on the hyperplasia of the convoluted tubule epithelium observed in all male animals at 24 months and the effects noted in the testes at this dose level. Hyperplasia is considered to be a pre-neoplastic lesion. For full details please refer to Section 4.1.2.6.

Using the conversion factors given in the Technical Guidance Document:
LOAEL = 5 mg/kg bw/d
NOAEL < 5 mg/kg bw/d

NOEC mammal = NOAEL mammal x CONV mammal

NOEC = < 5 mg/kg bw/d x 20 (study > 6 weeks)
= < 100 mg/kg food

Toxicokinetics data show that there is 100% absorption by the oral route.

Calculation of $PNEC_{oral}$

According to the Technical Guidance Document an assessment factor of 30 is appropriate for the results of a study of this duration. Therefore, applying this assessment factor:

PNEC oral = NOAEL/AF

PNEC oral = < 100/30
= < 3.3 mg/kg food

A PNEC for secondary poisoning of < 3.3 mg/kg food will be used. This value is also applicable for the assessment of secondary poisoning in the marine environment.

MARINE EFFECTS ASSESSMENT

Calculation of Predicted No Effect Concentration (PNEC)

PNEC*seawater*

No measured data are currently available for marine organisms therefore the PNEC is derived from data obtained for freshwater species (NOEC = 0.5 mg/l), applying an assessment factor of 500 to give PNECseawater = 0.001 mg/l.

PNEC*marine sediment*

No measured data are currently available for marine sediment organisms therefore the PNEC is derived from data obtained for freshwater species (NOEC = 1.8 mg/kg wwt), applying an assessment factor of 50 to give PNECmarine sediment = 0.036 mg/kg.

RISK CHARACTERISATION

The industry has confirmed that the confidential application C of TDCP (life cycle stages C1a, C1b and C2) is no longer applicable in Europe and supply has ceased. While risk characterisation has been performed for these life cycle stages, it should be recognised that the risks are not believed to be relevant. They are retained only in view of the possibility that supply could resume in future.

PEC values for fresh and marine water, sediment and soil, and for predators are given in **Tables 3.11** to **3.12, 3.22** to **3.24** and **3.30** to **3.33**. PEC/PNEC ratios are given in **Tables 3.37** to **3.42**.

For ease of reference, the PNECs used in the risk assessment are summarised in **Table 3.36**.

Table 3.36 PNECs used in the risk assessment of TDCP

Compartment	Value of PNEC
Freshwater	0.01 mg/l
Freshwater sediment	Based on measured data: 0.18 mg/kg wet weight) *0.08 mg/kg wwt (alternative value for comparison)* 0.395 mg/kg wet weight (equilibrium partitioning)
WWTP micro-organisms	≥ 10 mg/l
Seawater	0.001 mg/l (extrapolation from freshwater)
Marine sediment	Based on measured data for freshwater species: 0.036 mg/kg wet weight) 0.0395 mg/kg wet weight (equilibrium partitioning)
Soil	0.29 mg/kg wet weight
Secondary poisoning	<3.3 mg/kg food

Aquatic compartment (incl. sediment)

Water and sediment

Table 3.37 PEC/PNEC ratios for surface water and freshwater sediments

Scenario	$PEC/PNEC_{water}$	$PEC/PNEC_{sediment}$
Producer 1	0.032	0.0701
Producer 2	3.41E-03	7.46E-03
A1a: Flexible foam - automotive - foaming large site	2.97E-03	6.49E-03
A1b: Flexible foam - automotive - foaming	2.32E-03	5.08E-03
A2: Foam cutting	2.56E-03	5.60E-03
B1: Flexible foam - furniture - foaming	2.96E-03	6.49E-03
B2: Foam cutting	2.37E-03	5.18E-03
C1a: CONFIDENTIAL	*0.0286*	*0.0626*
C1b: CONFIDENTIAL	*0.684*	*1.5*
C2: CONFIDENTIAL	*4.08*	*8.92*
D1: CONFIDENTIAL	2.23E-03	4.88E-03
D2: CONFIDENTIAL	2.23E-03	4.88E-03
E1a: CONFIDENTIAL	0.0104	0.0228
E1b: CONFIDENTIAL	0.0841	0.184
F1: CONFIDENTIAL	0.0635	0.139
G1: CONFIDENTIAL	0.0108	0.0237
I1: Flexible foam - Furniture, seating, mattresses - re-bonding of scrap	2.23E-03	4.88E-03
J1: Loose Crumb	2.23E-03	4.88E-03

$PEC/PNEC_{regional(water)}$ = 2.23E-03 from the EUSES v2.03 model.

$PEC/PNEC_{regional(freshwater\ sediment)}$ = 7.63E-03 from the EUSES v2.03 model.

Conclusions to the risk assessment for the aquatic compartment (water and sediment):

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

This conclusion applies to all life cycle stages

A potential risk is identified for the surface water and sediment compartments for confidential life cycle stage C2 and to sediment only for use C1b. There is limited scope for refinement of the exposure scenarios (for C1b, releases have been read across from a specific site, but otherwise it is a generic scenario; scenario C2 is also generic, although information from risk assessments of other flame retardants has been used). There is some potential to refine the

$PNEC_{water}$ since currently an assessment factor of 50 is applied (a chronic fish toxicity would be needed). The PNEC for sediment is based on three long-term tests. Due to the concentration losses observed, there is uncertainty over the interpretation of the *Chironomus* study which is the basis for the $PNEC_{sediment}$. A worst case interpretation of the study would be to use the 28-day geometric mean concentration to derive the NOEC. In this case the PNEC could become 2.25 times lower, and hence any RCR above 0.44 is a potential risk. No additional life cycle stages would be affected.

However, neither of these two life cycle stages is understood to be relevant in Europe any more, on the basis of industry information.

Wastewater treatment processes

Table 3.38 PEC/PNEC ratios for wastewater treatment plants

Scenario	$PEC/PNEC_{WWIP}$
Producer 1	<0.00299
Producer 2	<1.19E-04
A1a: Flexible foam - automotive - foaming large site	<7.38E-06
A1b: Flexible foam - automotive – foaming	<9.24E-07
A2: Foam cutting	<3.28E-06
B1: Flexible foam - furniture – foaming	<7.36E-06
B2: Foam cutting	<1.37E-06
C1a: CONFIDENTIAL	*<2.65E-04*
C1b: CONFIDENTIAL	*<6.84E-03*
C2: CONFIDENTIAL	*<0.0409*
D1: CONFIDENTIAL	Not applicable
D2: CONFIDENTIAL	Not applicable
E1a: CONFIDENTIAL	<0.0328
E1b: CONFIDENTIAL	<8.21E-04
F1: CONFIDENTIAL	<6.14E-04
G1: CONFIDENTIAL	<8.62E-05
I1: Flexible foam - Furniture, seating, mattresses - re-bonding of scrap	Not applicable
J1: Loose Crumb	Not applicable

Conclusions to the risk assessment for wastewater treatment plant micro-organisms:

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

This conclusion applies to all life cycle stages.

Terrestrial compartment

Table 3.39 PEC/PNEC ratios for agricultural soil

Scenario	PEC/PNEC for soil
Producer 1	3.54E-03
Producer 2	0.0346
A1a: Flexible foam - automotive - foaming large site	5.45E-03
A1b: Flexible foam - automotive – foaming	3.71E-03
A2: Foam cutting	4.35E-03
B1: Flexible foam - furniture – foaming	5.42E-03
B2: Foam cutting	3.83E-03
C1a: CONFIDENTIAL	*0.0707*
C1b: CONFIDENTIAL	*1.86*
C2: CONFIDENTIAL	*10.7*
D1: CONFIDENTIAL	3.46E-03
D2: CONFIDENTIAL	7.05E-03
E1a: CONFIDENTIAL	0.149
E1b: CONFIDENTIAL	0.219
F1: CONFIDENTIAL	0.165
G1: CONFIDENTIAL	0.0262
I1: Flexible foam - Furniture, seating, mattresses - re-bonding of scrap	3.50E-03
J1: Loose Crumb	3.48E-03

$PEC/PNEC_{regional(soil)}$ = 4.24E-03 from the EUSES v2.03 model.

Conclusions to the risk assessment for the terrestrial compartment:

> There is at present no need for further information and/or testing and no need for risk reduction measures beyond those which are being applied already

This conclusion applies to all life cycle stages.

A potential risk is identified for confidential life cycle stage C1b and C2. As explained for the aquatic compartment, there is limited scope for refinement of the exposure scenarios. There is no potential to further refine the PNEC for the terrestrial compartment since an assessment factor of 10 is applied.

However, neither of these two life cycle stages is understood to be relevant in Europe any more, on the basis of industry information.

Atmosphere

Neither biotic nor abiotic effects on the atmosphere are likely because of the low predicted environmental concentrations of TDCP (all concentrations are below 0.1 μg/m^3).

Conclusions to the risk assessment for atmosphere:

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

This conclusion applies to all life cycle stages.

Secondary poisoning

PEC/PNEC ratios for secondary poisoning are presented in **Table 3.40**.

The available effects data mean that PNEC is based on a limit value. This means that all PEC/PNEC ratios are presented as 'greater-than' values, which could be interpreted as potential concerns. However, almost all of the PEC/PNEC ratios are at least one order of magnitude below 1, and only two life cycle stages (C1b and C2) have significantly higher ratios (>0.145 and >0.83 respectively, for the earthworm food chain). Whilst these could be interpreted as indicating a risk, recent information from industry indicates that these uses are no longer supported by the suppliers. The ratios also depend on assumptions about earthworm accumulation, which could be unrealistic based on BCF data for fish (which is lower than would be predicted from the log K_{ow}).

Table 3.40 PEC/PNEC ratios for secondary poisoning

Scenario	$PEC/PNEC_{fish\ eating}$	$PEC/PNEC_{worm\ eating}$
Producer 1	>7.97E-04	>6.05E-04
Producer 2	>3.2E-04	>3.01E-03
A1a: Flexible foam - automotive - foaming large site	>3.42E-04	>7.53E-04
A1b: Flexible foam - automotive - foaming	>3.06E-04	>6.18E-04
A2: Foam cutting	>3.19E-04	>6.67E-04
B1: Flexible foam - furniture - foaming	>3.24E-04	>7.51E-04
B2: Foam cutting	>3.09E-04	>6.28E-04
C1a: CONFIDENTIAL	*>7.4E-04*	*>5.92E-03*
C1b: CONFIDENTIAL	*>0.0117*	*>0.145*
C2: CONFIDENTIAL	*>0.0802*	*>0.832*
D1: CONFIDENTIAL	>3.01E-04	>5.99E-04
D2: CONFIDENTIAL	>3.01E-04	>8.83E-04
E1a: CONFIDENTIAL	>3.24E-04	>0.0119
E1b: CONFIDENTIAL	>6.79E-04	>0.0173
F1: CONFIDENTIAL	>2.19E-03	>0.0131
G1: CONFIDENTIAL	>4.6E-04	>2.36E-03
I1: Flexible foam - Furniture, seating, mattresses - re-bonding of scrap	>3.01E-04	>6.02E-04
J1: Loose Crumb	>3.01E-04	>6E-04

Conclusions to the risk assessment for secondary poisoning:

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

This conclusion applies to all life cycle stages. Although a potential risk is possible for the earthworm food chain for confidential life cycle stages C1b and C2, these are no longer considered relevant in Europe. If supply were to recommence in future, then potential secondary poisoning risks would need to be considered alongside risks for other end points (e.g. surface water).

Marine environment

PBT assessment

Persistence

The persistence criteria currently laid down in the TGD require a half-life >60 days in marine water (or >40 days in fresh water) or >180 days in marine sediment (or >120 days in freshwater sediment). The available screening studies show that TDCP is not readily biodegradable so the screening criterion for persistence is met.

Bioaccumulation

The criterion used in the TGD for bioaccumulation is a bioconcentration factor (BCF) >2,000 l/kg. TDCP has a measured fish BCF of 31-59 in the only acceptable result of three studies and hence does not meet the B criterion.

Toxicity

The toxicity criterion used in the TGD is a chronic NOEC <0.01 mg/l or substances classified as Carcinogenic (category 1 & 2), Mutagenic (category 1 & 2), or Toxic to Reproduction (category 1,2, & 3) or with other evidence of chronic toxicity. The lowest aquatic NOEC for TDCP is 0.5 mg/l from a 21-day *Daphnia* study. Regarding human health effects, TDCP is classified as Carcinogenic Category 3 R40 (Limited evidence of a carcinogenic effect). This classification is based on the results of a 2-year carcinogenicity study. Based on the current evidence, combined with the aquatic toxicity results, there is no definite concern for chronic toxicity and hence the T criterion is not met.

Summary of PBT assessment

For the PBT assessment, TDCP can be considered to be potentially persistent (P) or potentially very persistent (vP) based on its ultimate mineralisation. The available information on bioaccumulation shows that TDCP does not meet the B or vB criterion. The T criterion is not met.

Marine risk characterisation

Table 3.41 PEC/PNEC ratios for sea water and marine sediments

Scenario	$PEC/PNEC_{sea\ water}$	$PEC/PNEC_{marine\ sediment}$
Producer 1	0.3	0.328
Producer 2	0.0166	0.0182
A1a: Flexible foam - automotive - foaming large site	3.13E-03	3.42E-03
A1b: Flexible foam - automotive - foaming	2.34E-03	2.56E-03
A2: Foam cutting	2.63E-03	2.88E-03
B1: Flexible foam - furniture - foaming	3.13E-03	3.42E-03
B2: Foam cutting	2.40E-03	2.62E-03
C1a: CONFIDENTIAL	*0.0344*	*0.0376*
C1b: CONFIDENTIAL	*0.833*	*0.911*
C2: CONFIDENTIAL	*4.97*	*5.44*
D1: CONFIDENTIAL	2.23E-03	2.44E-03
D2: CONFIDENTIAL	2.23E-03	2.44E-03
E1a: CONFIDENTIAL	0.102	0.112
E1b: CONFIDENTIAL	0.102	0.112
F1: CONFIDENTIAL	0.0769	0.0841
G1: CONFIDENTIAL	0.0127	0.0139
I1: Flexible foam - Furniture, seating, mattresses - re-bonding of scrap	2.23E-03	2.44E-03
J1: Loose Crumb	2.23E-03	2.44E-03

Note: The ratio for life cycle stage D1 is based on emissions data for a single site, although a marine scenario is not strictly relevant since it is situated inland.

$PEC/PNEC_{regional(sea\ water)}$ = 2.23E-03 from the EUSES v2.03 model

$PEC/PNEC_{regional\ (marine\ sediment)}$ = 3.19E-03 from the EUSES v2.03 model.

Conclusions to the risk assessment for the marine environment:

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

This conclusion applies to all life cycle stages.

A potential risk is identified for the marine water and sediment compartments for confidential life cycle stage C2. There is limited scope for refinement of the exposure scenario (it is generic, although information from risk assessments of other flame retardants has been used). There is some potential to refine the PNECs (further marine organism toxicity data would be needed). However, this life cycle stage is understood to be no longer relevant in Europe, on the basis of industry information.

Secondary poisoning in the marine environment

Table 3.42 PEC/PNEC ratios for secondary poisoning in the marine environment

Scenario	$PEC/PNEC_{marine\ predator}$	$PEC/PNEC_{marine\ top\ predator}$
Producer 1	>5.26E-04	>1.29E-04
Producer 2	>5.36E-05	>3.48E-05
A1a: Flexible foam - automotive - foaming large site	>3.51E-05	>3.11E-05
A1b: Flexible foam - automotive - foaming	>3.08E-05	>3.03E-05
A2: Foam cutting	>3.23E-05	>3.06E-05
B1: Flexible foam - furniture - foaming	>3.29E-05	>3.07E-05
B2: Foam cutting	>3.11E-05	>3.03E-05
C1a: CONFIDENTIAL	*>8.37E-05*	*>4.08E-05*
C1b: CONFIDENTIAL	*>1.41E-03*	*>3.07E-04*
C2: CONFIDENTIAL	*>9.77E-03*	*>1.98E-03*
D1: CONFIDENTIAL	>3.01E-05	>3.01E-05
D2: CONFIDENTIAL	>3.01E-05	>3.01E-05
E1a: CONFIDENTIAL	>5.78E-05	>3.57E-05
E1b: CONFIDENTIAL	>7.62E-05	>3.94E-05
F1: CONFIDENTIAL	>2.61E-04	>7.62E-05
G1: CONFIDENTIAL	>4.95E-05	>3.4E-05
I1: Flexible foam - Furniture, seating, mattresses - re-bonding of scrap	>3.01E-05	>3.01E-05
J1: Loose Crumb	>3.01E-05	>3.01E-05

The available effects data mean that PNEC is based on a limit value. This means that all PEC/PNEC ratios are presented as 'greater-than' values, which could be interpreted as potential concerns. However, the ratios are all several orders of magnitude below 1, and due to the lack of any significant bioaccumulation potential of TDCP, it is reasonable to conclude that there are no risks.

Conclusions to the risk assessment for secondary poisoning in the marine environment:

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

This conclusion applies to all life cycle stages.

Areas of uncertainty in the environmental risk assessment

The main area of uncertainty is the assumption regarding limited availability of TDCP for release from foams. This is discussed in section 3.1 and will affect all life cycle stages

associated with foam production, processing and use (local life cycle stages A1a, A1b, A2, B1, B2, I1 and J1, and the regional background). The sensitivity of the risk assessment to this uncertainty has been considered, as follows. While the exact level of availability is uncertain, it would be very unlikely to be as high as 40%, which is the level that applies for the related substance TCPP (which is well supported by experimental evidence). Taking this as the worst case, PEC/PNEC ratios could potentially be (in most cases) four times higher for TDCP foam-related life cycle stages. It is clear that even in this worst case, no additional risks would be identified for these local life cycle stages.

It is understood that the life cycle stages associated with Confidential Use C (i.e. C1a, C1b and C2) are no longer relevant in Europe, on the basis of industry information. Should it be the case that supply for Use C resumes in future, conclusion (i) or (iii) would apply for some compartments and some life cycle stages.

The Rapporteur has no reason to anticipate significant tonnage increases in the near future, based on industry information and general research.

HUMAN HEALTH

HUMAN HEALTH (TOXICITY)

Exposure assessment

Occupational exposure

In the following sections, unless otherwise stated, the term exposure is used to denote personal exposure as measured or otherwise assessed without taking into account the attenuating effects of any personal protective equipment (PPE) which might have been worn as not enough information was available to take the actual protection of any PPE worn into account.

Occupational exposure information has been made available through the manufacturers and users of TDCP.

Overview of exposure

TDCP is a liquid at room temperature with a low vapour pressure of 5.6×10^{-6} Pa at 25^0C and a calculated saturated vapour pressure (SVC) of 1 $\mu g/m^3$ at 21^0C.

Occupational exposure to TDCP may occur during its manufacture and during the manufacture and cutting of polyurethane (PUR) foam. Inhalation of vapours and skin contact are the predominant routes of exposure. Oral exposure is not considered to be a significant route of exposure under normal working practices. The total number of people occupationally exposed to TDCP is not known.

Descriptions of the processes and sources of occupational exposure are discussed below along with a discussion of exposure levels. Most of the data used in this assessment has been supplied by industry, either directly or through trade organisations. Data supplied by industry for the risk assessment report for V6 has also been used where appropriate. The data has been used in more than one scenario where it was felt appropriate by the Rapporteur. The occupational exposure scenarios are:

Manufacture of TDCP
Manufacture of flexible PUR foam
slabstock foams
moulded foams
*Cutting of flexible PUR foam
Production of foam granules and rebonded PUR foam
Manufacture of automotive parts

* Scenario 3 covers the cutting of foam by furniture manufacturers, where it occurs.

Following manufacture, most of TDCP produced in the EU in 2000 was used in the production of flexible foam in Europe. Most of the TDCP used in flexible foam is for the automotive industry with some used in furniture. TDCP is added directly at the point of production of flexible foams.

Occupational exposure limits

There are no occupational exposure limits set for TDCP.

Scenario 1: Occupational exposure during the manufacture of TDCP

TDCP is manufactured by two producers in the EU. In the year 2000, the total EU production was less than 10,000 tonnes, with production taking place in the UK and Germany. Between 1998 and 2003, production has fluctuated slightly but the total EU sales tonnage has remained reasonably stable within approximately 10 %. Neither producer imported TDCP into the EU in the year 2000. Both are of the opinion that TDCP is not imported into the EU by any other party. Both producers exported TDCP from the EU in the year 2000.

In both production facilities, TDCP is produced in a closed system by reacting phosphorous oxychloride with an organic epoxide chemical in the presence of a catalyst. The crude product is washed and dehydrated to remove acidic impurities and residual traces of water and volatile chemicals. The product is then filtered, transferred to storage tanks for despatch in road tankers or packed into drums (pers. Comm. 30th April 2001, Rhodia).

Measured inhalation and dermal exposure data

Production plant 1

In a study conducted by industry (2002), inhalation and hand exposures of 2 operators in one of the TDCP manufacturing plants were evaluated under actual working conditions. At this plant, TDCP is produced in a closed system. Filling stations for drumming are semi-automatic and equipped with local exhaust ventilation to remove vapours from the operator area. The plunger is also equipped to avoid drops falling down when the lance is transferred from one drum to another. Although the operator moves the lance from drum to drum, it is carried out using a boom so that the operator does not come into contact with the lance. The operator does secure lids and fits seals to the drums. The entire reaction, washing, drying and storage tanks are closed and either purged with nitrogen or under vacuum. The processes are computer-controlled. The computers monitor and control reactors, reaction conditions such as temperature and pressure, chemical additions and process alarms. This limits the possibilities of operator contact with TDCP during the production steps. One operator per shift is assigned to the plant. The operators spend most of their time in the control room. Highest inhalation and dermal exposures are likely to occur during drumming and activities such as material sampling and maintenance. Samples are taken from a sampling valve into a 250 g bottle. There is no local exhaust ventilation at the sampling point. The operator wears PVC gloves, safety spectacles, hard hat and work coveralls. Sampling takes less than 1 minute to complete. Analysis is carried out by a laboratory technician. Extraction ventilation and personal protective equipment are employed to reduce exposure.

Operators monitored were involved in production and drumming (one operation of drumming was monitored). In addition, a laboratory operator was monitored. Air-sampling pumps and a sampling tube were used for the assessment of inhalation exposure. The air sampler was attached to the collar of the operator, thus positioning it in his breathing zone. The pumps were operated for the duration of the monitoring period. The pump was calibrated to a nominal sample flow rate of approximately 1 L/min ± 10 % L/min. The sample tube was extracted with toluene containing trioctyl phosphate. The final extract was chromatographed with flame photometric detection.

For dermal exposure monitoring, 100 % cotton absorbent gloves were used as dosimeters. If protective gloves were used, the absorbent gloves were worn beneath them. The protective gloves used were Vygen plus PVC gloves, cotton lined. The absorbent gloves were peeled off and replaced at times when the worker normally washed his hands and were placed in a plastic bag. They were extracted with toluene before chromatography.

The methods for both inhalation and dermal monitoring have been developed and validated for TDCP. The limit of detection was evaluated to be 0.1 μg for TDCP on sampling tubes and 3 μg on cotton gloves. **Table 4.1** below gives a summary of these monitoring results.

Table 4.1 Results of personal inhalation and dermal monitoring carried out on operators involved in production of TDCP, drumming and laboratory work

Operator's Task	Length of time monitored (mins)	Inhalation exposure TDCP (μg/m³)	Dermal exposure TDCP (mg/kg bw)
Production	412	0.7	0.21
Drumming	151	5.6	0.07
Laboratory Operator	400		0.34

During the monitoring period (for both dermal and inhalation), the production operator supervised the production of 3 batches, cleaned the funda filter and took a sample from the funda filter. He was located in the control room for most of the time. During these activities, he wore protective gloves (Vygen plus gloves). The operator carrying out the task of drumming TDCP drummed 25 drums (300 kg each), containing 90 % TDCP, for a period of 2.5 hours. He did not wear PPE when carrying out his tasks. The laboratory operator carried out TDCP crude testing (20 mins), TDCP stock tank testing (20 mins) and TDCP filtered testing (20 mins) during the monitoring period. Dermal exposure of 0.34 mg/kg bw was measured for a laboratory worker. He did not wear any PPE while carrying out his tasks.

In parallel to the personal monitoring, static measurements, with the same equipment as for personal monitoring, were performed. In the TDCP plant, the static monitoring was carried out near a sampling valve; however, no sampling was carried out during the monitoring period. The monitoring period was for 406 mins. This static measurement gave an airborne concentration of TDCP of 0.70 $\mu g/m^3$.

Production plant 2

In a second TDCP production plant, inhalation exposure was measured by industry by means of personal sampling systems (2002). In this plant, TDCP is produced in a batch-wise manner. The system is a closed one, except for loading stations. All of the processes are computer controlled, with a specific operator permanently present in the control room. The filling stations are automatic and equipped with LEV.

The method used for measuring TDCP was the same as that described for plant 1 above. Monitoring was carried out on the chemical production and quality control line (1 operator monitored) and during drumming of the final product into steel drums and IBCs (1 operator monitored) for the duration of a typical working day. Both operators were monitored for the duration of their 8-hour shift. The monitoring indicated that both operators were exposed to an airborne concentration of TDCP of <0.002 mg/m^3.

Modelled dermal exposure data

EASE is a general purpose predictive model for workplace exposure assessments. For workers involved in the manufacture of TDCP, the appropriate EASE scenario would be a closed system (breached for sampling and maintenance) with no direct handling. For this, EASE has predicted the dermal exposure to be very low.

For sampling of TDCP during the manufacturing process, default values are taken from the TGD for the scenario quality control sampling of liquids. It is considered however, that the contact is intermittent, rather than incidental, with non-dispersive use and an exposure area of 210 cm^2. The exposure estimate for this is 0.1 to 1 mg/cm^2/day. The exposure area of 210 cm^2 was selected as there is little opportunity for large-scale dermal exposure during normal operations as most of the production takes place in closed systems with breaches for sampling and drumming.

For drumming of TDCP and TDCP blends, using the default values of reasonable worst-case dermal exposure for the scenario of drumming of liquids given in the TGD (non-dispersive use, with intermittent contact and an exposure area of 210 cm^2), gives an estimate of 0.1 to 1 mg/cm^2/day.

Summary of occupational exposure to TDCP during its manufacture

For the measured data, there are few data points from the monitoring carried out in the 2 production plants. However, the tasks carried out during the monitoring periods are typical of the normal work patterns and the results obtained appear to be representative of the TDCP production industry. **Table 4.2** below summarises the exposure measurements taken in the two production plants.

Table 4.2 Summary table of exposure measurements taken in two production plants

Operator's Task	Length of time monitored (mins)	Inhalation exposure TDCP (μg/m³)	Dermal exposure TDCP (mg/kg bw)
Plant 1			
Production	412	0.7	0.21
Drumming	151	5.6	0.07
Laboratory Operator	400	-	0.34
Plant 2			
Operator 1 - production	480	<2.0	-
Operator 2 - drumming	480	<2.0	-

Values taken forward for risk characterisation

The value taken forward for a worst-case inhalation exposure is 5.6 $\mu g/m^3$, 8-hour TWA. This was the higher of the four data points reported by industry. Although it was for 151 minutes drumming, it is assumed that drumming of TDCP could theoretically be carried out for a full shift if production required it. The value taken forward as a typical exposure value is 2.8 $\mu g/m^3$, 8-hour TWA. This is half the highest exposure obtained during sampling, and is in line with TGD guidance. It is possible that actual exposure is typically less than this, but a lack of sufficient data means that this value has been taken to err on the side of caution.

For dermal exposure the value taken forward for reasonable worst-case dermal exposure is 0.1 mg/cm^2/day or 21 mg/day, which is equivalent to the highest value obtained during sampling (0.34 mg/kg bw, assuming a bodyweight of 70 kg and an exposure area of 210 cm^2). Although this is the highest value obtained, there was another sample (0.21 mg/kg bw) of the same order of magnitude. The value taken forward for typical dermal exposure is 0.05 mg/cm^2/day or 10.5 mg/day. This is equivalent to 0.15 mg/kg bw assuming an exposure area of 210 cm^2 and a bodyweight of 70 kg. It is within the range of the reported data from industry, although lower than the range of 0.1 to 1 mg/cm^2/day predicted using EASE.

Scenario 2a: Occupational exposure during the production of slabstock foam

Introduction

Flexible polyurethane foams can be manufactured in continuous or batch processes. In a typical process, the initial ingredients (mainly water, isocyanate, polyether polyols and any other additive such as a flame retardant) are mixed together at a mixing head and then immediately applied to the bottom lining of a continuously moving trough formed by a horizontal bottom paper or foil and two vertical side papers or foils. After a few seconds, a cream is formed, the volume expands and the foam reaches its maximum height in 1-3 minutes. The blocks of foam are cut off immediately after paper take-off, and then transferred through a transfer conveyer to the weigh scale and to the curing area. Some blocks can be randomly transferred to a specific area for temperature probing.

The amount of TDCP used depends on the foam grade required and is controlled by a meter. Continuous foaming machines can produce polyurethane foam at rates up to 500 kg/minute. The foaming section of the process is enclosed within a tunnel fitted with extraction for removal of di-isocyanate vapours and blowing agent emissions (HMIP, 1995).

The main areas of potential occupational inhalation exposure during slabstock foam manufacture are at the mixing head and when operators have to enter the tunnel to carry out duties such as removing the paper and supervising the block at cut-off areas. The potential for dermal exposure can occur in the mixing head area where raw materials are mixed and contact with chemicals can occur. It can also occur during temperature supervision and cutting of the foam.

Measured Exposure data

An industry consortium measured inhalation and dermal exposure to TDCP at two polyurethane foam production and cutting facilities during February and May 2005. These plants are not identified in this document and are referred to throughout as Plant A and Plant B to distinguish them from the TDCP production plants which have been referred to in this document as production plants 1, 2, 3 and 4.

At Plant A, a total of twenty-eight 8-hour inhalation samples and 9 short-term samples were collected, covering all activities. At Plant B, a total of 12 inhalation samples were collected. The samples were collected by drawing air at 1 litre per minute through XAD-2 OVS tubes, which were clipped to the operators' collar in order to sample from within the breathing zone. The samples were subsequently analysed using analytical method Akzo Nobel CG/6.089.2 (extraction with toluene containing tri-octyl phosphate and subject to gas chromatograph with flame photometric detection).

Twenty-eight dermal shift exposure samples were collected at Plant A as well as one short-term sample. At Plant B twelve dermal shift exposure samples were collected. The samples were collected by the operators wearing cotton gloves throughout their shift which were collected for analysis. The analysis technique used was the same as for analysis of the tubes, except the volume of desorbent used was greater.

The LOD for the method used was 0.1μg for the sampling tubes and 10 μg for the gloves.

The activities covered during the sampling exercise included operators working at the mixing head area, the paper take-off area, the cut-off area, the production area supervisors, the laboratory technician, and the operators in the foam conversion (loop slitting) area. The results for foam cutting are considered in Scenario 3. The result for the rebond operator is considered in Scenario 4.

During the shifts monitored, TDCP-containing foam was manufactured for 1 hour (Plant B) and at Plant A, between approximately 5 hours (day 1) and almost 8 hours (day 2). The amount of TDCP in the runs varied between 3 % and 15 %. These were typical days at the plants were the sampling took place, and the 8-hour TWA results reflect this. That is, the sampling that took place was carried out over a whole shift regardless of how long the operatives were working with TDCP-containing foam, to determine typical shift-length exposures.

The dermal exposure results were presented by industry in a number of ways; total mg TDCP/pair of gloves; mg TDCP/hour; mg TDCP/kg bw and μg TDCP/cm^2/day. Total mg TDCP/pair of gloves has been used as representative of mg TDCP/day, which is the preferred reporting method for risk assessment reports.

Short-term samples were also taken at Plant A during foam production start up and stop activities, and during tanker unloading. The six samples taken during start up and stop activities were all below the limit of detection for 15 minutes (6.7 μg/m^3). Three samples were taken during tanker unloading; one 15 minute sample was taken at the start of the unloading, one 10 minute sample at the end of the unloading and one, 1 hour sample throughout the unloading activity. The first sample was below the limit of detection for 15 minutes (6.7 μg/m^3), one was 10 μg/m^3 (the limit of detection for 10 minutes) and the one hour sample result was 4.8 μg/m^3 (1 hour TWA), which was equivalent to 0.6 μg/m^3 8-hour TWA, assuming no other exposure that day.

Inhalation exposure

Table 4.3 summarises the measured inhalation exposures at Plant A and **Table 4.4** summarises the inhalation exposures at Plant B. **Table 4.7** summarises the short-term inhalation exposures to TDCP in plant A. In addition, inhalation sampling data collected during the manufacture of flexible foam using TCPP and V6 have been presented here. The manufacturing process is the same and the vapour pressures of the three flame retardants are all very low, so use of the data on TCPP and V6 is valid. These data are presented in **Tables 4.5** and **4.6**.

Table 4.3 Personal inhalation exposures to TDCP measured at Plant A

Job title or work area	n	Inhalation TWA 8 h (µg/m³)
Supervisor/ Ass. supervisor	4	0.5, 0.8, 0.9, 2.2
Mixing head area	6	<0.2, 0.2, 0.9, 0.9, 1.5, 1.9
Paper take-off area	4	1.1, 1.1, 2.7, 3.5
Cut-off area	2	<0.2, 1.7
Lab technician	3	<0.2, <0.2, 1.3

Table 4.4 Personal inhalation exposures to TDCP measured at Plant B

Job title or work area	Inhalation TWA 8 h (µg/m³)
Raw material/ Tank Form	<0.20
Mixing head op. I	<0.20
Mixing head op. II	1.25
Mixing head op. III	<0.20
Supervisor	0.23
Side Paper take-off operator	<0.20
Cut-off block operator	<0.20
Cut-off Start/End operator	<0.20
Bottom Paper operator	0.39
Lab technician	<0.20

Table 4.5 Personal sampling data summarising exposure to TCPP during the manufacture of flexible PUR foam

Operator	Operator Activity or Location	PPE Worn	Length of time monitored (mins)	Measured TCPP (µg/m^3)	Calculated 8-hr TWA (µg/m^3)j)
Production op. 1 (plant 1)	Mixing head area	Protective gloves	429	10	8.9
Production op. 2 (plant 1)	Paper take-off area	Respirator with replaceable filter and protective gloves (when entering the tunnel)	404	32	26.9
Production op. 3 (plant 1)	Temperature supervision and probing	None	426	15	13.3
Production op. 4 (plant 1)	Cut-off area	Protective gloves	445	33	30.5
Production op. 5 (plant 2)	Mixing head area	Disposable gloves	239	7.3	3.6
Production op. 6 (plant 2)	Different areas of the line	Respirator with replaceable filter and protective gloves when removing polyethylene film and cleaning tunnel	242	9.7	4.8
Production op. 7 (plant 2)	End of the tunnel	Respirator with replaceable filter and protective gloves when marking block and putting polyethylene film on	236	9.4	4.6
Sampling op. (plant 2) *	Sampling and baler production	Protective gloves	403	17	14.2

Table 4.6 Summary of personal exposure to V6 during the manufacture of flexible PUR foam

Plant identification	Operator	n	Inhalation Exposure 8-hr TWA (µg /m^3)
Plant X	Mixing Head	2	<0.62, <0.62
Plant X	Asst. Mixing Head	4	<0.60, <0.53, <0.61, <0.63
Plant X	Side Paper Take Off	4	<0.62, 5.29, <0.63, <0.53
Plant X	Bottom Paper	4	<0.59, <0.56, <0.59, <0.57
Plant X	Block Cutter	2	<0.64,<0.59
Plant Y	Raw Material/Tank Farm	1	<0.61
Plant Y	Mixing Head	3	0.77, <0.58, <0.58
Plant Y	Supervisor	1	<0.62
Plant Y	Side Paper Take Off	1	<0.63
Plant Y	Cut Off Block	1	<0.59
Plant Y	Cut Off Start/End	1	<0.58
Plant Y	Bottom Paper	1	<0.59
Plant Y	Lab Tech	1	<0.60

Table 4.7 Short-term personal inhalation exposures to TDCP measured at Plant A

Job title or work area	n	Inhalation TWA 15 min (µg/m³)
Foam production start up and stop activities	6	<6.7, <6.7, <6.7, <6.7, <6.7, <6.7
Tanker unloading	2	<6.7, 10
Tanker unloading	1	4.8 (1hr TWA)

The inhalation exposures for TDCP ranged from <0.2 µg/m^3 to 3.5 µg/m^3. The highest result was for an operator at paper take-off area in Plant A. In addition, personal inhalation sampling data from flexible foam manufacturing plants using TCPP and V6 have been used here, as the processes are identical and the flame retardants are used in the same way. The range of exposures taking all of the personal sampling results into account is <0.2 to 30.5 µg/m^3.

Dermal exposure

Tables 4.8 and **4.9** summarise the dermal result from Plants A and B, respectively. Dermal sampling data during the manufacture of flexible foam using TCPP and V6 are presented in **Tables 4.10** and **4.11**, respectively.

Table 4.8 Dermal exposure to TDCP measured at Plant A

Job title or work area	n	mg TDCP /pair of gloves (mg/day)
Supervisor/Ass. supervisor	4	1.0, 1.9, 2.0, 3.7
Mixing head area	6	3.4, 3.9, 11.5, 36.9, 41.6, 49.5
Paper take-off area	4	2.0, 3.0, 8.0, 12.6
Cut-off area	1	27.0
Lab technician	3	0.01, 0.02, 1.1
Truck unloading	1	0.71

Table 4.9 Dermal exposure to TDCP measured at Plant B

Job title or work area	mg TDCP/ pair of gloves (mg/day)
Raw material/ Tank Form	0.22
Mixing head op. I	0.032
Mixing head op. II	0.052
Mixing head op. III	0.17
Supervisor	0.047
Side Paper take-off operator	0.029
Cut-off block operator	0.173
Cut-off Start/End operator	0.124
Bottom Paper operator	0.141
Lab technician	0.048

Table 4.10 Dermal exposure to TCPP during manufacture of flexible PUR foam

Operator	Length of time monitored (mins)	Measured TCPP (mg/kg bw)	Mg/day
Production op. 1 (plant 1)	430	1.5	105
Production op. 2 (plant 1)	443	0.45	31.5
Production op. 3 (plant 1)	429	0.68	47.6
Production op. 4 (plant 1)	445	0.09	6.3
Production op. 5 (plant 2)	239	0.32	22.4
Production op. 6 (plant 2)	242	0.39	27.3
Production op. 7 (plant 2)	236	0.01	0.7
Sampling op. (plant 2)	313	0.003	0.21
Laboratory op. (plant 2)	417	0.003	0.21

Table 4.11 Dermal exposure to V6 during the manufacture of flexible PUR foam

Plant Identification	Operator	n	mg V6 /pair of gloves (mg/day)
Plant X	Mixing Head	2	0.06, 1.39
Plant X	Asst. Mixing Head	4	0.20, 0.31, 0.79, 1.47
Plant X	Side Paper Take Off	4	0.08, 0.12, 0.21, 0.48
Plant X	Bottom Paper	4	0.28, 0.39, 1.18, 7.99,
Plant X	Block Cutter	2	0.14, 0.28
Plant Y	Raw Mat'l/Tank Farm	1	5.2
Plant Y	Mixing Head	3	0.49, 0.54, 0.75
Plant Y	Supervisor	1	0.89
Plant Y	Side Paper Take Off	1	0.39
Plant Y	Cut Off Block	1	0.34
Plant Y	Cut Off Start/End	1	0.23
Plant Y	Bottom Paper	1	0.24

The dermal exposures for TDCP measured in the two plants ranged from 0.01 to 49.5 mg/day. The highest result was obtained by an operator in the mixing head area of Plant A. According to the industry report, the operator did not have much contact with foam, but may have had contact with the chemicals, although it was not clear when or how this had occurred. In addition, personal dermal sampling data from flexible foam manufacturing plants using TCPP and V6 have been used here, as the processes are identical and the flame retardants are used in the same way. The range of exposures taking all of the personal sampling results into account is 0.01 to 105 mg/day or 2.4×10^{-4} to 0.25 mg/cm^2/day assuming an exposure area of 420 cm^2.

Static monitoring study with TDCP

In addition, some measured data is available from work carried out by industry in an EU polyurethane foam manufacturing plant in 2002. Air sampling was carried out using sampling

pumps at 9 different static points on both the polyether and the polyester production lines. TDCP concentration in the air was determined by automated thermal desorber-gas chromatography (ATD-GC). **Table 4.12** below summarises the points monitored around the plant and the results obtained. The personal measurements made at the foam cutting area are reported in Scenario 3.

Table 4.12 Results of static monitoring for exposure to TDCP during PUR foam production

Sampling point	Length of time monitored (mins)	TDCP (μg/m³)
Polyether line, mixing head	5	<5
	20	<5
Polyether line, paper removal	20	<5
Polyether line, cutting point	5	<5
	20	<5
	15	<5
TDCP filling tank	18	<5
	18	<5
Polyester line, mixing head	10	<5
Polyester line, paper removal	5	11
	10	14
	4.5	7
Polyester line, cutting point	18	<5
	18	<5

The limit of detection for TDCP was 5 $\mu g/m^3$. At one monitoring point only, the level of TDCP measured was above the limit of detection. This was at the paper removal point. No further details are available on this.

Values taken forward to risk characterisation

For the purposes of risk characterisation, the personal exposure data has been used, including the data for TCPP and V6. It is considered that these data best represent personal exposure, as the older data was from static monitoring.

For inhalation exposure, the reasonable worst case taken forward to risk characterisation is 5.1 $\mu g/m^3$. This was the 90^{th} percentile of all the measured values obtained in the exposure monitoring carried out. The typical exposure value to be taken forward to risk characterisation is 0.62 $\mu g/m^3$, which is the median value for all the data presented.

For dermal exposure, the RWC taken forward to risk characterisation is 29.8 mg/day or 0.07 mg/cm^2/day, assuming an exposure area of 420 cm^2. This is the 90^{th} percentile of the measured values. For typical exposure, a value of 0.7 mg/day or 0.002 mg/cm^2/day will be taken forward. This is the median number from all the measured exposure values available.

Scenario 2b: Occupational exposure during production of moulded foam

Introduction

Moulded foams can be produced from TDI and also from a mixture of TDI and MDI. Predetermined quantities of mixed reactants are automatically or manually dispensed discontinuously into moulds, which may be stationary or continuously circulating on a track (HMIP, 1995 and BASF, undated). The moulds are normally temperature conditioned prior to filling (HMIP, 1995) to around 40^0C. After the reactants have been dispensed, the lid of the mould is closed and foaming takes place. Alternatively, the mixture is automatically injected into a closed mould with defined vents. With hot cure moulding, the moulds are heated to temperatures typically in the range 150 ^{0}C to 230 ^{0}C (HMIP, 1995). On completion of the curing cycle, the moulds are opened and the moulded shapes are removed for trimming and finishing. Some moulded items are subject to a crushing stage or vacuum treatment in order to break open the closed cells in the moulding. After removal of the moulded article the mould is cleaned by removal of residual foam material from the lid and from vents, etc. The mould is then treated with a mould release agent such as a wax, which may be an organic solvent or an aqueous dispersion (HMIP, 1995).

Measured inhalation exposure data

There are no exposure data for the production of moulded foam products. However, it is thought that the dispensing of the liquid foam into moulds would be similar to the dispensing of the foam mixture from the mixing head during PUR foam block manufacture. Although not directly comparable, it is also felt that the results for work at the cutting of foam blocks would give an indication of the likely range of exposures during cutting and trimming of moulded parts.

Industry carried out inhalation and dermal exposure monitoring in 2005 at two flexible PUR foam manufacturing plants. Relevant results from this exercise have been used to illustrate the likely exposures during the manufacture of moulded products. These data were also used in Scenario 2a. **Tables 4.13** and **4.14** below summarise the inhalation exposure results for TDCP from Plants A and B, respectively. In addition, similar data is presented here for inhalation exposure to V6 and TCPP in **Tables 4.15** and **4.16**,

Table 4.13 Inhalation exposure results for TDCP measured at Plant A

Job title or work area	n	Inhalation TWA 8 h (µg/m^3)
Mixing head area	6	<0.2, 0.2, 0.9, 0.9, 1.5, 1.9
Paper take-off area	4	1.1, 1.1, 2.7, 3.5
Cut-off area	2	<0.2, 1.7
Block preparation	2	3.0, 0.8
Machine operator	7	1.7, 1.9, 3.8, 3.8, 4.1, 4.4, 4.8,

Table 4.14 Inhalation exposure results for TDCP measured at Plant B

Job title or work area	Inhalation TWA 8 h (µg/m³)
Mixing head op. I	<0.20
Mixing head op. II	1.25
Mixing head op. III	<0.20
Side Paper take-off operator	<0.20
Cut-off block operator	<0.20
Cut-off Start/End operator	<0.20
Bottom Paper operator	0.39
Loop slitter operator	<0.20

Table 4.15 Inhalation exposure results for V6 measured at Plants X and Y

Plant identification	Operator	n	Inhalation Exposure 8-hr TWA (µg /m³)
Plant X	Mixing Head	2	<0.62, <0.62
Plant X	Asst. Mixing Head	4	<0.60, <0.53, <0.61, <0.63
Plant X	Side Paper Take Off	4	<0.62, 5.29, <0.63, <0.53
Plant X	Bottom Paper	4	<0.59, <0.56, <0.59, <0.57
Plant X	Block Cutter	2	<0.64,<0.59
Plant X	Laminator	4	1.7, 2.7, 6.0, 7.0
Plant X	Cutter	2	2.0, 2.6
Plant Y	Mixing Head	3	0.77, <0.58, <0.58
Plant Y	Side Paper Take Off	1	<0.63
Plant Y	Cut Off Block	1	<0.59
Plant Y	Cut Off Start/End	1	<0.58
Plant Y	Bottom Paper	1	<0.59
Plant Y	Loop slitter	1	<0.59

Table 4.16 Inhalation exposure for TCPP measured at flexible foam manufacturing plants

Operator	Operator Activity or Location	PPE Worn	Length of time monitored (mins)	Measured TCPP (μg/m³)	Calculated 8-hr TWA (μg/m³)[j]
Production op. 1 (plant 1)	Mixing head area	Protective gloves	429	10	8.9
Production op. 2 (plant 1)	Paper take-off area	Respirator with replaceable filter and protective gloves (when entering the tunnel)	404	32	26.9
Production op. 3 (plant 1)	Temperature supervision and probing	None	426	15	13.3
Production op. 5 (plant 2)	Mixing head area	Disposable gloves	239	7.3	3.6
Production op. 6 (plant 2)	Different areas of the line	Respirator with replaceable filter and protective gloves when removing polyethylene film and cleaning tunnel	242	9.7	4.8
Production op. 7 (plant 2)	End of the tunnel	Respirator with replaceable filter and protective gloves when marking block and putting polyethylene film on	236	9.4	4.6
Sampling op. (plant 2) *	Sampling and baler production	Protective gloves	403	17	14.2

The range of results for inhalation exposure deemed to be relevant to this scenario is <0.2 to 26.9 μg/m^3.

Measured dermal exposure data

Dermal exposure results from Plants A and B are presented in **Tables 4.17** and **4.18**, respectively. Relevant dermal exposure data with V6 and TCPP are presented in **Tables 4.19** and **4.20** below.

Table 4.17 Dermal exposure results for TDCP measured at Plant A

Job title or work area	n	mg TDCP / pair of gloves (mg/day)
Mixing head area	6	3.4, 3.9, 11.5, 36.9, 41.6, 49.5
Paper take-off area	4	2.0, 3.0, 8.0, 12.6
Cut-off area	1	27.0
Block preparation	2	0.4, 1.8
Machine operator	7	0.06, 0.1, 0.2, 0.3, 0.6, 2.5, 3.0

Table 4.18 Dermal exposure results for TDCP measured at Plant B

Job title or work area	mg TDCP/ pair of gloves (mg/day)
Mixing head op. I	0.032
Mixing head op. II	0.052
Mixing head op. III	0.17
Side Paper take-off operator	0.029
Cut-off block operator	0.173
Cut-off Start/End operator	0.124
Bottom Paper operator	0.141
Loop slitter operator	0.41

Table 4.19 Dermal exposure results for V6 measured at Plants X and Y

Plant Identification	Operator	n	mg V6 /pair of gloves (mg/day)
Plant X	Mixing Head	2	0.06, 1.39
Plant X	Asst. Mixing Head	4	0.20, 0.31, 0.79, 1.47
Plant X	Side Paper Take Off	4	0.08, 0.12, 0.21, 0.48
Plant X	Bottom Paper	4	0.28, 0.39, 1.18, 7.99,
Plant X	Block Cutter	2	0.14, 0.28
Plant X	Cutter	2	2.79, 6.33
Plant X	Laminator	4	3.86, 4.0, 5.36, 6.16
Plant Y	Mixing Head	3	0.49, 0.54, 0.75
Plant Y	Supervisor	1	0.89
Plant Y	Side Paper Take Off	1	0.39
Plant Y	Cut Off Block	1	0.34
Plant Y	Cut Off Start/End	1	0.23
Plant Y	Bottom Paper	1	0.24
Plant Y	Loop slitter	1	0.38

Table 4.20 Dermal exposure results for TCPP measured at flexible foam manufacturing plants

Operator	Length of time monitored (mins)	Measured TCPP (mg/kg bw)	Mg/day
Production op. 1 (plant 1)	430	1.5	105
Production op. 2 (plant 1)	443	0.45	31.5
Production op. 3 (plant 1)	429	0.68	47.6
Production op. 4 (plant 1)	445	0.09	6.3
Production op. 5 (plant 2)	239	0.32	22.4
Production op. 6 (plant 2)	242	0.39	27.3
Production op. 7 (plant 2)	236	0.01	0.7
Sampling op. (plant 2)	313	0.003	0.21

The range of results for dermal exposure deemed to be relevant to this scenario is 0.029 to 105 mg/day.

Values taken forward to risk characterisation

The RWC inhalation exposure value taken forward for risk characterisation is 4.8 μg/m^3. This is the 90th percentile of the data set used for this scenario. The typical exposure taken forward for risk characterisation is 0.63 μg/m^3, which is the median value of the data set used for this scenario, in line with guidance in the TGD.

The RWC dermal exposure value taken forward for risk characterisation is 7.5 x 10^{-2} mg/cm^2/day or 31.5 mg/day. This is the 90th percentile of the data set used for this scenario, and assumes a bodyweight of 70 kg and an exposure area of 420 cm^2. The typical dermal exposure value taken forward for risk characterisation is 1.5 x 10^{-3} mg/cm^2/day or 0.63 mg/day. This is the median value of the data set used for this scenario and is taken forward in line with TGD guidance, and assumes the same bodyweight and exposure area as above.

Scenario 3: Occupational exposure during cutting of flexible PUR foam

Blocks of polyurethane foam generally have to be cut into the required size/shape of the final product. This operation usually occurs after the blocks have cured and cooled. Blocks are sold to foam cutters who cut them into the required size and shape. Foam producers operate their own cutting facilities, but also sell to a large number of foam cutters, most of which (in the UK at least) are small, privately owned companies. The trimmed blocks of foam are cut into the required shapes/pieces by band-knives. In the UK alone, there are hundreds of foam cutters. Therefore, the potential number of workers exposed is extensive.

This scenario also covers the instance where furniture manufacturers may cut their own foam to shape, although it has been stated by industry that this rarely happens.

Measured exposure data

A small number of inhalation and dermal exposure measurements have been taken in the foam cutting departments of two polyurethane foam manufacturing plants by industry. These samples were collected in 2005. The samples were collected and analysed as described in Scenario 2, manufacture of flexible polyurethane foam. These data have been used in Scenarios 2a and 2b.

Inhalation exposure data

In Plant A, nine personal inhalation samples were collected; two during block preparation for cutting and seven on operators operating the loop slitter. In Plant B, one inhalation result was obtained from a loop slitter operator. These are presented in **Table 4.21**.

In addition to data from Plants A and B, data are also included from a V6 monitoring exercise in Plants X, Y and Z, and from a flexible foam manufacturing plant using TCPP. The activities are the same and there is the possibility of exposure to dust from cutting foam containing flame retardant. It is therefore considered valid to utilise these data to supplement the TDCP data. These data are presented in **Tables 4.22** and **4.23**.

Table 4.21 Personal inhalation exposure to TDCP measured at Plants A and B

Plant identification	Job title or work area	n	Inhalation TWA 8 h (µg/m³)
Plant A	Block preparation	2	3.0, 0.8
Plant A	Machine operator	7	1.7, 1.9, 3.8, 3.8, 4.1, 4.4, 4.8,
Plant B	Loop slitter operator	1	<0.20

Table 4.22 Personal inhalation exposures to V6 measured at Plants X, Y and Z

Plant identification	Operator	n	Inhalation TWA 8 h (µg /m³)
Plant X	Block Cutter	2	<0.64,<0.59
Plant X	Loop slitter	1	<0.59
Plant Y	Loop slitter	1	<0.59
Plant Z	Cutter	2	2.0, 2.6

Table 4.23 Personal inhalation exposure to TCPP during cutting of foam

Operator	Operator activity or location	PPE worn	Length of time monitored (mins)	Measured TCPP (µg/m³)	Calculated 8-hr TWA (µg/m³)
Operator at convoluter	Convoluter	None	135	5.4	1.5

The 17 personal inhalation exposures ranged between <0.2 µg/m^3 to 4.8 µg/m^3.

Dermal exposure data

In Plant A, nine personal dermal samples were collected; two during block preparation for cutting and seven on operators operating the loop slitter. In Plant B, one dermal exposure value was obtained from a loop slitter operator. **Table 4.24** below summarises the data. In addition, monitoring data during the cutting of flexible foam with V6 and TCPP are presented in **Tables 4.25** and **4.26** below.

Table 4.24 Personal dermal exposure to TDCP measured at Plants A and B

Plant identification	Job title or work area	n	mg TDCP /pair of gloves (mg/day)
Plant A	Block preparation	2	0.4, 1.8
Plant A	Machine operator	7	0.06, 0.1, 0.2, 0.3, 0.6, 2.5, 3.0
Plant B	Loop slitter operator	1	0.41

Table 4.25 Personal dermal exposure to V6 measured at Plants X, Y and Z

Plant Identification	Operator	n	mg V6 /pair of gloves (mg/day)
Plant X	Block Cutter	2	0.14, 0.28
Plant Y	Cut Off Block	1	0.34
Plant Y	Loop slitter	1	0.38
Plant Z	Cutter	2	2.79, 6.33

Table 4.26 Dermal exposure to TCPP in flexible foam manufacturing plants

Operator	Length of time monitored (mins)	Measured TCPP (mg/kg bw)	mg/day
Operator 1 at convoluter	135	0.28	19.6
Operator 2 at convoluter	130	0.017	1.19

The 18 personal dermal exposures ranged from 0.06 mg/day to 19.6 mg/day. The highest result was obtained from a machine operator who was operating a convoluter and was exposure to TCPP. During the other sampling periods the operators were handling foam which contained a maximum of 3.5% TDCP.

Values taken forward to risk characterisation

The value taken forward for risk characterisation for inhalation exposure is 4.1 $\mu g/m^3$, which is the 90th percentile of the data set. The typical exposure taken forward is 1.9 $\mu g/m^3$, which is the median value of the results presented by industry.

The value taken forward for risk characterisation for dermal exposure is 7.1 x 10^{-3} mg/cm^2/day or 3.0 mg/day. This is the 90th percentile of the results presented by industry, and assumes a bodyweight of 70 kg and an exposure area of 420 cm^2. The typical dermal exposure value taken forward for risk characterisation is 9.8 x 10^{-4} mg/cm^2/day or 0.41 mg/day which is the median value of the results, assuming the same bodyweight and exposure area as above.

Scenario 4: Occupational exposure during the production of foam granules and rebonded foam

Introduction

TDCP is present in off-cuts of slabstock foam, which can be recycled into rebonded foam. Scrap foam can be shredded and granulated for use as a loose crumb for low grade furnishing such as garden furniture. The shredding and granulating processes do not introduce new TDCP. The scrap foam is supplied in bales. In larger factories the bale would be fed directly into a breaker using a forklift truck. In other factories the foam would be fed onto a conveyor by hand and then into the breaker. The breaker breaks the scrap foam into smaller pieces for the granulator machine which has extraction. The operators would have no exposure during these processes as they are closed. Once the foam is granulated it is bagged for use in furniture manufacture. Scrap foam can also be shredded, granulated and rebonded into foam blocks.

As described in section 2.2.2.1.4, approximately 45 kilotonnes of rebonded foam were produced in the EU, and it was estimated that approximately 60 kilotonnes are rebonded in total. A high proportion of this is produced in the UK (approximately 22 kilotonnes). Across the EU, only a low proportion of this will contain flame retardants. Cheaper non-FR foam trim can be obtained exclusively but it is likely that a site rebonding FR-PUR will also be handling non-FR foam. It has been estimated that a typical site might rebond 3-5 kilotonnes of foam per year in total. Some of this scrap foam will contain TDCP. In Europe, the major use of rebond is reported to be in garden furniture (pers. comm., not attributable).

In addition a proportion of scrap foam is shredded and granulated, but is not rebounded. This loose crumb is reportedly used in low-grade furniture, such as cushions for garden furniture.

Measured inhalation exposure data

There is only one data point for inhalation exposure during the production of rebonded foam. This was from Plant B, which was collected and analysed as described in Scenario 2, manufacture of flexible polyurethane foam. This result was <0.2 μg/m^3, which is lower than the limit of detection for the method. However, there are other data that are considered to be relevant to this scenario; the results for operators handling newly-formed foam as it leaves the tunnel and is cut into blocks, in Plants A and B, as described in section 4.1.1.1.2.

In addition to the monitoring data on TDCP from Plants A and B, data are also included from a V6 monitoring exercise in Plants X and Y. The activities are the same. It is therefore considered valid to utilise these data to supplement the TDCP data. There are also two data points from exposure measurements made at a foam manufacturing plant using TCPP which are considered relevant here.

Table 4.27 below summarises the measured inhalation exposure data from Plants A and B. **Table 4.28** summarises the inhalation exposure for V6 measured at Plants X and Y. **Table 4.29** summarises the inhalation data from a plant using TCPP.

Table 4.27 Inhalation exposure for TDCP measured at Plants A and B

Plant Identification	Job title or work area	n	Inhalation TWA 8 h (μg/m^3)
Plant A	Cut-off area	2	<0.2, 1.7
Plant B	Rebond operator	1	<0.20
Plant B	Cut-off block operator	1	<0.20

Table 4.28 Inhalation exposure for V6 measured at Plants X and Y

Plant identification	Operator	n	Inhalation Exposure 8-hr TWA (μg /m^3)
Plant X	Block Cutter	2	<0.64,<0.59
Plant Y	Rebond	1	<0.60
Plant Y	Cut Off Block	1	<0.59
Plant Y	Cut Off Start/End	1	<0.58

Table 4.29 Inhalation exposure for TCPP measured at a foam manufacturing plant

Operator	Operator activity or location	PPE worn	Length of time monitored (mins)	Measured TCPP (μg/m^3)	Calculated 8-hr TWA (μg/m^3)
Production op. 7 (plant 2)	End of the tunnel	Respirator with replaceable filter and protective gloves when marking block and putting polyethylene film on	236	9.4	4.6
Sampling op. (plant 2)	Sampling and baler production	Protective gloves	403	17	14.2

The range of all results is <0.2 to 14.2 μg/m^3. Of the eleven results available, eight were below the limit of detection.

Measured dermal exposure data

There is only one data point for dermal exposure during the production of rebonded foam. This was from Plant B, which was collected and analysed as described in Scenario 2, manufacture of flexible polyurethane foam. The result was 0.01 mg/day. There are other data that are considered to be relevant to this scenario: the results for operators handling newly-formed foam as it leaves the tunnel and is cut into blocks, in Plants A and B, as described in section 4.1.1.1.2.

In addition to the monitoring data on TDCP from Plants A and B, data are also included from a V6 monitoring exercise in Plants X and Y. The activities are the same. It is therefore considered valid to utilise these data to supplement the TDCP data. There are also two data points from a foam manufacturing plant using TCPP which are considered relevant here.

The dermal exposures to TDCP measured at Plants A and B are presented in **Table 4.30** below. **Table 4.31** summarises the measured dermal exposures to V6 at Plants X and Y. The results from the TCPP plant are presented in **Table 4.32**.

Table 4.30 Dermal exposure for TDCP measured at Plants A and B

Plant Identification	Job title or work area	n	mg TDCP/pair of gloves (mg/day)
Plant A	Cut-off area	1	27.0
Plant B	Rebond operator	1	0.01
Plant B	Cut-off block operator	1	0.173

Table 4.31 Dermal exposure for V6 measured at Plants X and Y

Plant Identification	Operator	n	mg V6 /pair of gloves (mg/day)
Plant X	Block Cutter	2	0.14, 0.28
Plant Y	Rebond	1	0.03
Plant Y	Cut Off Block	1	0.34
Plant Y	Cut Off Start/End	1	0.23

Table 4.32 Dermal exposure for TCPP measured at a foam manufacturing plant

Operator	Length of time monitored (mins)	Measured TCPP (mg/kg bw)	mg/day
Production op. 7 (plant 2)	236	0.01	0.7
Sampling op. (plant 2)	313	0.003	0.21

The range of results is 0.01 to 27 mg/day. The variation is quite wide. There are eight results, with seven of the eight results less than 0.5 mg/day.

Values taken forward to risk characterisation

The RWC exposure value for inhalation taken forward for risk characterisation is 4.6 $\mu g/m^3$. This is the 90^{th} percentile of the data presented. The typical inhalation value taken forward for risk characterisation is 0.59 $\mu g/m^3$, which is the median value.

The RWC taken forward for dermal exposure is 0.7 mg/day or 1.7 x 10^{-3} mg/cm^2/day, with an exposure area of 420 cm^2. This value is the second highest of the dataset gathered from relevant operations from manufacture of foam containing TCPP, TDCP or V6. The highest value was two orders of magnitude higher than the next, so is considered to be an outlier.

The typical exposure taken forward for risk characterisation for dermal exposure is 0.23 mg/day or 5.5 x 10^{-4} mg/cm^2/day, which is the median value for the dataset gathered from relevant operations from manufacture of foam containing TCPP, TDCP and V6.

Scenario 5: Occupational exposure during the manufacture of automotive parts

Introduction

Data have been provided by producers of TDCP and by companies using TDCP in the production of foams for automotive applications. The number of sites using TDCP is known. Many parts of motor cars are made from PUR foam, including interior trim, seats, headrests and dashboards, soundproofing, filters, etc (Europur, 2002).

The manufacture of moulded foam is covered in Scenario 2b. This scenario covers the use of flexible foam in the manufacture of automotive products. Data provided by a foam producer indicates that TDCP is used in the production of foams for use with textiles in the manufacture of car seat, door panels, soundproofing, head-liners and cushions. The bulk of the seats are made using foam that does not contain flame retardant. It is only the outer covering of foam associated with the covering fabric that contains TDCP. The assembly processes will vary depending on the product being made, but will usually involve the use of adhesives to laminate foam and the material being used for the interior of the car, cutting, trimming and stitching of components. Different operatives would carry out different tasks, so that, for example, one operator would laminate the foam and fabric, another would stitch and trim the seat covering and another would assemble the seat. These processes may be carried out within the same company, but it is more usual for different companies to carry out different stages of the production.

There are no exposure data available for the manufacture of automotive products so exposure data from the handling and cutting of flexible PUR foam provided by industry have been used. The potential for exposure arises during the handling of the foam, and during the cutting and trimming of the foam-backed material.

In addition to the monitoring data on TDCP from Plants A and B, data are also included from a V6 monitoring exercise in Plants X and Y and for cutting TCPP foam. The activities are the same. It is therefore considered valid to utilise these data to supplement the TDCP data.

The activities are not strictly directly comparable, as the flexible foam manufacturers will be handling much larger quantities of foam and the cutting takes place using machinery, whereas the automotive product manufacturers will be handling smaller quantities of foam, but will be trimming and cutting by hand. However, it is considered that real exposure data will give a better approximation of exposure than using EASE in this instance.

Measured inhalation exposure data

Table 4.33 below summarises the TDCP inhalation exposure data from Plants A and B and **Table 4.34** details the inhalation exposure to V6 at Plants X, Y and Z. The relevant TCPP data is presented in **Table 4.35**

Table 4.33 Inhalation data for TDCP measured at Plants A and B

Plant identification	Job title or work area	n	Inhalation TWA 8 h (µg/m^3)
Plant A	Block preparation	2	0.8, 3.0
Plant A	Machine operator	7	1.7, 1.9, 3.8, 3.8, 4.1, 4.4, 4.8,
Plant B	Loop slitter operator	1	<0.20

Table 4.34 Inhalation data for V6 measured at Plants X, Y and Z

Plant identification	Operator	n	Inhalation Exposure 8-hr TWA (µg /m^3)
Plant X	Block Cutter	2	<0.64,<0.59
Plant Y	Cut Off Block	1	<0.59
Plant Y	Cut Off Start/End	1	<0.58
Plant Y	Loop slitter	1	<0.59
Plant Z	Cutter	2	2.0, 2.6

Table 4.35 Inhalation exposure at foam manufacturing plant using TCPP

Operator	Operator activity or location	PPE worn	Length of time monitored (mins)	Measured TCPP (µg/m^3)	Calculated 8-hr TWA (µg/m^3)
Production op. 7 (plant 2)	End of the tunnel	Respirator with replaceable filter and protective gloves when marking block and putting polyethylene film on	236	9.4	4.6
Sampling op. (plant 2)	Sampling and baler production	Protective gloves	403	17	14.2

The range of inhalation values relevant to this scenario is <0.2 to 14.2 µg/m^3.

Measured dermal exposure data

Dermal exposures to TDCP at Plants A & B and to V6 at Plants X,Y and Z are presented in **Tables 4.36** and **4.37**, respectively. **Table 4.3**8 summarises the dermal exposure to TCPP during foam manufacture.

Table 4.36 Dermal exposure data for TDCP measured at Plants A and B

Plant identification	Job title or work area	n	mg TDCP/pair of gloves(mg/day)
Plant A	Block preparation	2	0.4, 1.8
Plant A	Machine operator	7	0.06, 0.1, 0.2, 0.3, 0.6, 2.5, 3.0
Plant B	Loop slitter operator	1	0.41

Table 4.37 Dermal exposure data for V6 measured at Plants X, Y and Z

Plant Identification	Operator	n	mg V6 /pair of gloves (mg/day)
Plant X	Block Cutter	2	0.14, 0.28
Plant Y	Cut Off Block	1	0.34
Plant Y	Cut Off Start/End	1	0.23
Plant Y	Loop slitter	1	0.38
Plant Z	Cutter	2	2.79, 6.33

Table 4.38 Dermal exposure data for TCPP measured at a foam manufacturing plant

Operator	Length of time monitored (mins)	Measured TCPP (mg/kg bw)	mg/day
Operator 1 at convoluter	135	0.28	19.6
Operator 2 at convoluter	130	0.017	1.19

The range of dermal exposure values relevant to this scenario is 0.06 to 19.6 mg/day.

Values taken forward for risk characterisation

The value taken forward for risk characterisation for inhalation exposure is 4.6 $\mu g/m^3$, which is the 90th percentile of the results presented by industry. The typical exposure taken forward is 1.9 $\mu g/m^3$, which is the median value of the results presented by industry.

The value taken forward for risk characterisation for dermal exposure is 7.1 x 10^{-3} mg/cm^2/day or 3.0 mg/day. This is the 90th percentile of the results presented by industry, and assumes a bodyweight of 70 kg and an exposure area of 420 cm^2. The typical dermal exposure value taken forward for risk characterisation is 9.8 x 10^{-4} mg/cm^2/day or 0.41 mg/day which is the median value of the results presented by industry, assuming the same bodyweight and exposure area as above.

Summary of occupational exposure

A summary of the inhalation and dermal exposures values taken forward to risk characterisation for each scenario are presented in **Table 4.39**.

Table 4.39 Summary of RWC and typical exposure values for inhalation and dermal exposure for all scenarios taken forward for risk characterisation

Scenario	Inhalation exposure ($\mu g/m^3$)		Dermal exposure ($mg/cm^2/day$)		Dermal exposure area (cm^2)
	RWC	Typical	RWC	Typical	
1.Occupational exposure during manufacture of TDCP	5.6	2.8	0.1	5×10^{-2}	210
2a. Occupational exposure during manufacture of flexible PUR foam	5.1	0.62	7×10^{-2}	2×10^{-3}	420
2b. Occupational exposure during manufacture of moulded foam	4.8	0.63	7.5×10^{-2}	1.5×10^{-3}	420
3. Occupational exposure during cutting of flexible PUR foam	4.1	1.9	7.1×10^{-3}	9.8×10^{-4}	420
4. Occupational exposure during production of foam granules & rebonded foam	4.6	0.59	1.7×10^{-3}	5.5×10^{-4}	420
5. Occupational exposure during manufacture of automotive parts	4.6	1.9	7.1×10^{-3}	9.8×10^{-4}	420

Consumer exposure

Potential exposure from flexible polyurethane foam

The current use pattern provided by industry indicates that most of the TDCP produced in the EU in 2000 was used in the production of flexible polyurethane foam in Europe. Most of the TDCP used in flexible foam is for the automotive industry, with some used in furniture. Consumers do not come into direct contact with these foams. The foam is only used in ways in which it is enclosed and therefore it is concluded that exposure to consumers is negligible.

Measured consumer exposure data

There is a small amount of measured data available for exposure of consumers to TDCP from flexible PUR foam. There is also some data available for TCPP, so it is proposed to use this additional data here to get an estimate of consumer exposure to TDCP. The study and results are outlined below.

Chamber tests of TCPP-containing flexible PUR foams for release of TCPP

In order to evaluate possible indoor air concentrations of TCPP from flexible foam used in mattresses, EUROPUR (European Association of Flexible Polyurethane Foam Block Manufacturers) ordered chamber tests at the Institute Miljo-Kemi in Denmark. In the study, a 'worst-case' scenario was applied. The foams were uncovered, the quantity of foam in the mattress was a maximum (i.e. full depth foam with no springs) and the chamber volume was small. In everyday use, the mattress foam is always covered with a fabric material and of course bedding sheets, blankets, etc.

Three types of flexible PUR foam used in mattresses were tested. The samples were 2000 x 1000 x 120 mm of full depth foam (i.e. no springs), were uncovered and were reported to contain TCPP at the high end of the typical level for this application (reported to be 2.5 –

14%, 7 – 8% on average, based on industry data collected for the risk assessment of TCPP). The mattresses were placed in a 3.2 m3 test chamber at 23°C and relative humidity of 50%, with an air exchange rate of 0.5 per hour. Volatile emissions were collected on Tenax TA absorbent and analysed by GC-MS. The limit of detection was reported as 2 μg/m3. **Table 4.40** below gives the results of this monitoring study.

Table 4.40 Results of chamber tests with mattresses made of TCPP-containing foam

Mattress Type	Air Concentration (μg/m³)				
	24h	48h	72h	120h	160h
HR[1]	6.0	22	25	19	10
CME 33[2]	9.1	16	16	19	17
CMHR[3]	1.8	1.7	2	<1	<1

[1]HR = High resilience foam, 36 kg/m³, 1.5% TCPP. [2]CME = Combustion modified ether, 33 kg/m³. [3] CMHR = Combustion modified high resilience foam, 35 kg/m³

The detection limit for this test was 2 $\mu g/m^3$. It can be seen from the results that after 160 hrs, the concentration of TCPP in the chamber is declining in the case of HR foam, whereas for CME foam, it remains relatively constant. No TCPP was detected from the CMHR foam from 120 hours onwards.

An estimation of TCPP indoor air concentration can be made from this study. As a worst-case approach, a room with a high PU foam load should be assumed. In the study, the CME foam gave the highest levels of TCPP in the chamber air. Therefore, this will be used for the estimation. The assumptions are as follows:

TCPP concentration in chamber air: 19 $\mu g/m^3$
Mattresses in the room: 2 Factor 2
Volume of room: 30 m^3 Factor 1/10
Air exchange: 0.5 h^{-1} Factor 1
This gives a concentration of TCPP in indoor air to be 3.8 $\mu g/m^3$ (19 x 2 x 1/10).

Determination of flame retardant retention in CMHR flexible foam sample

Polyurethane foam storage trials have been performed in two UK foam companies. The British Rubber Manufacturer's Association (BRMA) has provided the rapporteur with the results of the biannual analyses for these trials. Initial tests determined the distribution of flame retardant across the foam sample. Foam pieces were taken from a foam block and analysed for phosphorous and chlorine content using an internal validated method. The results obtained in this initial study showed good flame retardant distribution across the foam. Through the rest of the study, phosphorous and chlorine measurements were made on the foam on a six monthly basis over a period of almost eight years (from 1998 – 2005). **Table 4.41** below gives a summary of the results obtained for this study.

Table 4.41 Results of BRMA long-term ageing trial on polyurethane foam from one company

Time (months)	Company A (TDCP)		Company B (TCPP)	
	% P	% Cl	% P	% Cl
0	0.75	2.6	0.40	1.3
80°C for 100 h	0.74	2.5	-	-
6	-	-	0.39	1.7
12	0.74	2.5	0.41	1.4
18	0.75	2.7	0.40	1.2
24	0.70	2.7	0.39	1.3
30	0.72	2.7	0.37	1.3
36	0.71	2.6	0.39	1.3
42	0.73	2.6	0.40	1.2
48	0.72	2.6	0.40	1.2
54	0.74	2.5	0.41	1.2
60	0.73	2.4	0.42	1.2
78*			0.44	1.42
84*			0.45	1.42
90			0.44	1.48

* Change of analytical laboratory

From this ageing study, it can be seen that flame retardants are retained within PUR foam, and so consumer exposure to flame retardants from these foams is expected to be very low.

Further work carried out by the University of Surrey looked at release of flame retardant from PUR foams. The results of this work suggest higher rates of release of FRs than the above two studies, but they looked at smaller pieces of foam and dust. The dust had a much higher rate of release, suggesting that the size of the foam pieces influenced the rate of release.

As the work carried out by EUROPUR and BRMA looked at mattress-sized pieces of foam, this data has been used to estimate consumer exposure via inhalation.

Dermal exposure

There are no data on dermal exposure. However, it is reasonable to assume that dermal exposure will not exceed inhalation exposure and therefore the data on inhalation will also be used for dermal exposure as a RWC. For dermal exposure the figure for inhalation will be put forward as a RWC for risk characterisation; that is 0.0011 mg/kg.

Oral exposure

This route of exposure is only of significance for young children, due to their hand to mouth behaviour. In this section, information has been taken from the TCEP exposure assessment (BAUA, 2006). This is considered a valid means of generating information for risk characterisation as the two substances have similar vapour pressures and molecular weights.

It has been estimated that a three year old child would consume 100 mg dust per day (including soil). It has also been shown that the range of TCEP in house dust is 0 to 121 mg/kg. The 95th percentile of this range is 11.9 mg/kg.

Oral TCEP uptake was calculated by the formula

$$E_{TCEP(oral)} = \frac{C_{TCEP,\,dust} * I_{orl,\,dust}}{BW}$$

where $C_{TCEP,\,dust}$ is the dust concentration, $I_{orl,dust}$ is the uptake of dust, and BW is the body weight. According to the age categories of the AUH Report (1995), the oral exposure was estimated for a 1-3 year old child. The dust uptake and body weight data (normal distribution, weighted for 1 to 3 year of age) are taken from the AUH Report (1995). The dust uptake data are primarily based on the data published by Calabrese *et al*. (1989). According to these data, the values for this assessment were set as follows: normal dust uptake is set to 20 mg/d and the 95th percentile to 100 mg/d.

This estimation of uptake includes soil uptake and therefore leads to a slight overestimate of exposure via dust. It should be mentioned that the upper range of the uptake determined by Calabrese is in agreement with newer data obtained by Freeman and Adgate (2003) who found a daily dust uptake of 100 mg in small children.

The 95th percentile, 99th percentile and the maximum value for children, representing a vulnerable population due to their specific hand-mouth behaviour are 0.1, 0.2 and 0.7 µg/kg/day, respectively.

The 99th percentile of TCEP ingested with house dust, of 0.2 µg/kg/day, has been taken forward as a RWC for oral ingestion for a child, in line with the TCEP risk assessment.

Values taken forward to risk characterisation

A RWC inhalation exposure value of 3.8 µg/m^3 24 hour TWA will be taken forward for risk characterisation. A typical exposure value of 2.8 µg/m^3 will be taken forward for risk characterisation, on the basis of a consumer spending 18 out of 24 hours in rooms where there is PU foam-containing furniture. These figures are based on TCPP exposure so are likely to be an over-estimate of exposure to TDCP, but there is no data available for TDCP.

For dermal exposure, the figure for inhalation will be put forward as a RWC for risk characterisation that is 0.0011 mg/kg.

These figures have been put forward on the basis of the chamber test work carried out as described above. However, the work ongoing to monitor the release of fire retardant from foam over years rather than hours seems to indicate that the loss of fire retardant is negligible, in which case exposure would be negligible. The values taken forward for risk characterisation may therefore be an over-estimate.

A value for a RWC oral ingestion for children has been taken from the risk assessment for TCEP of 0.2 µg/kg/day, assuming a bodyweight of 9.1 kg.

Humans exposed via the environment

Table 4.42, which is taken from section 3 of this report, gives the predicted environmental exposures to TDCP and the daily human doses arising from releases from production, processing, manufacture and use of TDCP. It also provides the predicted environmental exposures at a regional level.

It can be seen that the daily human intake via the environment based upon typical human consumption and inhalation rates at the regional level is 1.52×10^{-5} mg/kg/day and the highest local exposure (industrial use) is 0.0346 mg/kg/day (Confidential Use C2).

However, the Rapporteur has been informed that TDCP is no longer supplied or used for Confidential uses C (C1a, C1b and C2) and D (D1 and D2) (pers. comm. 30th October 2007, Supresta). Therefore, although the two highest local exposures result from these uses, they will not be taken forward to risk characterisation as they are not current uses.

The next highest local total daily intake is 6.99×10^{-4} mg/kg/day (Confidential Use E1b) and this value, along with the regional exposure estimate of 1.52×10^{-5} mg/kg/day will be taken forward to risk characterisation.

Table 4.42 Indirect exposure of humans to TDCP via the environment

	Air [mg.kg-1.d-1]	Drinking water [mg.kg-1.d-1]	Fish [mg.kg-1.d-1]	Leaf crops [mg.kg-1.d-1]	Meat [mg.kg-1.d-1]	Milk [mg.kg-1.d-1]	Root crops [mg.kg-1.d-1]	Local total daily intake [mg.kg-1.d-1]
Producer 1	2.21E-08	2.74E-06	7.08E-06	2.58E-06	8.51E-09	5.02E-09	8.28E-06	2.07E-05
Producer 2	4.12E-09	8.99E-06	1.86E-06	1.93E-05	2.78E-08	1.64E-08	8.06E-05	1.11E-04
A1a: Flexible foam - automotive - foaming large site	1.59E-08	1.42E-06	2.10E-06	3.47E-06	7.63E-09	4.50E-09	1.27E-05	1.97E-05
A1b: Flexible foam - automotive - foaming	5.58E-09	9.69E-07	1.70E-06	2.22E-06	5.70E-09	3.36E-09	8.68E-06	1.36E-05
A2: Foam cutting	9.34E-09	1.13E-06	1.85E-06	2.68E-06	6.40E-09	3.77E-09	1.02E-05	1.58E-05
B1: Flexible foam - furniture - foaming	1.06E-08	1.41E-06	1.90E-06	3.31E-06	7.27E-09	4.28E-09	1.27E-05	1.93E-05
B2: Foam cutting	6.29E-09	1.00E-06	1.73E-06	2.31E-06	5.83E-09	3.44E-09	8.96E-06	1.40E-05
*C1a: CONFIDENTIAL	1.63E-05	1.88E-05	6.46E-06	4.93E-04	1.13E-06	6.65E-07	1.68E-04	7.05E-04
*C1b: CONFIDENTIAL	1.63E-05	4.85E-04	1.26E-04	1.49E-03	2.42E-06	1.43E-06	4.35E-03	6.47E-03
*C2: CONFIDENTIAL	3.28E-08	2.79E-03	8.77E-04	5.95E-03	7.73E-06	4.56E-06	0.025	0.0346
*D1: CONFIDENTIAL	2.18E-05	8.65E-04	3.99E-04	2.45E-03	3.83E-06	2.26E-06	7.75E-03	0.0115
*D2: CONFIDENTIAL	8.75E-07	1.86E-06	1.65E-06	2.83E-05	6.53E-08	3.85E-08	1.66E-05	4.94E-05
E1a: CONFIDENTIAL	6.94E-08	3.90E-05	1.90E-06	8.50E-05	2.28E-07	1.34E-07	3.49E-04	4.76E-04
E1b: CONFIDENTIAL	1.13E-07	5.70E-05	5.79E-06	1.25E-04	1.68E-07	9.90E-08	5.11E-04	6.99E-04
F1: CONFIDENTIAL	4.18E-09	4.28E-05	2.24E-05	9.14E-05	1.22E-07	7.16E-08	3.84E-04	5.41E-04
G1: CONFIDENTIAL	2.81E-08	6.81E-06	3.39E-06	1.53E-05	2.34E-08	1.38E-08	6.10E-05	8.66E-05
I1: Flexible foam - Furniture, seating, mattresses - re-bonding of scrap	1.35E-08	9.14E-07	1.65E-06	2.32E-06	6.07E-09	3.58E-09	8.19E-06	1.31E-05
J1: Loose Crumb	8.14E-09	9.08E-07	1.65E-06	2.16E-06	5.70E-09	3.36E-09	8.14E-06	1.29E-05

	Air [mg.kg-1.d-1]	Drinking water [mg.kg-1.d-1]	Fish [mg.kg-1.d-1]	Leaf crops [mg.kg-1.d-1]	Meat [mg.kg-1.d-1]	Milk [mg.kg-1.d-1]	Root crops [mg.kg-1.d-1]	Local total daily intake [mg.kg-1.d-1]
Regional	4.11E-09	1.11E-06	1.65E-06	2.47E-06	6.59E-09	3.88E-09	9.92E-06	1.52E-05

Combined exposure

The combined exposure to TDCP is the sum of all the specific sources (occupational exposure, consumer exposure and indirect exposure via the environment) and by all routes of exposure (oral, dermal and inhalation). Therefore, a worst case estimate for this combined exposure would be the sum of the RWC estimates, for inhalation and dermal exposures, for the three populations; i.e. workers, consumers and man exposed via the environment.

Occupational inhalation and dermal exposures for the identified worker exposure scenarios are presented in **Table 4.39** (see section 4.1.1.1.7). As can be seen from this table, the occupational exposure levels are significantly higher than the estimated exposure to consumers or indirect exposure via the environment. Therefore, as the occupational exposure estimate will dominate the combined exposure estimate, it is not considered necessary to include it in the combined exposure calculation.

Consumers may be exposed to TDCP indirectly from flexible foam used in upholstery and bedding. Exposure is also possible indirectly via environmental sources.

The RWC exposures used in calculating the combined exposure are presented in **Table 4.43** below.

Table 4.43 Exposures taken into account for combined TDCP exposure estimate (excluding occupational exposure)

Source of exposure	Exposure
Consumer	
Release of TCPP from flexible polyurethane foam	
Inhalation	0.0038 mg/m^3
Dermal	0.0011 mg/kg
Man via the environment	
Local exposure	6.99 x 10^{-4} mg/kg/day
Regional exposure	1.52 x 10^{-5} mg/kg/day

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

Toxicokinetics, metabolism and distribution

Studies in animals

In vivo studies

Inhalation

No studies are available.

Oral

The intestinal absorption and subsequent distribution of radioactively labelled TDCP was examined following oral administration of 0.2, 2 and 20 μmol/kg (corresponding to 86 μg/kg, 860 μg/kg and 8.6 mg/kg, respectively) in bile duct-cannulated male Sprague Dawley rats (Nomeir *et al*., 1981). The report does not say exactly how many rats were used per dose level, but 'at least 3 rats were used per treatment' (the study also investigated the i.v. and dermal routes; see relevant sections below). Faeces, urine and expired CO_2 were collected. Absorption from the rat GI tract was >90 % within 24 hours (the report does not detail exactly how this was measured). Tissue distribution after 24 hours indicates the following distribution pattern: kidney > liver > lung > blood > muscle.

Distribution of the radioactivity was unaffected by the size of the dose. There was no apparent effect of the route of administration on tissue distribution. The tissue/blood ratios for the total TDCP derived radioactivity at day 1 following oral or i.v. administration were similar for all tissues except the lung which may have been altered by first pass effect.

There was no information provided in the report on the excretion of TDCP following oral administration, as the report concentrated on the i.v. route for this.

In a comparative study on absorption, distribution, and excretion of flame retardants halogenated alkyl phosphate in rats (Minegishi *et al*., 1988), a group of 5 male Wistar rats were orally administered 50 μmol/kg ^{14}C-TDCP in olive oil (corresponding to 21.5 mg/kg). Urine and faeces were collected every 24 hours for 7 days. Expired $^{14}CO_2$ was determined after 72 and 96 hours. An additional five male rats received a single oral administration of 50 μmol/kg of ^{14}C-TDCP and bile was collected via cannulation every 2 hours for 30 hours, from 30 – 46 hours and from 46 – 48 hours. Tissue samples were taken at 3, 6, 12, 24, 72 and 168 hours.

The recovery of radioactivity after 168 hours was urine (43.2 %), faeces (39.2 %), expired air (16.24 %) and carcass (2.51 %) (total recovery was 101.8 %). Approximately 40 % of administered radioactivity was excreted via the bile. The average T_{max} value (average time at which TDCP reached the maximum concentration in the tissue) for TDCP radioactivity in blood and tissues was 9.6 hours. Tissue/blood ratios calculated at various intervals over 7 days were > 1 for liver and kidney indicating incorporation of radioactivity into these tissues. The decrease in radioactivity in all tissues was biphasic. The longest t½ was recorded in adipose tissue in both phases of elimination (17.8 hours and 92.4 hours, respectively). However, the concentration was low implying no bioaccumulation. (For example, the concentration in adipose tissue at 168 hrs post administration was 2.21 nmoles/g tissue). The biliary/faecal excretion ratio was 1.04 at 48 hours, suggesting no enterohepatic recirculation from the GI tract.

The disposition of TDCP following oral administration was studied in the rat by Matthews and Anderson (1979). The study was provided only in the form of an abstract, so very little information was available. The authors indicated that at least 90 % of an oral dose of TDCP was absorbed from the GI tract. Following GI absorption, TDCP was rapidly distributed throughout the body, the highest concentrations being recorded in the liver, kidney and lung. Traces of TDCP-derived radioactivity were detected in most tissues 10 days after exposure. Metabolic degradation was extensive. Metabolites were eliminated in bile, faeces, urine and air, as CO_2. Elimination was rapid with > 80% of the dose eliminated within 24 hours post dosing. Elimination in bile was greater than excretion via the faeces suggesting enterohepatic

recirculation. However, due to the very limited information supplied on this study, none of the data could be quantified.

Dermal

No studies are available.

Intravenous

The same authors (Nomeir *et al*, 1981) also investigated the metabolism and disposition of TDCP following intravenous administration to male Sprague Dawley rats. TDCP ([1,3-^{14}C]2-propyl, specific activity 12.5 mCi/mmol, radiochemical purity >99 %) was administered intravenously (tail; 2 μmol/kg, 0.867 mg/kg) dissolved in Emulphor EL-620:ethanol:water (1:1:8). At least 3 animals were used (same comment on this as previous).

Fifteen minutes after intravenous administration, the highest concentrations of administered radioactivity were detected in the lung (22.72 nmoles TDCP derived radioactivity per gram tissue), followed by the liver (6.19 nmoles/g tissue), kidney (3.73 nmoles/g tissue) and blood (3.45 nmoles/g tissue). TDCP derived radioactivity in most of the tissues, except lung, showed little decrease during the first 2 hours of the study. The higher concentration observed in the lung is possibly due to the product of first pass effect resulting from i.v. administration. A decrease in the radioactivity of most tissues (except skin) became apparent by 7 hours post exposure, and a marked decrease was obvious in all tissues by 24 hours after TDCP administration. By day 10, the remaining radioactivity was only 1-5 % of that observed 15 mins post-exposure.

The metabolites recovered from rat urine following i.v. administration were bis(1,3-dichloro-2-propyl) phosphate (BDCP 67.2 % of total urine radioactivity), an unidentified polar metabolite (32 %), 1,3-dichloro-2-propyl phosphate (0.29 %) and un-metabolised TDCP (0.45 %). The absence of 3-chloro-1,2-propanediol and 1,3-dichloro-2-propanol metabolites in the urine and the excretion of 20% of total radioactivity in exhaled air within 24 hours suggests that these metabolites may have been completely metabolised to CO_2. Metabolites in bile and faeces were not identified but accounted for ≥ 99% of the radioactivity in both cases.

TDCP was rapidly excreted. Following intravenous administration, approximately 34, 20 and 20 % of total radioactivity was excreted in the urine, faeces and expired air, respectively, within the first 24 hours by which time a marked (approximately 90 %) decrease in radioactivity was also recorded in all tissues. This was followed by a protracted decrease over the next 10 days by which time 1 - 5% of the original radioactivity remained. Approximately 47 % and 21 % of the total TDCP dose was excreted in the urine and faeces, respectively, within 10 days after administration. Biliary excretion of the administered dose was 27 % within 4 hours.

The tissue concentration of parent TDCP decreased exponentially during the first 2 hours after administration (the authors could differentiate between parent compound and metabolites as radioactivity was extracted from each tissue and fractioned by TLC in order to separate the parent compound and its metabolites). The half-life of TDCP clearance in tissues was between 1.5 and 5.4 hours. One day after exposure, only 20-30 % of the radioactivity remaining in tissue was the parent compound. The t½ of the remaining radioactivity was much longer than that of the parent TDCP.

A portion of TDCP was also metabolised to CO_2 and eliminated from the body in the exhaled air. Approximately 20% of total dose was exhaled as CO_2 during the first 24 hours after exposure. In all cases, the radioactivity excreted was primarily TDCP metabolites, rather than the parent compound since less than 1% of the dose was eliminated as parent compound.

In another study the disposition of TDCP following intravenous administration in male Sprague Dawley rats was investigated (Lynn *et al*., 1981). Four animals were used for investigation of excretion, 2 for biliary excretion, 4 for plasma kinetics and 8 for tissue distribution studies. Unlabelled TDCP and propyl-1,2-^{14}C-TDCP (purity 99 %; 10.04 μCi, specific activity 12.5 mCi/mmol) in 200 μl of aqueous Emulphor 719 (25 % v/v) was administered intravenously through jugular vein catheters (dose not given in study). Urine, faeces and CO_2 ($BaCO_3$) were collected at 24-hour intervals for 5 days and analysed by HPLC-LSC. Bile was collected over 24 hours and urine, faeces and CO_2 ($BaCO_3$) were analysed from these animals also. For plasma kinetic studies, blood samples were withdrawn at various intervals up to 24 hours after administration. Tissue distribution was determined in 2 animals/time point after 5 minutes, 30 minutes, 8 hours and 24 hours (the animals from the excretion studies used at this time point) and 120 hours.

TDCP was eliminated primarily through metabolism as opposed to excretion. The $t_{½}$ of TDCP in plasma was < 5 minutes. The decline in TDCP concentration was reciprocated by an increase in BDCP concentration which itself began to decline after 2 hours, giving a t½ of 4-6 hours. 46 % of TDCP was metabolised within 5 minutes, of which BDCP accounted for 16 %. This had risen to 82 % by 30 minutes, of which BDCP accounted for 27 %. Thereafter, at 8, 24 and 120 hours, 56, 59 and 63 % of TDCP radioactivity was recovered as BDCP. Neither compound showed any tendency toward bioaccumulation in fat.

The cumulative percentage of administered radioactivity recovered by 120 hours was 54.0, 16.4, 22.2 and 3.8 in the urine, faeces, expired $^{14}CO_2$, and carcass, respectively (mean ± SD of n = 4). TDCP rapidly distributed from plasma into tissues. Parent TDCP could be detected in all tissues at 5 min and 30 mins, but could be detected in fat only at 8 hours. TDCP was not detected in any tissue at 24 hours. Concentration of TDCP was highest in the kidney at 5 mins (6.75 nmoles/g), followed by the liver (2.75 nmoles/g), small intestine (1.98 nmoles/g) and the blood (1.84 nmoles/g). Highest concentrations of BDCP at 5 mins were in the lung (12.2 nmoles/g), blood (5.34 nmoles/g), liver (4.79 nmoles/g) and kidney (1.17 nmoles/g). BDCP was detectable up to 24 hours in all tissues but was not detected after 5 days.

Identification and quantification of the diester metabolites of TDCP in the rat was carried out in a study by Lynn *et al*. (1980). TDCP (0.342 mg, 10μCi) was administered intravenously to 4 male Sprague Dawley rats. Urine was collected for 5 days at 24 hr intervals. After 5 days 54 % of the intravenously administered radiolabel had been excreted in the urine. The radiolabelled components present in composite urine samples (0-120 hrs) were separated by HPLC. Bis (1,3-dichloro-2-propyl) phosphate was identified as the major component of radiolabel in the urine (62.3 %). The study did not attempt to identify any other metabolites present in the urine samples.

In a study investigating the *in vivo* binding of TDCP to macromolecules of mouse liver, kidney and muscle (Morales & Matthews, 1980), single intravenous doses of ^{14}C-TDCP (chemical/radiochemical purity 99 %; 94.4 μmoles/kg, 376 μCi/kg) dissolved in a 1:1:4 mixture of emulphor, ethanol and water were administered to 3 male CD-1 mice. The animals were sacrificed 6 hours later. Protein, DNA, ribosomal RNA and low molecular weight RNA were extracted from liver, kidney and muscle. At the 6-hour sacrifice, TDCP-derived radioactivity was greatest in the liver (51 ± 4 pmoles/mg) followed by kidney (31 ± 8

pmoles/mg) and muscle (5.2 ± 0.8 pmoles/mg). The highest concentration of bound radioactivity in the three tissues was to low molecular weight RNA (67 and 93 pmoles/mg for liver and kidney, respectively) followed by protein (57, 43 and 7.2 pmoles/mg, respectively), rRNA (28, 13 and 5.6 pmoles/mg, respectively) and DNA (8.3 and <1.0 pmoles/mg for liver and kidney, respectively). ≥ 95 % of the radioactivity associated with a macromolecule was covalently bound. Metabolism was through dealkylation of the phosphate group. The resulting halogenated alkyl group was metabolised to CO_2 that was expired or incorporated into endogenous molecules.

In vitro studies

Metabolism

In a study investigating the metabolism of phosphoric acid triesters (one being TDCP) by rat liver homogenate (Sasaki *et al*., 1984), microsomes and soluble fraction were isolated from freshly-excised male Wistar rat liver. The soluble fraction was dialyzed for 24 hours. The reaction mixtures (containing 20 - 50 μl of a 400 μM solution of TDCP substrate in ethanol) were incubated for 30 minutes at 37 ^{o}C. TDCP was metabolised by rat liver microsomes with an optimum pH of 7.4. The reaction went to 43 % completion within 30 minutes in the presence of NADPH and 7% completion in its absence. A number of MFO inhibitors were investigated and from the results obtained it was concluded that MFO in microsomes play an important role in the metabolism of TDCP. Bis(1,3-dichloroisopropyl) hydrogen phosphate accounted for 75% of the MFO-metabolised TDCP. TDCP was also metabolised by rat liver soluble fraction with a broad optimum pH range, the reaction going to 36 % completion. Dialyzed soluble fraction or soluble fraction incorporating 2,4-dinitrochlorobenzene (an inhibitor of glutathione-S-transferase) was incapable of metabolising TDCP. However, this capability was wholly or partly restored on addition of 1 mM GSH. These results indicate that glutathione-S-transferase in the soluble fraction is a major contributor to the TDCP metabolism. Since no metabolites were extracted by organic solvent from the soluble fraction incubation mixture, it appears that TDCP is directly conjugated with glutathione.

In a study by Nomeir *et al*., 1981 (also described in the *in vivo* section below), TDCP was rapidly metabolised *in vitro* by enzymes in the microsomal and soluble fractions of liver homogenate but not by blood plasma. The results obtained indicate that the microsomal and soluble fractions of rat liver are the location of the enzymatic systems that account for the majority of the metabolism of TDCP by oxidative and conjugating pathways. The small amount of metabolism by the mitochondrial fraction was thought to be due to contamination by the supernatant. NADPH greatly enhanced TDCP metabolism while the mixed function oxidase inhibitor, SKF 525A decreased microsomal metabolism by 83 %, indicating that the mixed function oxidases are involved in metabolism. The addition of GSH to the soluble fraction dramatically increased the metabolism of TDCP. Comparison of metabolic activity of dialyzed and undialyzed fractions in the presence of GSH indicated the presence of an endogenous transferase inhibitor. The metabolites generated by the microsomal fraction were bis(1,3-dichloro-2-propyl) phosphate (BDCP 64 % of total metabolites), 1,3-dichloro-2-propanediol (20 %), 1,3-dichloro-2-propanol (5.7 %) and an unknown metabolite (11 %). It was noted when studying the effects of NADPH concentration on metabolism by the microsomal fraction that as the NADPH concentration increased, the relative amount of 1,3-dichloro-2-propanol and the unknown metabolite decreased while the relative amount of 1,3-dichloro-2-propanediol increased and the amount of bis(1,3-dichloro-2-propyl) phosphate remained unchanged, suggesting that 1,3-dichloro-2-propanol is possibly subject to further metabolism by the microsomal enzymes to 1,3-dichloro-2-propanediol. The lack of further

metabolism of bis(1,3-dichloro-2-propyl) phosphate is thought to be due to the polarity of the acid formed by dealkylation. The polar metabolite would be unavailable for further metabolism as it should partition out of the microsomes and into the aqueous phase. Metabolism of TDCP by the soluble fraction resulted almost exclusively in one metabolite that, the experimental evidence suggests, was a γ-glutamylcysteinyl conjugation product of parent TDCP.

An *in vitro* comparative metabolism study was carried out with TDCP and the structurally similar substances TCPP and TCEP (BASF Aktiengesellschaft, 2007). Two assays were performed: in the first, ^{14}C-TDCP, ^{14}C-TCPP and ^{14}C-TCEP were incubated in rat liver S9 fraction for 4 hours, and in the second, the radiolabelled substances were incubated in rat liver slices for 24 hours. Following incubation, the metabolic profiles of the S9 and liver slice incubates were measured by radio HPLC. Mass spectrometry was performed using HPLC/MS-MS. TDCP was mainly metabolised to a glutathione conjugate and derived metabolites (Gly-Cys-adduct and Cys-adduct) in the liver S9 fraction. 55 % and 87 % of unmetabolised parent compound was detected in the S9 fraction and liver slices, respectively.

Dermal

An *in vitro* percutaneous absorption study (TNO Quality of Life, 2006) conducted to GLP guidelines and to OECD Guideline No. 428, was carried out to determine the rate and extent of absorption following topical application of [^{14}C]-TDCP to human skin for 8 hours. Three dose levels were tested, 0.003, 0.01 and 0.12 mg/cm^2, which corresponded to the typical exposure during manufacture of moulded foam, a logarithmically derived intermediate dose and the reasonable worst case exposure during manufacture of TDCP, respectively.

Human skin membranes, six membranes per dose level, were placed in 9 mm flow-through automated diffusion cells. Receptor fluid was pumped at a speed of ca. 1.6 ml/h. Prior to commencement of the study, the solubility of TDCP in the receptor fluid was determined to be 7.03 µg/ml. The integrity of the skin membranes was evaluated by measuring the permeability coefficient (K_p) for tritiated water and 18 skin membranes with a K_p value below the cut-off value of 3.14 x 10^{-3} cm/h were selected for the study.

The dose solutions were prepared on the day of application. [^{14}C] TDCP was mixed with non-radiolabelled TDCP to obtain a target amount of radioactivity of ca. 1 x 10^6 dpm per skin membrane. For the lowest concentration, ca. 0.5 x 10^6 dpm per membrane was the maximum amount of radioactivity possible. In order to ensure equal distribution over the skin surface, the relevant dose of TDCP was applied in a small volume of acetone (20 µl) which was evaporated directly after application using a warmed air-flow. Receptor fluid samples were collected from 0-1 h and 1-2 h, followed by 2-hour intervals until 24 hours after application. At 8 hours post dose, unabsorbed TDCP was removed from the skin using 3% Teepol solution in water and cotton swabs. The diffusion cell was dismantled at 24 hours post dose and the receptor and donor compartments were washed twice with 1.0 mL ethanol, each skin membrane was tape stripped 10 times and the remaining skin was solubilised. All samples were analysed using liquid scintillation counting. **Table 4.44** below gives a summary of the amount of TDCP found in each sample.

Table 4.44 Summary of percutaneous penetration of TDCP through human skin *in vitro*

	A		B		C	
Concentration measured [mg/ml]	0.092		0.329		3.842	
Dose [µg/cm²]	2.87		10.27		120.06	
n	6		5		6	
Penetration into the receptor fluid after 24 h	% of dose	µg/cm²	% of dose	µg/cm²	% of dose	µg/cm²
	6.10	0.175	3.66	0.376	1.88	2.252
Maximal flux [µg/cm²/h]	0.010		0.023		0.136	
Lag time [h]	6.5		7.8		7.5	
Mean total absorption* [%] (SD)	15.4 (7.5)		10.7 (5.3)		6.0 (3.3)	

* Total absorption is defined as the amount in the receptor fluid, the receptor compartment wash and skin membrane, excluding tape strips.

The mean penetration of TDCP into the receptor fluid after 24 hours was 0.18, 0.38 and 2.25 µg/cm^2, for the low, mid and high dose, respectively. The mean maximal flux was 0.010, 0.023 and 0.136 µg/cm^2/h, for the three doses respectively. The mean total absorption is defined as the compound related radioactivity present in the receptor fluid, the receptor compartment wash and the skin membranes (excluding tape strips). At 0.003 mg/cm^2, the total absorption ranged from 7.0 % to 26.1 %, with a mean absorption of 15.4 %. At the mid dose of 0.01 mg/cm^2, the percentage absorption ranged from 7.2 % to 20.0 %, with a mean absorption of 10.69 %. At the highest dose tested, 0.12 mg/cm^2, the absorption ranged from 3.0 % to 11.9 % and the mean absorption was 6.0 %.

In *in vitro* dermal absorption studies, the amount of penetrated substances found in the receptor fluid are considered to be systemically available. The epidermis (except for the stratum corneum) and the dermis are considered as a sink, and therefore amounts found in these tissues should also be considered absorbed (SCCNFP/0321/00 Final, October 2003). Therefore, a worst case mean total absorption value of 15% has been taken forward to risk characterisation for exposure scenarios where there is potential exposure to "neat" TDCP. This is considered to be a reasonable worst case value since 13 of the 17 individual membrane measurements taken were found to be 15 % or lower.

Two *in vitro* studies were conducted on the structurally similar substance, TCPP: one to determine the rate and extent of absorption following topical application of "neat" TCPP to the skin and the second to determine the percentage of TCPP absorbed across the skin as a result of handling flexible PUR foam (report in HSA/EA, 2008a). The results showed that the percentage absorption from handling foam is approximately twice that obtained following contact of the skin with "neat" TCPP (40 % compared with 23 %). Therefore, taking account of the increased dermal absorption (40 %) observed in the TCPP foam study, a figure of 30% dermal absorption for TDCP, which is twice the absorption value observed in the TDCP "neat" study , will be taken forward to risk characterisation for exposure scenarios 3, 4 and 5, where there is exposure due to handling of foam containing TDCP.

As part of the study reported in the previous section Nomeir *et al*., (1981) investigated the absorption and distribution of TDCP via the dermal route. ^{14}C TDCP was applied dermally in 60µl of a methanol solution (0.867 mg/kg) to a 4 cm^2 area of shaved dorsal skin of male Sprague-Dawley rats. At least 3 rats were used (as stated previously, this is all the information

that was provided regarding the number of animals used). Faeces, urine and expired air were collected.

TDCP was readily absorbed through rat skin (it was not possible to determine the rate of dermal absorption). The resulting distribution pattern showed the greatest concentration in the liver, followed in decreasing concentrations by the lung, skin, blood, kidney, adipose tissue and muscle. Tissue concentrations ranged from 0.10 nmoles TDCP derived radioactivity/g tissue for muscle to 1.38 nmoles TDCP derived radioactivity/g tissue for liver when measured 4 hours after dermal administration.

The excretion of TDCP was measured following i.v. administration.

Studies in humans

No studies are available.

Summary of toxicokinetics, metabolism and distribution

TDCP was well absorbed by the oral route of exposure and based on available studies, 100 % absorption will be assumed. In accordance with the default values given in the TGD, 100 % absorption via the inhalation route will also be assumed. An *in vitro* percutaneous absorption study using human skin membranes was conducted to determine the absorption following topical application of [^{14}C]-TDCP. The skin membranes were exposed to TDCP for 8 hours, mimicking a normal working day. The mean total absorption was 15.4 %, 10.69 % and 6.0 %, for doses 0.003, 0.01 and 0.12 mg/cm^2, respectively. A value of 15 % dermal absorption is taken forward to risk characterisation for exposure scenarios where there is potential exposure to "neat" TDCP and 30 % dermal absorption is assumed for scenarios 3, 4 and 5, where there is exposure due to handling of foam containing TDCP.

Distribution studies showed highest levels in the liver and kidney and lung following oral, dermal and i.v. administration. Tissue concentrations of either the parent compound or metabolites were always low due to rapid excretion. Rapid and extensive (essentially 100 %) oxidative metabolism, mainly to the metabolite bis (1,3-dichloro-2-propyl) phosphate (BDCP almost 70% of metabolites), occurred. Excretion was mainly via the urine (approx 50 %), but also occurred via faeces and expired air.

Elimination was rapid and so no accumulation in the body is expected.

Acute toxicity

Studies in animals

<u>*In vivo* studies</u>

Inhalation

In a GLP study, conducted in accordance with OECD Guideline No. 403 (1981), groups of 5/sex Sprague Dawley rats were exposed, nose only, to a liquid aerosol of TDCP for a period of 4 hours at nominal concentrations of 11.72, 17.54, 23.99 mg/l. Measured gravimetric

concentrations were 2.07, 1.16, and 5.22 mg/l air (Inveresk Research, 1990a). The measured gravimetric concentration is the aerosol concentration to which the animals were exposed. The difference between this and the nominal concentration mainly arises because the calculated nominal concentration includes all of the larger particles at the centre of the aerosol stream which are rarely present in the animals' breathing zone or atmospheric samples measured from this breathing zone. Thus, the difference noted can be explained by the difference between total and measured aerosol mass and is a reflection of the efficiency of generation of the test aerosol. Animals were observed for 14 days. Estimation of particle size distribution revealed that 29.5% of particles were < 3.5 μm for 5.22 mg/l with a MMAD of 4.0 μm. There were no mortalities, no clinical signs of toxicity and no abnormalities detected at necropsy. The LC_{50} was > 5.22 mg/l

In a poorly reported study, 5 rats/sex were exposed for one hour to a nominal concentration of TDCP of 9.8 mg/l (Stauffer Chemical Company, 1974). The actual exposure concentration was not given nor was any information on particle size. There were no mortalities and moderate depression was the only sign of toxicity. The 1-hr LC_{50} was estimated to be > 9.8 mg/l.

Oral

In a GLP study conducted to OECD Guideline No. 401 (1981), doses of 1000, 1710, 2924 and 5000 mg/kg TDCP were administered by gavage to 5 Sprague Dawley rats/sex (Safepharm Laboratories Ltd., 1985a). Observations were made for 14 days.

There were 2 mortalities at 1710 mg/kg (two males on days 1 and 3), 7 mortalities from day 2 (four males on days 1, 2, 3 and 4 and three females on days 1, 2 and 3) at 2924 mg/kg and all animals died at 5000 mg/kg. All animals treated with 1710 mg/kg and greater showed non-specific signs of toxicity, in addition to ptosis, decreased respiratory rate, pallor of the extremities, loss of righting reflex (from 1710 mg/kg) and vocalisation. Abnormalities were observed in the lungs (congested and red), liver (pale/dark/mottled) and stomach (occasional haemorrhage, ulceration). The LD_{50} was calculated as 2236 mg/kg (1651-3029) for males, 2489 mg/kg (1773-3495) for females, giving a combined LD_{50} of 2359 mg/kg (2898-2933).

In a GLP study conducted to OECD Guideline No. 401 (1987), a single dose of 2000 mg/kg of TDCP was administered by gavage to 5 Sprague Dawley rats/sex (Inveresk Research, 1989a). The vehicle was corn oil. Observations were made for 14 days. 2 female animals died on day 4. Clinical signs of toxicity were non-specific and included hypokinesia, piloerection, soiled coat, ataxia, dacryorrhoea, chromodacryorrhoea, rhinorrhoea and salivation. No abnormalities were detected at necropsy. The oral LD_{50} was determined to be greater than 2000 mg/kg. In a range finding study conducted prior to the main study, using two animals per dose level, at 3000, 4000 and 5000 mg/kg all animals died.

A toxicity range-finding study was performed preliminary to a male fertility study in Dutch-belted rabbits (Stauffer Chemical Company, 1982a). Five males per group were dosed by oral gavage with TDCP as supplied (liquid form) at 5000, 7500 and 10000 mg/kg. A control group of 5 rabbits were sham-treated. All 5 rabbits died between days 1 and 6 following administration of 10000 mg/kg, 3 deaths occurred on days 5 and 6 following treatment with 7500 mg/kg and 1 rabbit at 5000 mg/kg died on day 8. Clinical signs observed included ataxia, teeth-grinding, prostration, shallow respiration, laboured respiration, salivation, diarrhoea, head nodding and biting of cage bars. Necropsy findings included red splotchy lungs, congested lungs, pale livers, white foci on the liver, purple spleens, yellow foci on small intestine and pale kidneys. All

survivors appeared normal at necropsy. An LD_{50} was identified as 6800 mg/kg (95% confidence limits = 5615-8234).

Dermal

A single dose of 2000 mg/kg TDCP was applied occluded to the clipped skin of 5 Sprague Dawley rats/sex, for a period of 24 hours in a GLP study conducted to OECD Guideline No. 402 (1987) (Inveresk Research, 1989b). Observations were made for 14 days. There were no mortalities and no clinical signs of toxicity. No abnormalities were detected at necropsy. The dermal LD_{50} was > 2000 mg/kg.

In a poorly reported study, 4 New Zealand white rabbits were exposed for 24 hours to 4640 mg/kg TDCP (Stauffer Chemical Company, 1973). A rubber dental damming sleeve (i.e. occlusive dressing) was used (it was not stated if the site was clipped or not). There were no mortalities and no signs of toxicity. The LD_{50} was estimated to be > 4640 mg/kg.

Intraperitoneal

In a briefly reported study (Soderlund *et al*., 1985), TDCP was administered to a group of 10 Wistar rats for 48 hours. A control group of 10 rats received the vehicle, DMSO, only. Kidney/body weight ratios were significantly increased over the controls; however, TDCP did not cause any signs of nephrotoxicity (no histopathological changes, no increases in plasma urea and creatinine levels) following acute intraperitoneal administration of 500 mg/kg to rats.

Studies in humans

No data are available.

Summary of acute toxicity

An inhalation exposure study yielded an LC_{50} value of >5.22 mg/l indicating that TDCP is of low acute toxicity following inhalation exposure.

Studies in rats indicated that TDCP is of low acute toxicity via the oral and dermal routes of exposure, with LD_{50} values of >2000 mg/kg in both cases.

Irritation

Skin

Studies in animals

In a study, conducted to GLP and to OECD Guideline No. 404 (1981), 0.5 ml of TDCP was applied semi-occluded to the clipped skin of 3 New Zealand white rabbits, for a period of 4 hours (Inveresk Research, 1989c). Observations were made for 72 hours. Well-defined erythema (grade 2) was recorded for 2/3 animals after one hour and persisted to 24 hours in one animal. Grade 1 erythema was noted in the third animal. There was no oedema. All reactions were reversed by 48 hours.

TDCP (0.5 ml) was applied to the intact and abraded skin of 6 New Zealand white rabbits (Stauffer Chemical Company, 1979a). An occlusive dressing was used. The exposure period was 24 hours and the animals were observed for 72 hours. Slight to moderate erythema was seen in all six animals after 24 hours (grade 1-2). This was fully reversed by 72 hours. There was no oedema.

Studies in humans

No data are available.

Eye

Studies in animals

In a GLP study conducted to OECD Guideline No. 405 (1987), 0.1 ml of TDCP was instilled into one eye of each of 3 New Zealand white rabbits (Inveresk Research, 1990b). The eyes were examined at 1, 24, 48 and 72 hours. Slight conjunctival erythema was seen in all animals after one hour. This had reversed by 24 hours. There were no other effects of treatment.

TDCP was non-irritating to rabbit eye when 0.1 ml of the test substance was instilled into one eye each of 9 New Zealand white rabbits (Stauffer Chemical Company, 1979a). In three animals the eye was washed after 20-30 seconds. Observations were carried out at 24, 48, 72 hours, at 4 and at 7 days. There were no effects of treatment.

Studies in humans

No data are available.

Respiratory tract

No studies are available. However, in acute inhalation studies (see section 4.1.2.2.1), there was no evidence of nasal/respiratory irritation effects seen at concentrations up to 5.22 mg/l air for 4 hours.

Summary of irritation

The available data indicate that TDCP produces only minimal dermal and eye irritation in animals following single exposure and any mild effects observed are fully reversible. The lack of any substantial skin or eye irritation and the lack of irritation observed in the acute inhalation studies suggests that TDCP would be unlikely to produce significant respiratory tract irritation.

Corrosivity

It can be concluded from the data presented on skin and eye irritation, that TDCP has no corrosive potential.

Sensitisation

Studies in animals

Skin

In a guinea pig maximisation test conducted in accordance with GLP and OECD Guideline No. 406 (1992), TDCP showed no evidence of dermal sensitisation (CIT, 2001). A group of 20 test animals received an intradermal injection of 25 % TDCP in corn oil and a topical application of 100 % TDCP, preceded by a topical application of 10 % sodium lauryl sulphate (administered on induction day 7). 10 control animals received vehicle only. Subsequent dermal challenge with 100 % TDCP resulted in no signs of erythema or oedema in any of the test or control animals. A positive control study was included using mercaptobenzothiazole, which gave appropriate responses.

Respiratory tract

No studies are available.

Studies in humans

No studies are available

Summary of sensitisation

Evidence from a study in guinea pigs indicates that TDCP does not possess significant skin sensitisation potential. No information is available on the respiratory sensitisation potential of TDCP.

Repeated dose toxicity

Studies in animals

In vivo studies

Oral

In a two-year carcinogenicity study, groups of 60 male and 60 female Sprague Dawley rats were fed diets containing TDCP to achieve dose levels of 0, 5, 20 and 80 mg/kg/day for 24 months (Stauffer Chemical Company, 1981a). 10 animals of each sex were selected for interim sacrifice at 12 months. Animals were routinely observed for morbidity, mortality and clinical signs of toxicity. Body weights and food consumption were measured and blood and urine samples taken periodically from selected animals for haematology, clinical chemistry and urinalysis. Full necropsy was carried out on all animals. Tissues from control and high dose animals were examined microscopically as were gross lesions, tissue masses, liver, kidney and testes of low and mid dose animals.

Mortality rates in all groups were low during the first 12 months of the study with no remarkable difference in incidence between control groups and groups receiving TDCP. Mortality remained low in most groups from 12 through 17 months; however a slight increase in the number of deaths in the high dose males over that in control males was apparent during this interval. After month 17, the mortality rate increased in all groups and remained high until the end of the study (this can be expected in ageing animals). Total mortality in low- and mid-dose males and in all TDCP-treated females was considered comparable to that of the controls. Significantly greater mortality ($p<0.05$) was recorded for high dose males, (38/60 animals died in highest treatment group compared to 26/60 in controls).

There were no apparent treatment-related clinical signs of toxicity. Ophthalmoscopic examination at 18 and/or 24 months revealed an increase in the incidence of sacculations along the course of the retinal arterioles in 4 males and 4 females at 80 mg/kg/day and one mid dose male also. While this lesion was reported to occur occasionally in aged control rats, there may have been an acceleration of this abnormal arteriolar process in TDCP treated rats exposed to 80 mg/kg/day for 24 months. (There was no information provided on the historical incidence of sacculations along the retinal arterioles given in the report).

There was a clear adverse effect on body weights throughout the study. Body weights of high-dose animals were lower than weights of control animals throughout most of the study. In females, statistically significant differences were present at all intervals; in males, differences were statistically significant from week 7 through to termination. The magnitude of the differences increased with time such that, at termination of the study, mean body weights of high-dose groups were >20 % lower than control weights.

Haemoglobin, haematocrit and total erythrocyte values of the high-dose animals were generally lower than values for control animals, frequently to a statistically significant degree (exact values were not provided in report). Differences in males were more pronounced than those in females. Values for low- and mid-dose animals were generally comparable to or slightly lower than control values with only occasional statistically significant differences. Reticulocyte counts were not increased, however.

At 24 months, prothrombin times (PT) and partial thromboplastin times (PTT) were statistically significantly increased in high dose males and PTT were statistically significantly increased in all treated female groups, when compared to control animals. There was no dose-response effect observed in females.

Serum alkaline phosphatase values for high-dose animals were lower than control values at most intervals throughout the study, frequently to a statistically significant degree. Values for low- and mid-dose groups were generally comparable to control values; at 24 months however, values for mid-dose males and females were statistically significantly lower than control values.

A few individual animals in the mid- and high-dose groups exhibited marked elevation in blood urea nitrogen (BUN) values at 18 and 24 months. This was consistent with microscopic evidence of renal pathology in these animals.

Plasma acetylcholinesterase activity, measured at 18 and 24 months was lower in high-dose females than in control females; the difference at 18 months was statistically significant. In males, plasma cholinesterase activity in low- and mid-dose groups was statistically significantly lower than controls at 18 months. Erythrocyte cholinesterase activity, measured at 18 and 24 months, was similar among groups with no dose- or test material-related differences.

Absolute and relative liver weights were increased at both 12 and 24 month sacrifices, in both sexes at 80 mg/kg/day. Some animals in the mid-dose group also showed a significant increase in liver weight, with absolute weights in males and relative weights in females reaching statistical significance. The liver weights of the low-dose animals were comparable to the concurrent control animals. At terminal sacrifice, the absolute liver weights for males were increased by 13 % and 16 % at 20 mg/kg/day and 80 mg/kg/day respectively and for females, the absolute weights were increased by 8 % and 16 %, respectively. The relative liver weights for males were increased by 36 % and 66 % and for females by 20 % and 48 % at 20 mg/kg/day and 80 mg/kg/day respectively.

Macroscopic findings in the liver for animals sacrificed at the termination of the study and which died or were killed *in extremis* after the 12 month interim sacrifice included various discolourations in the mid and high dose males and in all treated females as well as masses/nodules/raised areas in the liver in high dose animals. Microscopically in the liver at 24 months, there was an increase in foci of hepatocellular alteration in both males and females at the highest dose (29/46 and 35/50 respectively vs. 20/45 and 15/49 in controls) and also in sinusoidal dilation (12/46 and 18/50 respectively vs. 4/45 and 7/49 in controls). Histological findings at 12 months were similar to controls.

Absolute and relative kidney weights were also statistically significantly ($p<0.05$) increased at 12 and 24 months in rats at 80 mg/kg/day. In males at terminal sacrifice, absolute kidney weights were increased by 46 % and 53 % over controls at doses of 20 mg/kg/day and 80 mg/kg/day, respectively. In female animals, the corresponding increases were 30 % and 64 %. The relative kidney weights for males were increased by 82 % and 115 % and for females by 38 % and 97 % at 20 and 80mg/kg/day respectively.

In the kidney, macroscopic findings noted in animals examined following scheduled and unscheduled deaths after the 12 month interim sacrifice included various discolourations and pitted surface irregularities in the mid and high dose males and in all treated females. Masses/nodules were also noted in the kidneys of high dose males and in all treated females. Kidney enlargement was observed in mid and high dose males and high dose females and cysts were evident in males at all doses and in mid and high dose females. Microscopically there was an increase in the incidence of hyperplasia of the convoluted tubule epithelium in females at the high dose and in males in all treatment groups when compared to control animals at 24 months. In control males 2/45 animals displayed hyperplasia, compared to 10/49, 28/48, 24/46 males in the low, mid and high dose groups, respectively. The incidence of hyperplasia in females was 1/48, 3/48, and 22/50 for low, mid and high dose animals respectively compared to none in control animals. There was also an increase in chronic nephropathy in males at the mid and high doses and in females at the high dose at 24 months.

Absolute thyroid weight was statistically significantly increased in females at 80 mg/kg/day (increased by 17 %). In addition to these findings, erythroid/myloid hyperplasia of the rib marrow, erythroid/myloid metaplasia of the spleen and hyperplasia of the parathyroid glands were also increased in high-dose animals.

Gross observations in the male reproductive tract noted in the mid and high doses included various discolourations, masses/nodules, enlargement and flaccidity in the testes as well as small seminal vesicles. These observations were made in animals which were killed at 24 months and which died or were killed when moribund after the 12 month interim sacrifice. The corresponding testes weights were not significantly higher than control males.

Histological abnormalities were identified in the testes and seminal vesicles. In the testes, germinal epithelial atrophy with associated oligospermia was noted in controls and all treated groups at both 12 months and 24 months. Accumulation of amorphous eosinophilic material in the tubular lumens, sperm stasis and periarteritis nodosa were observed in all animals at 24 months only. There was a greater occurrence of the effects in treated animals when compared to control animals at 24 months. Decreased secretory product was observed one high dose animal at 12 months and in all treated animals at 24 months (it was observed only in one control animal at 24 months).Atrophy of the seminal vesicles was noted in all treated animals at 24 months, but not in the control animals. Exact details of the findings are provided in **Tables 4.47** and **4.48**, in the reproductive toxicity section of this report. Mid- and high-dose males exhibited a higher incidence of testicular enlargement as compared to control males. The corresponding testes weights were not significantly higher than control males.

In the epididymides, oligospermia was noted in one high dose animal at 12 months and in controls and all treated groups at 24 months, with a greater occurrence in the high dose group. Degenerated seminal product was observed in all animals at 24 months only, with the greatest increase in the high-dose group.

There are limited details available on the numbers of animals affected, severity, etc. in the study report provided. Individual results are not recorded in the report nor are the group values for macroscopic observations.

Regarding the derivation of a N(L)OAEL, a LOAEL of 5 mg/kg/day is chosen and taken forward to risk characterisation. This is based on the hyperplasia of the convoluted tubule epithelium observed in all male animals at 24 months and the effects noted in the testes at this dose level. The hyperplasia is considered to be a pre-neoplastic lesion. It is thought that the pathogenesis of proliferative lesions of renal tubule epithelium proceeds from hyperplasia to adenoma to carcinoma. Renal cortical tumours are observed at 24 months in the mid and high dose groups in this 2-year carcinogenicity study.

A 90-day study to investigate the possible neurotoxicity of TDCP was carried out in hens. (Stauffer Chemical Company, 1979b). Following a range-finding study, doses of 0, 4, 20, and 100 mg/kg/day TDCP were administered to 10/group white leghorn hens by gastric intubation, daily for 90 days. A positive control of TOCP (Tri-o-cresyl phosphate) was used. Birds were observed for toxic effects daily and locomotor impairment three times weekly. Appropriate tissues (brain, spinal cord, sciatic nerves) were excised and preserved for histological examination at death or terminal sacrifice.

There were no mortalities in TDCP-treated birds. Mean body weights were decreased (to 87 %) in the 100 mg/kg/day group from week 7 –13. This was accompanied by signs of decreased activity, with incidence increasing with time. There was no evidence of impaired locomotion in controls or TDCP-treated birds at any dose level. The positive control birds showed symptoms of toxicity and positive signs of locomotor impairment from day 17. Histopathological changes seen in treated birds were the same as those found in controls. Therefore, under these test conditions, there was no evidence of TDCP induced delayed neurotoxicity.

In a very poorly reported study (Stauffer Chemical Company, 1978a), it is not possible to evaluate the information other than to say that it appears that TDCP did not cause *in vitro* inhibition of hen brain neurotoxic esterase measured 24 hours after dosing of hens with the maximum tolerated dose (10,000 mg/kg). TOCP, the positive control, caused 85 % inhibition.

Dermal

No studies are available.

Inhalation

No studies are available.

Studies in humans

A morbidity survey was carried out at a TDCP manufacturing plant (Stauffer Chemical Co., 1981b. This survey serves as an adjunct to the mortality study discussed in section 4.1.2.8 in determining if any adverse health effects were associated with occupational exposure to TDCP. The principal aim of the survey, based on the excess of lung cancer findings from the mortality study, was to determine if there was an excess of respiratory conditions among workers exposed to TDCP. The survey was based on a review and analysis of reports from physical examinations performed on a total of 124 workers. The survey population was defined as all currently employed male, full-time workers who had an occupational health program physical examination during 1981. Groups were divided according to age, with the following grouping applied: 20-29, 30-39, 40-49 and >50. The numbers of exposed: non-exposed workers in each of these groups were 19:8, 48:15, 15:4 and 11:4, respectively. The total number of exposed: non-exposed was 93:31. Full-shift, time weighted average (TWA) breathing zone sampling was conducted during the period December 1978 to May 1979. The report indicates that they were exposed to 'extremely low levels of TDCP in the workplace'; TDCP levels were always near or below the limit of detection (8 ppb). Breathing zone sampling was performed between 1978 and 1979. A 175-item self-administered health questionnaire, a physical examination, a pulmonary function test, a chest x-ray and electrocardiogram and a spectrum of clinical and biochemical analyses were performed on the workers at the plant.

31 % of exposed workers were non-smokers compared to 42 % of non-exposed workers. The exposed workers had lower prevalence rates than non-exposed workers for a history of respiratory conditions. The percentage of workers with impaired pulmonary function as detected by x-ray was one sixth that of non-exposed workers. Therefore, there was no increased risk of adverse respiratory effects from exposure to TDCP. There were no abnormal clinical findings in either group. There was an excess of benign neoplasms, (primarily lipomas), (5.4% Vs 0%), dermatitis (6.5 % Vs 3.2 %) and gynaecomastia (3.3 % Vs 0 %) in exposed workers when compared to non-exposed workers.

The limitations associated with the study design included the fact that the control cohort was approximately one third the size of the exposed cohort whereas equally sized populations would have substantiated the validity of the study outcome. However, the logistics of the plant location and workforce size prevented this. In addition, some of the workers classified as non-exposed may have been exposed prior to 1975 from which time the earliest payroll records are available.

Summary of repeated dose toxicity

In a 2-year carcinogenicity study in which groups of 60 male and 60 female rats were fed diets containing TDCP to achieve dose levels of 0, 5, 20 and 80 mg/kg/day for 24 months,

significantly greater mortality was recorded for high dose males. There was a clear adverse effect on body weight in the 80 mg/kg/day groups throughout the study, with body weights at termination >20 % lower than controls. A significant reduction in red blood cell parameters was noted for high-dose animals. Absolute and relative kidney, liver and thyroid weights were also increased in mid- and high-dose animals.

A LOAEL of 5 mg/kg/day (based on the hyperplasia, considered a pre-neoplastic lesion, observed in the kidneys in all treated groups and the testicular effects observed at this dose) can be derived from this study.

In a 90-day study to investigate the possible neurotoxicity of TDCP in hens, doses of 0. 4, 20 and 100 mg/kg/day TDCP were administered to hens. There were no mortalities in TDCP-treated birds. Under the conditions of the test, there was no evidence of TDCP induced delayed neurotoxicity. In an epidemiology study carried out in a TDCP manufacturing plant as an adjunct to a mortality study, no adverse health effects linked to TDCP exposure were determined.

No data are available on inhalation and dermal repeated dose toxicity.

Overall, a LOAEL of 5 mg/kg/day from the 2-year carcinogenicity study will be taken forward to risk characterisation.

Mutagenicity

Studies *in vitro*

Studies in bacteria

In two plate incorporation mutagenicity tests, TDCP did not produce any increase in the number of revertants (Safepharm Laboratories Ltd., 1984 and 1985b). In both studies, *Salmonella typhimurium* strains TA 1535, TA 100, TA 1537, TA 98, and TA 1538 were tested with concentrations of 20-12500 μg/plate both in the presence and absence of metabolic activation. In the study performed in 1985, the results were confirmed in an independent plate incorporation assay. Appropriate positive controls were used and they produced marked increases in the number of revertants.

TDCP did not produce any increase in the number of revertants when tested in two plate incorporation mutagenicity tests (Stauffer Chemical Company, 1976 and 1977a). *Salmonella typhimurium* strains TA 1535, TA 100, TA 1537, TA 98, and TA 1538 were tested with concentrations of TDCP of 0.001-5.0 μl/plate (equivalent to 1.5-7565 μg/plate), both in the presence and absence of metabolic activation. However, the studies did not meet current regulatory standards, as cells were plated singly rather than in triplicate, and no duplicate experiment was performed.

TDCP was tested for mutagenic potential using a standard plate test without S9 and with both Aroclor-induced rat liver and PB-induced mouse liver S9 (Stauffer Chemical Company, 1983a). *Salmonella typhimurium* TA100 was tested with concentrations of TDCP of 0.98-500 μg/plate. A significant, positive dose-related response was seen at 500 μg/plate, with metabolic activation, using both systems.

TDCP was also tested in a modified quantitative suspension assay in this study. Under these conditions, the number of viable bacteria at the time of selection can be determined and an estimate of the mutant frequency calculated. Also, the response can be related to toxicity, as well as a dose level. *Salmonella typhimurium* TA100 was tested with concentrations of TDCP of 50-10000 μg/plate. A significant increase in revertants (2 to 5 fold increase in mutant frequency over background levels) was found at doses >1000 μg/plate, which was associated with considerable toxicity (<5% survival). The extreme toxicity accompanying the mutagenic response indicates that this effect may not be biologically significant and thus the result cannot be regarded as a true positive.

The results of an interlaboratory comparison of data with respect to the Ames test, conducted on 270 chemicals, were reported by Mortelmans *et al*., (1986). A consistent positive response was obtained in two separate laboratories with TDCP in the presence of metabolic activation, from 333 μg/plate in strains TA 100 and TA 1535, showing dose-relatedness of response. Both used Aroclor-induced rat liver S9 and hamster liver S9 and tested in the dose range 10 – 10000 μg/plate using *S. typhimurium* strains TA 100, 1535, 1537, 97 and 98.

Multiple Ames tests were reported in a publication by Gold, *et al.,* 1978. The purpose of this review was to explore the possible mutagenicity of TDCP, its hydrolysis product 1,3-dichloro-2-propanol and a proposed metabolite of TDCP, 1,3-dichloro-2-propanone. PCB- and PB-induced mouse S9 and PCB-induced rat liver S9 and *S. typhimurium* TA 100 were used. It was reported that TDCP and dichloropropanonol were weakly mutagenic in the presence of PB-induced mouse liver S9; dichloropropanone was strongly positive without activation; and that six independent experiments with TDCP and PB-induced mouse S9 showed a positive response with dose dependency. In addition, a repeatable dose-response was found in two experiments with PCB-induced mouse S9 and in three experiments with PB-induced rat S9. The authors reported additional confirmatory results with three tests using PCB-induced mouse liver S9 and in four tests using PCB-induced guinea pig liver S9.

As part of a study investigating the disposition of TDCP (Lynn *et al.,* 1981, see section 4.1.2.1.2), the mutagenicity of TDCP and its metabolites was investigated using the Salmonella-microsome pour plate assay of Ames et al. TDCP was weakly mutagenic to *S. typhimurium* strain TA 100 in the presence of phenobarbital-induced mouse liver S9 fraction. 1,3-dichloro-2-propanol displayed mutagenic activity in the absence of S9 fraction. TDCP was 3-20 times more potent than 1,3-Dichloro-2-propanol. This indicates the presence of another mutagenic metabolite (unidentified). BDCP and 1,3-dichloro-2-propyl phosphate were not mutagenic.

A report published by Ishidate, M., (1983) looked at the usefulness of combining an Ames test with a test for chromosomal aberrations in screening for possible carcinogenicity of chemicals (industrial, pharmaceutical, agrochemical). The *S. typhimurium* strains TA 92, 1535, 100, 1537 and 98 were used. TDCP was positive at a concentration of 0.5 mg/ml in the Ames test with metabolic activation.

TDCP gave a positive mutagenic response when tested in the *Salmonella typhimurium* strain TA 100 with PB-induced rat liver S9 (Soderlund *et al.,* 1985). The concentrations tested were 50, 100, 250, 500 and 1000 μg/plate with the maximal response obtained at 500 μg/plate. A clear dose response was observed. Cytotoxicity was likely to be responsible for the decrease in the number of revertant colonies noted at the highest dose, 1000 μg/plate. Induction of cytochrome P450 by phenobarbitone increased TDCP mutagenicity by 7-8 fold while inhibitors of cytochrome P450 reduced mutation frequency, as did the addition of glutathione.

Studies in fungi

In a standard Ames test using *Saccharomyces cereviseriae* strain D4, concentrations of TDCP of 0.001-1.0 µl/plate and 0.001-5.0 µl/plate (equivalent to 1.5-1513 µg/plate and 1.5-7565 µg/plate, respectively) did not produce any increase in the number of revertants either in the presence or absence of metabolic activation, respectively (Stauffer Chemical Company, 1976 and 1977a).

Studies in mammalian cells

TDCP was shown to cause mutations in mouse lymphoma L5178Y cells in the presence of S9 activation at doses ≥ 80 µg/ml, which were within the toxic range in a GLP study based on the methods of Clive *et al.* (1972 & 1977) and Amacher *et al.* (1979) (Inveresk Research International, 1985). Two independent assays were carried out. Cells were exposed to a dose range of 1.25–60 µg/ml in the first assay and from 10-120 µg/ml in the second, in the presence or absence of S9 prepared from Sprague Dawley rats induced with Aroclor 1254 in corn oil. Positive controls used were ethylmethanesulfonate (250 µg/ml EMS) and 3-methyl-cholanthrene (2.5 µg/ml 3-MC). Triplicate plates were used to estimate TFT resistant mutants. In the first assay there was a slight increase in mutation frequency from 40 µg/ml (+S9) with relative total growth at the higher doses of 40 and 60 µg/ml at 63 % and 53 % of controls, respectively. In the second assay a clear dose-related increase in mutation frequency was observed in the presence of S9. The increases were statistically significant at 80, 100 and 120 µg/ml. The relative total growth at these concentrations was 36 %, 15 %, and 11 %, respectively, of controls. There was no evidence of mutagenic activity in the absence of S9.

The potential for TDCP to increase the frequency of cells with chromosomal aberrations was investigated in Chinese Hamster Ovary cells (CHO) in a GLP study conducted to OECD Guideline No. 473 (1997) (Covance Laboratories Inc., 2004). Two independent assays were performed both in the presence and absence of S9 prepared from Aroclor 1254-induced rat livers. DMSO was used as a solvent and vehicle control, while the negative controls consisted of cells and culture medium with or without metabolic activation. The positive non-activation control used was mitomycin C and the positive activation control was cyclophosphamide.

In the initial assay, the treatment period was three hours. The concentrations of TDCP tested were in the range 6.78 to 1000 µg/ml with and without S9 fraction. From this range, the following cultures were analysed for chromosomal aberrations: 9.69, 19.8, 28.2 and 57.6 µg/ml without metabolic activation and 28.2, 40.4, 57.6 and 82.4 µg/ml with metabolic activation.

In the confirmatory chromosomal aberration assay, the duration of treatment was 20 hours without S9 fraction and three hours in the presence of S9. Concentrations in the range of 1.25 to 80 µg/ml were tested without S9 fraction and 10 to 150 µg/ml were tested with S9 fraction. Cultures treated with 1.25, 2.5, 5 and 10 µg/ml without S9 and 40, 60, 80 and 100 µg/ml with S9 were chosen for chromosomal analysis. Where possible, the first 100 well-spread metaphases from each culture were analysed for chromosomal aberrations. Approximately 2000 cell nuclei were used to estimate the mitotic index. All experiments were performed in duplicate and the positive and negative controls gave the expected responses.

In the initial experiment, the mitotic index was reduced by treatment with TDCP reaching 71 % of negative control values at 57.6 µg/ml and 54 % at 82.4 µg/ml in the absence and presence of S9 fraction, respectively. In the confirmatory test, reductions in the mitotic index reached 51 % of the negative control at 10 µg/ml without S9 fraction and 58 % at 100 µg/ml

with S9 fraction. These were the maximum dose levels that yielded scorable metaphase chromosomes. In the confirmatory test in the presence of S9 fraction, the percentage of endoreplicated cells was above the historical control value recorded at the highest dose analysed only but was not statistically significant. It was therefore considered not to be of toxicological significance. No significant increase in cells with chromosomal aberrations or polyploidy were observed in either experiment in the absence or presence of metabolic activation.

A study was carried out to evaluate the ability of TDCP to induce point mutations, sister chromatid exchanges and chromosome aberrations in the L5178Y mouse lymphoma cell line (Stauffer Chemical Co., 1977b). TDCP did not induce an increase in specific locus mutations in the L5178Y lymphoma cell line when tested at in three independent assays at concentrations of 0.002 – 0.032 μl/ml (equivalent to 3-48.4 μg/ml), 0.024 – 0.098 μl/ml (equivalent to 36.3-148.3 μg/ml) or at 0.016 – 0.085 μl/ml (equivalent to 24.2-128.6 μg/ml) with or without S9 in each experiment. Positive controls, ethylmethanesulfonate (EMS) and dimethylnitrosamine (DMN), gave appropriate responses.

TDCP gave an overall negative result in the sister chromatid assay when cells were treated at concentrations from 0.004 to 0.072 μl/ml (equivalent to 6-108.9 μg/ml) (+S9 only). Three different S9 fractions were constructed for the assay; from non-induced males; from mice pre-treated with Aroclor 1254; and from mice pretreated with phenobarbitol. While there were a number of increases in SCEs over individual doses, no dose-related increase was apparent.

The results of the chromosomal aberrations (without gaps) were poorly reported. There was a trend towards an increase in the occurrence of chromosomal aberrations at concentrations of 0.004 to 0.072 μl/ml (corresponding to 6-108.9 μg/ml) +S9 only (the % frequencies of chromosome aberrations at 0, 0.004, 0.008, 0.016, 0.032, 0.064 and 0.072 μl/ml (corresponding to 0, 6, 12.1, 24.2, 48.4, 96.8 & 108.9 μg/ml) +S9 induced with Aroclor 1254 were 0, 2, 0, 6, 14, 16 and 41 respectively). This result shows a dose-related trend in the induction of chromosome aberrations. There was no indication in the report of this effect being statistically significant and the historical control values were not recorded. However, the increase in percentage frequency could be considered significant at 0.072 μl/ml (108.9 μg/ml). Significant toxicity was seen from the next dose, 0.085 μl/ml to the highest dose of 0.125 μl/ml (corresponding to 128.6 - 189.1 μg/ml, respectively).

A reported published by Ishidate, M., (1983) looked at the usefulness of combining an Ames test with a test for chromosomal aberrations in screening for possible carcinogenicity of certain chemicals. TDCP (0.5 mg/ml) was positive in the chromosomal aberration test in Chinese hamster fibroblast cells, following treatment for 3 hours in the presence of metabolic activation.

TDCP did not induce sister chromatid exchanges when tested in the dose range 8 – 5000 μg/embryo in the avian embryo system (chick embryo cytogenetic test (CECT) (Bloom, S.E., 1982 & 1984). The author of the report claimed a high positive correlation between SCE induction in the CECT and mutagenic potency.

TDCP did not cause malignant transformation of BALB/3T3 cells *in vitro* when tested at concentrations of 0.02 to 0.312 μl/ml (corresponding to 30.3 - 472.1 μg/ml) in 10 replicate plates /dose (Stauffer Chemical Co., 1978b). The positive control was 3-MC (10 μg/ml). Only doses from 0.078 μl/ml (equivalent to 118 μg/ml) were used as the lower dose groups were contaminated. The positive control response was considered appropriate.

In a briefly reported study, TDCP was negative when tested in a V79 Chinese Hamster Lung cell point mutation assay when tested at a concentration of 0.02 mM (Soderlund *et al*., 1985).

In the above briefly reported study of Soderlund *et al*., 1985, TDCP was tested at concentrations of 0.025, 0.05 and 0.1 mM and caused a minimal response at 0.1 mM with hepatocytes from non-induced rat livers in an unscheduled DNA synthesis assay (it was not possible to quantify the response from the information given in the study).

In the same study, TDCP was tested at concentrations of 10, 20 and 30 μM and was found to increase transformation frequencies in the Syrian hamster embryo cells at 20 and 30 μM.

The potential mutagenicity of TDCP was also investigated in the study of Soderlund *et al*., 1985 in the *Salmonella typhimurium*/hepatocyte assay using liver cells isolated from either untreated or PB-induced rats. TDCP was tested at concentrations of 0.025, 0.05 and 0.1 mM. A small increase in revertants was seen with TDCP (0.05 mM) using hepatocytes from non-induced rat livers and no increase was seen using PB-induced hepatocytes.

Studies *in vivo*

TDCP was tested in a mouse micronucleus test (Safepharm Laboratories Ltd., 1985c) in a GLP study adhering to OECD Guideline No. 474 (1983). 5/sex/group CFLP mice received a dose of 2000 mg/kg TDCP in arachis oil once by gavage. This was based on the outcome of a preliminary range-finding experiment. 3 kill times of 24, 48 and 72 hours were used. The positive control was 50 mg/kg bw cyclophosphamide with a single sampling time of 24 hours. A second (verification) study was also conducted using a single time point of 24 hours and dose levels of 200, 630 and 2000 mg/kg. The incidence of micronucleated cells per 1000 polychromatic erythrocytes per animal was scored. In addition, the number of normochromatic erythrocytes associated with 1000 polychromatic erythrocytes were counted; these cells were also scored for incidence of micronuclei. The occurrence of micronucleated polychromatic erythrocytes (PCE's) and the PCE/NCE ratios were examined in bone marrow preparations of 5/sex/time point per dose in both studies. There was no statistically significant increase in micronucleated polychromatic erythrocytes or micronucleated normochromatic erythrocytes. There were no decreases in the PCE/NCE ratio at any dose level. The positive control gave an appropriate response.

TDCP was negative in this *in vivo* micronucleus assay under these assay conditions. There was no alteration in the PCE:NCE ratio even at the highest dose tested, therefore it was not demonstrated that the test substance reached the bone marrow. However, systemic toxicity was observed in the 2 year carcinogenicity study (described in section 4.1.2.8), demonstrating that the substance was absorbed, which could possibly be considered concordant with exposure of the target organ i.e. bone marrow.

TDCP was not clastogenic in a mouse bone marrow cytogenetic assay at doses of 0.5 ml/kg, 0.17 ml/kg and 0.05 ml/kg (Stauffer Chemical Co., 1978c). These doses correspond to 756.5 mg/kg, 257.2 mg/kg and 75.65 mg/kg respectively. Both an acute and sub-chronic test was carried out. In the acute test, 8 mice /timepoint were dosed with TDCP and sacrificed at 6 hr, 24 hr and 48 hr post-dosing. In the sub-chronic test, 8 animals were dosed each day for 5 days and sacrificed 6 hours after the final dose. 50 metaphases were scored per animal dosed. There were no significant increases at any dose level or kill time.

In a single, poorly reported, non-standard test, CD-1 mice were treated with TDCP at concentrations of 0.05 to 0.5 ml/kg/day (corresponding to 75.65, 257.2 and 756.5 mg/kg) for

5 days after which time the urine was tested for the ability to induce an increase in revertants in TA strains 1535, TA 1537, TA 98 and TA 100 (Stauffer Chemical Company, 1978d). Both deconjugated and non-deconjugated urine samples were tested. The test gave a negative result. A single plate was used for each data point and no positive control data were included.

In an *in vivo*/*in vitro* unscheduled DNA synthesis (UDS) assay in primary rat hepatocyte cultures, TDCP showed no evidence of UDS and, was therefore, considered negative under the conditions of the test (Covance Laboratories Inc., 2005). The assay was carried out to GLP and in accordance with OECD Guideline No. 486 and EC Method B.39. A preliminary range finding study was performed by dosing three rats/sex/dose group by oral gavage at doses of 125, 250, 500, 1000 and 2000 mg/kg. Mortality was induced in one female dosed at 2000 mg/kg on day 2 post dosing and clinical signs at this dose included red perioral crust, hypoactivity, squinted eyes, recumbency and/or irregular respiration.

In the main study, TDCP was administered by oral gavage to four to six male Hsd:SD rats per dose group per sacrifice time at doses of 500, 1000 and 2000 mg/kg in 0.5 % methylcellulose (MC). Two sacrifice time points were employed: 2 to 4 hours and 14 to 16 hours following dosing. Vehicle control (MC) animals and positive control (dimethylnitrosamine) animals were treated concurrently by oral gavage and IP injection, respectively. During animal observations, oral discharge was noted in two animals at 500 and 1000 mg/kg at the 2 to 4 hour time point and hypoactivity, anal discharge, nasal crust and red urethral discharge were noted in animals at the 14 to 16 hour time point.

Hepatocytes were harvested following perfusion of the livers of four rats from each group at the two selected time points. Hepatocyte cultures were established from two or three animals per group and, following an attachment period of up to two hours, three replicate cultures from each animal were labelled with 10 μCi/ml ^{3}H-TdR for four hours. The labelled cultures were analysed for nuclear labelling by autoradiography following washing out the unincorporated label and a further incubation period.

The mean net nuclear grain counts in the vehicle controls were -0.21 and -0.38 and the average percent of cells containing five or more net nuclear grains were 0.22 % and 0.00 %, for the 2 to 4 hour and the 14 to 16 hour timepoints, respectively. None of the treated groups yielded a positive mean net nuclear grain count. The highest percent cells with ≥ 5 grains in the treatment groups were 0.67 % and 0.39 % for the 2 to 4 hour and 14 to 16 hour timepoints, respectively. Therefore, none of the treatment groups exceeded the criteria for a positive response at either the 2 to 4 or the 14 to 16 hour time points. The vehicle and positive controls yielded acceptable results

Studies in Drosophila

In a sex-linked recessive lethal assay in Drosophila (Stauffer Chemical Co., 1978e), 25 male flies were fed a 1 or 3 % aqueous sucrose solution containing 2.5 or 25 % TDCP for a period of 24 hours. Males were then mated with females. Two stages of the spermatogenic cycle were examined; mating I examined sperm and spermatids and mating III examined early spermatids and spermatocytes exposed to Fyrol FR-2. No increase in sex-linked recessive lethal mutations was observed. For insect systems such as this, there is too little comparative data with mammalian cells and the relevance of findings to the mammalian *in vivo* systems is uncertain.

Summary of mutagenicity

Table 4.45 below summarises the results from the *in vitro* and *in vivo* mutagenicity tests.

Table 4.45 Summary of mutagenicity data for TDCP

Test	Endpoint	Result	Comments	Ref.
In vitro plate incorporation assay, bacteria (Ames)	Gene mutation	Non-mutagenic		SafePharm Labs ('84&85b)
In vitro plate incorporation assay, bacteria (Ames)	Gene mutation	Non-mutagenic	Studies did not meet current regulatory stds	Stauffer Chem. Co. ('76 &'77a)
In vitro plate incorporation assay, bacteria (Ames)	Gene mutation	Significant positive response at 500 μg/plate +S9 (TA 100)		Stauffer Chem. Co. ('83a)
Ames modified quantitative suspension assay	Gene mutation	Mutagenic only at toxic doses (>1000μg/plate (+&-S9)	Not a true positive response	Stauffer Chem. Co. ('83a)
Ames assays	Gene mutation	Positive response +S9 in strains TA 100 & 1535 from 333 μg/plate.	Dose-related response (Interlaboratory comparison)	Mortelmans *et al*. ('86)
Ames assays	Gene mutation	Weakly mutagenic +S9 with TA 100. Positive in 6 independent expts + PB-induced S9. Positive in 2 expts + PCB-induced S9 and in 3 expts +PB-induced S9. Confirmatory results with PCB-induced mouse & guinea pig liver S9.	Dose dependency observed in multiple assays	Gold *et al.* ('78)
Ames (Pour plate assay)	Gene mutation	Weakly mutagenic + S9 with TA 100.		Lynn *et al*. ('81)
Ames assay	Gene mutation	Positive at 0.5mg/ml +S9.		Ishidate ('83)
In vitro plate incorporation assay, bacteria (Ames)	Gene mutation	Positive mutagenic response +S9 with TA 100 at 500 μg/plate		Soderland *et al*. ('85)
In vitro plate incorporation assay, fungi (Ames)	Gene mutation	Non-mutagenic in *Sacc. cereviseriae*		Stauffer Chem. Co. ('76 &'77a)
In vitro mouse lymphoma assay	Gene mutation	Positive +S9 at >80μg/ml. Non-mutagenic -S9.	Clear dose-related increase	Inveresk ('81)
In vitro chromosome aberration assay	Chromosome aberration	Negative with or without S9		Covance (2004)
In vitro mouse lymphoma assay	Gene mutation	Negative with or without S9		Stauffer Chem. Co. ('77b)
Sister chromatid exchange assay (L5178Y $TK^{+/-}$ cells)	SCE	Negative		Stauffer Chem. Co. ('77b)

Test	Endpoint	Result	Comments	Ref.
Chromosome aberration assay (L5178Y TK$^{+/-}$ cells)	Chromosome aberration	Increase at highest dose analysed (118 µg/ml) +S9.	Considered equivocal.	Stauffer Chem. Co. ('77b)
Combined Ames/chromosomal aberration assay	Gene mutation / chromosome aberration	Positive +S9 at 0.5 mg/ml		Ishidate ('83)
Sister chromatid exchange (CECT assay)	SCE	Negative		Bloom ('82 & '84)
In vitro transformed foci in BALB/3T3 cells	Cell transformation	Negative		Stauffer Chem Co. ('78b)
In vitro point mutation assay in V79 cells	Gene mutation	Negative		Soderland *et al*. ('85)
In vitro UDS assay	DNA damage & repair	Minimal response at 0.1mM	Not possible to quantify response	Soderland *et al*. ('85)
In vitro transformation assay in Syrian hamster embryo cells	Cell transformation	Positive at 20 & 30µM		Soderland *et al*. ('85)
In vitro Salm. typhimurium mutagenicity assay with hepatocyte activation	Gene mutation	Small increase in revertants at 0.05 mM (non-induced rat livers). No increase using PB-induced hepatocytes		Soderland *et al*. ('85)
In vivo Mouse micronucleus assay	Chromosome aberration	Non-clastogenic		SafePharm Labs Ltd. ('85)
In vivo Mouse micronucleus assay	Chromosome aberration	Non-clastogenic		Stauffer Chem Co. ('78c)
In vivo/in vitro urine mutagenicity assay	Mutation	Negative		Stauffer Chem Co. ('78d)
In vivo/in vitro unscheduled DNA synthesis assay	DNA damage & repair	Negative		Covance Laboratories Inc. (2005)
Recessive lethal mutation assay in Drosophilia	Chromosomal mutation	Negative		Stauffer Chem Co. ('78e)

Evidence was provided to suggest that TDCP is a bacterial cell mutagen *in vitro*. Positive responses were seen in the Ames mutation assay, following metabolic activation only. Some papers reviewed included positive data for possible metabolites of TDCP. In mammalian cell studies, TDCP caused mutations in mouse lymphoma L5178Y cells in the presence of metabolic activation. TDCP also caused an increase in the occurrence of chromosomal aberrations in mouse lymphoma cells, again in the presence of metabolic activation. However, in a chromosomal aberration study in CHO cells, no increase in cells with chromosome aberrations or polyploidy were recorded.

In vivo, TDCP was not clastogenic in a mouse micronucleus assay and was found not to induce unscheduled DNA synthesis in studies conducted to OECD guidelines. Negative results were also obtained in a second *in vivo* micronucleus assay and in an *in vivo*/*in vitro* urine mutagenicity assay.

In summary, there is some evidence to suggest that TDCP is mutagenic *in vitro*. However, *in vivo*, the mouse micronucleus assays were negative, results which were further supported by a negative result in an *in vivo*/*in vitro* unscheduled DNA synthesis assay.

Carcinogenicity

Studies in animals

In vivo studies

Inhalation

No studies are available

Dermal

No studies are available

Oral

Groups of 60 male and 60 female Sprague Dawley rats were fed diets containing TDCP to achieve dose levels of 0, 5, 20 and 80 mg/kg/day of the test substance for 24 months (Stauffer Chemical Company, 1981a; Freudenthal, R.I. and Henrich, R.T., 2000). 10 animals of each sex were selected for interim sacrifice at 12 months. Animals were routinely observed for morbidity, mortality and clinical signs of toxicity. Body weights and food consumption were measured and blood and urine samples taken periodically from selected animals for haematology, clinical chemistry and urinalysis. Full necropsy was carried out on all animals. Tissues from control and high dose animals were examined microscopically as were gross lesions, tissue masses, liver, kidney and testes of low and mid dose animals.

Mortality rates in all groups were low during the first 12 months of the study with no remarkable difference in incidence between control groups and groups receiving TDCP. Mortality remained low in most groups from 12 through 17 months; however a slight increase in the number of deaths in the high dose males over that in control males was apparent during this interval. After month 17, the mortality rate increased in all groups and remained high until the end of the study (this can be expected in ageing animals). Total mortality in low- and mid-dose males and in all TDCP-treated females was considered comparable to that of the controls. Significantly greater mortality ($p<0.05$) was recorded for high dose males, (38/60 animals died in highest treatment group compared to 26/60 in controls).

There was a clear adverse effect on body weight in the 80 mg/kg/day groups, throughout the study, with body weights at termination >20 % lower than control. Slight decreases (most differences did not exceed 5 %) in male body weights in the 20 mg/kg/day at some intervals of the study may also have related to treatment. Food consumption for controls and high dose animals was generally comparable except for slight increases in values for the high dose groups during the last few months of the study.

Examination of the tissues from the 12-month interim group and those animals found dead prior to 12 months found an increased incidence of neoplastic nodules in the livers of rats in the high-dose group, which were identified as hepatocellular adenomas. There was also an

increase in interstitial cell tumours in the testes of males at the mid and high doses. The incidence of neoplasms in all other tissues was similar in control and treated animals at this time.

An evaluation of the tissues from all remaining study animals at the conclusion of the 24-month exposure period revealed a significant increase in the incidence of renal cortical tumours and testicular interstitial cell tumours in the mid and high dose animals, and hepatocellular adenomas and adrenal cortical adenomas in the high dose animals (see **Table 4.46** below for tumour incidences).

Table 4.46 Tumour incidence in Sprague Dawley rats fed TDCP in a 2 year bioassay

			Dose group (mg/kg/day)			
Organ	**Tumour identification**	**Sex**	**0**	**5**	**20**	**80**
Kidney	Renal cortical adenoma					
	12 months[a]:	M	0/15	0/12	0/13	0/13
		F	0/11	0/13	0/9	0/10
	24 months only:	M	1/45	3/49	9/48*	32/46*
		F	0/49	1/48	8/48*	29/50*
Testes	Interstitial cell tumour					
	12 months[a]:	M	0/14	0/12	3/13	3/11
	24 months:	M	7/43	8/48	23/47*	36/45*
Liver	Hepatocellular adenomas					
	12months[a]:	M	0/15	0/12	0/13	3/14
		F	0/11	0/13	0/9	1/10
	24 months:	M	2/45	7/48	1/48	13/46*
		F	1/49	1/47	4/46	8/50*
	Hepatocellular carcinomas					
	12months[a]:	M	0/15	0/12	0/13	0/14
		F	0/11	0/13	0/9	0/10
	24 months only:	M	1/45	2/48	3/48	7/46
		F	0/49	2/47	2/46	4/50
Adrenal	Cortical adenomas					
	12 months[a]:	M	0/15	#	#	2/13
		F	5/11	#	#	1/10
	24 months:	M	5/44	3/14	5/16	3/44
		F	8/48	5/27	2/33	19/49*

* Statistical significance (p<0.05)

[a] Scheduled deaths at 12 months and unscheduled deaths prior to 12 months. The report states the numbers of animals "suitable for evaluation" for each tissue listed, although in some cases the results of actual incidence in these tissues is not reported. It has therefore been assumed, that where tissues were suitable for evaluation but no results for 12 month assessment for that lesion are reported, that no lesion was present.

animals not evaluated at 12 months

In the kidney, the incidence of renal cortical adenomas at 24 months in males was 1/45 (2 %), 3/49 (6 %), 9/48 (19 %) and 32/46 (70 %) in control, low, mid and high dose animals respectively (reaching statistical significance from the mid dose group, 20 mg/kg/day). At 24 months in females, the corresponding percentages were 0 %, 2 %, 17 % and 58 %, respectively, with statistical significance again from 20 mg/kg/day. There was no reported incidence at 12 months. In addition to the tumours, there was an increase in the incidence of hyperplasia of the convoluted tubule epithelium at 24 months in females at the high dose and in males in all treatment groups when compared to control animals.

Histological abnormalities were identified at a higher incidence in the livers of treated rats, as described in the repeated dose toxicity section, 4.1.2.6. This was associated with evidence of neoplastic alterations. In the livers of male animals at 24 months, the incidence of hepatocellular adenomas was 2/45 (4 %), 7/48 (14.5 %), 1/48 (2 %) and 13/46 (28 %) in control, low-, mid- and high-dose animals respectively, with statistical significance reached at the highest dose. In females, the corresponding percentages were 2 %, 2 %, 9 % and 16 %, respectively, with statistical significance again at the highest dose. At the 12 month interim sacrifice, the incidence of hepatocellular adenomas was 3/14 and 1/10 for males and females respectively at the highest dose only compared to none in control animals.

At 24 months, the incidence of hepatocellular carcinoma was also increased in males and females, with the incidence in males being 1/45 (2 %), 2/48 (4 %), 3/48 (6 %) and 7/46 (15 %) in control, low, mid- and high dose animals respectively, although this did not reach statistical significance. The corresponding values in females were 0/49, 2/47 (4 %), 2/46 (4 %) and 4/50 (8 %). There was no reported incidence at 12 months.

The incidence of interstitial cell tumours of the testes (benign tumours) at 24 months was 7/43 (16 %), 8/48 (17 %), 23/47 (49 %) and 36/45 (80 %) in the control, low-dose, mid-dose and high-dose animals, respectively. The effects were statistically significant in the mid-dose and high dose groups. At 12 months, 3/13 mid dose animals and 3/11 high dose animals were observed to have interstitial cell tumours; no tumours were observed in control animals at 12 months.

There was also an increased incidence of adrenal cortical adenomas in high dose females. The incidence of this finding at 24 months was 8/48 (17 %) in control females and 19/49 (39 %) in high-dose females; the difference being statistically significant. At 12 months the incidence was in females was 5/11 (45 %) and 1/10 (10 %) for control and high dose groups, respectively.

Studies in humans

The mortality experience of workers employed at a TDCP manufacturing plant was investigated in a retrospective cohort study (Stauffer Chemical Co., 1983b). The study recruited all male workers who were employed for a minimum of 3 months during the 1956-77 study period and were followed through to 1980. This included active, terminated, retired and deceased employees. Of the 289 workers eligible for the study, 50% had worked at the plant for < 5 years while 42 workers had been employed for ≥ 15 years. The age of the workers was not provided. The vital status (living or deceased) of each individual in the cohort as of December 31, 1980, was sought. Ascertainment of vital status was 100 % complete. For those individuals known to be deceased, certified copies of death certificates were obtained from various state health departments.

Ten workers died during the study period. The report indicates that all workers were exposed to 'extremely low levels of TDCP in the work environment'. Breathing zone sampling was performed between 1978 and 1981; TDCP levels were always below the limit of detection (8 ppb).

The overall mortality of the study population was 75 % of that expected in a comparable population of US males. For the category 'all causes', the SMR (observed deaths/expected deaths x 100) was 75. Mortality due to 'all malignant neoplasms' was slightly higher than expected with an SMR of 131. Three cases of lung cancer were observed (vs 0.8 expected). However, the numbers were too small to calculate a p-value. One case had worked as a janitor in the plane office and was considered non-exposed. The second case had only worked at the plant 2 years prior to onset of disease and the third case had worked for 19 years, as a production operator and a mechanic. All three decedents were moderate to heavy cigarette smokers. Overall, it was concluded that there was no evidence linking these lung cancers with TDCP exposure. This was the only elevated cancer observed. Due to the findings of liver, kidney and testicular tumours in the 2-year carcinogenicity study in rats, this study also aimed to determine whether tumours would also occur in humans at these sites. No cancers at these sites were observed. However, this was a very small study and thus one cannot place much reliance on the negative result.

Summary of carcinogenicity

In a 2-year carcinogenicity study, in which groups of 60 male and 60 females rats were fed diets containing TDCP to achieve dose levels of 0, 5, 20 and 80 mg/kg/day, there was a significant increase in the incidence of renal cortical adenomas in mid and high dose animals at 24 months. There was no increase at 12 months. The incidence of benign testicular interstitial cell tumours was also increased in the mid- and high-dose animals at both 12 and 24 months. Hepatocellular adenomas and adrenal cortical adenomas were statistically increased in the high dose animals at 24 months.

In the testes, there was an increased incidence of Leydig cell tumours in the mid and high dose males at both 12 and 24 months. The mechanism by which TDCP induces such tumours is not known. It is reported that one non-genotoxic mode of action by which chemicals can induce such tumours is attributed to alterations in the Hypothalmus-Pituitary-Testis (HPT) Axis which results in elevated levels of luteinising hormone (LH). Increases in LH levels have been shown to be necessary for the induction of Leydig cell tumours through chronic stimulation of the Leydig cells. There are seven known non-genotoxic hormonal mechanisms which have the potential to disrupt the HPT axis leading to Leydig cell tumour induction. Two of these modes of action are not considered of relevance to humans (GnRH antagonism and dopamine agonism) (Clegg *et al.,* 1997). However, the other five mechanisms, (5 α-reductase inhibition, androgen receptor antagonism, inhibition of testosterone biosynthesis, aromatase inhibition and exogenous oestrogen agonism) have been considered to be potentially relevant to humans.

Overall, while the mode of action by which these tumours are induced cannot be identified, there may be some concern for man regarding their formation.

Regarding the derivation of a N(L)OAEL for carcinogenicity to take forward to risk characterisation, this is taken as a LOAEL at 5 mg/kg/day. This is based on the hyperplasia of the convoluted tubule epithelium with increased incidences observed in all treated male animals and in high dose females at 24 months (as outlined in the repeated dose toxicity

section 4.1.2.6.1). Hyperplasia was observed from the lowest dose tested. Hyperplasia is often considered as a pre-neoplastic lesion, which can lead to tumour formation. The study report does not provide enough detailed information to conclude whether the hyperplasia observed following treatment with TDCP would progress to cancer or whether the tumours observed with TDCP arise through a different mechanism. However, it is not unreasonable to assume that the tumours have developed through hyperplastic changes.

There is some evidence to suggest that TDCP is mutagenic *in vitro.* However, *in vivo* mutagenicity studies were negative, indicating that, *in vivo,* TDCP is non-genotoxic. This indicates that TDCP may be assumed to be a threshold carcinogen.

TDCP is classified as Carc. Cat. 3 R40 "Limited evidence of a carcinogenic effect" based on the results of the above carcinogenicity study further supported by a non-genotoxic mode of action for carcinogenic effects for TDCP[15].

In a study carried out to look at the mortality experience of worker in a TDCP manufacturing plant, there was a higher than expected number of lung cancers among male workers. However, the report concluded that there was no evidence linking these lung cancers with exposure to TDCP. There were no other cancers observed.

Toxicity for reproduction

Effects on fertility

Studies in animals

A fertility study in male rabbits was carried out using 40 male and 80 female Dutch belted rabbits (Stauffer Chemical Company, 1982b). Male animals were assigned to the study on the basis of their weight, general health and reproductive performance in a pre-dose fertility test. Ten male rabbits were assigned to each of four dose groups and treated with 2, 20, or 200 mg/kg/day TDCP in mazola oil for twelve weeks by oral gavage. Animals were examined throughout the treatment period for signs of treatment-related toxicity. During the last week of treatment, each male was mated with the two females it was paired with during the pretreatment fertility test. Each was mated with one female and then with the second three days later. The females were returned to their cages and sacrificed mid-gestation. The reproductive tract was removed and examined to determine the number of corpora lutea in each ovary, the number of implantation sites and viable foetuses. The reproductive tract (testes, epididymides, spermatic cord with blood and lymphatic vessels and ductus deferens, ampullary gland, vesicular gland, seminal vesicle, prostate gland, paraprostatic gland, urinary bladder, urethra, and bulbo-urethral glands) was removed for histological examination. Sperm were taken from one epididymus and analysed for sperm concentration, motility and morphology. Viability was not measured due to the subjectivity in sample readings.

Mating, fertility and pregnancy parameters were unaffected by treatment. There were no treatment-related effects on numbers of corpora lutea, implantations, viable foetuses or resorptions. Sperm analysis was not affected by treatment. There were no histopathological changes detected in the male reproductive tract.

[15] Commission Working Group on the Classification and Labelling of Dangerous Substances Meeting on the Health Effects of Pesticides, Existing Chemicals & New Chemicals November 14-18, 2005.

Blood samples were taken from the males for haematological and clinical chemical analysis. Males were sacrificed at the end of the mating period and certain organs weighed (pituitary, liver and kidneys). Two animals in each of the control, 2 mg/kg/day, and 20 mg/kg/day groups and one in the 200 mg/kg/day died prior to scheduled sacrifice. These deaths were not considered treatment-related. There were no clinical signs of toxicity. There was a treatment-related increase in absolute and relative kidney (14 and 19 %, respectively) and liver weights (18 and 23 %, respectively), in the 200 mg/kg/day males. Overall, it is considered that there is no concern for male fertility in the rabbit.

In the 2-year carcinogenicity study (Stauffer Chemical Company, 1981a; and also reported in Freudenthal, R.I. and Henrich, R.T., 2000), as described previously (sections 4.1.2.6.1 & 4.1.2.8.1), effects were observed on the reproductive system of the male rat. As stated in the carcinogenicity section, all of the information from this study was not available to the Rapporteur, the reporting is somewhat limited. All available information is reported here.

For some effects, only control and high dose animals were evaluated at 12 months; all animals in the control and treatment groups were evaluated at 24 months.

In animals which were killed at 24 months and which died or were killed when moribund after the 12 month interim sacrifice, gross observations noted in the male reproductive tract in the mid and high doses included various discolourations, masses/nodules, enlargement and flaccidity in the testes as well as small seminal vesicles. The corresponding testes weights were not significantly higher than control males.

Histological changes were also noted in the testes, the epididymides and the seminal vesicles both in control animals and all treatment groups.

In the testes, germinal epithelial atrophy with associated oligospermia was noted in controls and all treated groups at both 12 months and 24 months. At 12 months, there was an increase above control values for the high dose group (statistical analysis was not performed on this data) and at 24 months there was an increase above control values in the mid and high dose animals. The incidence of sperm stasis was increased above control values (approx 11 %) at the mid and high doses (approx 23 % and 31 %, respectively) at 24 months. Again, statistical analysis was not performed on this data. There was also an increase in the incidence of amorphous eosinophilic material in the tubular lumens and periarteritis nodosa were observed in all treated animals at 24 months. These effects on sperm stasis, the incidence of amorphous eosinophilic material and periarteritis nodosa in the testes were only reported to be observed at 24 months. The report indicated that the testes were “suitable for evaluation” at 12 months, although no result was presented in the report for this time point, so it can only be assumed that the testis were evaluated for these effects at 12 months, and that no effects were observed.

In the epididymides, oligospermia was noted in one high dose animal at 12 months. There was none noted in any control animals and the epididymides from the low and mid dose animals were not evaluated, apart from one unscheduled mid dose animal. Oligospermia was also noted in controls and all treated groups at 24 months, with a greater occurrence in the high dose group. 26 % of the control group showed oligospermia at 24 months, with 28 %, 54 % and 79 % displaying it at the low, mid and high doses, respectively. Degenerated seminal product was observed in all animals at 24 months (this was not examined in the low and mid doses at 12 months; it can only be presumed that it was examined at the high dose at 12 months, and did not occur), with the greatest increase in the high-dose group. 19 % of the control group showed degenerated seminal product, with 22 %, 23 % and 50 % displaying it at the low, mid and high doses, respectively.

In the seminal vesicles, secretory product was decreased in the seminal vesicles of one high dose animal at 12 months (not noted in any control animals and the effect was not examined in the low and mid doses at 12 months) and in all treated animals at 24 months. At 24 months, 2 % of control animals displayed decreased secretory product, compared with 84 %, 89 % and 52 % of the low, mid and high dose animals, respectively. Atrophy of the seminal vesicles was observed in all treated animals at 24 months (30 %, 31 % and 23 % of the low, mid and high dose animals, respectively), but not in any of the control animals. Only the control and high dose12 month animals were examined for atrophy of the seminal vesicles; no indication was given on an effect observed in the high dose animals. **Table 4.47**, below details the histopathological findings.

Table 4.47 Histopathological observations in the male reproductive organs in Sprague Dawley Rats fed TDCP in a 2 year Bioassay (Stauffer Chemical Company, 1981a)

Tissue/	**Males**			
mg/kg/day b.w.	**0**	**5**	**20**	**80**
Testes:				
Seminiferous tubules – germinal epithelial atrophy with associated Oligospermia				
12 months	5/14	2/12	3/13	7/11
24 months	30/43	29/48	42/47	44/45
Tubular lumens – Amorphous Eosinophilic material:				
24 months only	2/43	4/48	12/47	11/45
Sperm stasis:				
24 months only	5/43	5/48	11/47	14/45
Periarteritis nodosa:				
24 months only	5/43	10/48	19/47	16/45
Epididymes:				
Oligospermia:				
12 months	0/14	#	#	1/11
24 months	11/41	9/32	7/13	35/44
Degenerated seminal product:				
24 months only	8/41	7/32	3/13	22/44
Seminal vesicle:				
Decreased secretory product:				
12 months	0/15	#	#	1/10
24 months	1/41	11/13	17/19	22/42
Atrophy:				
24 months only	0/41	4/13	6/19	10/42

Animals not evaluated at 12 months

Statistical analysis was not performed to analyse the data in this study (Stauffer Chemical Company, 1981a). Therefore, statistical significance for the effects in the male reproductive

tract is not available. Statistical analysis was provided, however, for these effects as recorded in the National Academy of Sciences paper, which reported the same data as from the Stauffer study (The National Academy of Sciences, 2000). The values for the effects as reported in this paper do not match exactly with the values recorded in the original Stauffer study report and, therefore, it may not be appropriate to assume that the same statistical significance applies to the data in the National Academy of Sciences paper as to the data in the Stauffer study report, as supplied to the Rapporteur. However, the data provided in the National Academy of Sciences paper is given here in **Table 4.48** for reference.

Table 4.48 Combined 12 and 24 month histopathological Observations in the Reproductive Organs in Sprague Dawley Rats fed TDCP in a 2 year Bioassay (The National Academy of Sciences, 2000)

Tissue/	**Males**			
mg/kg/day b.w.	**0**	**5**	**20**	**80**
Testes:				
Oligospermia	35/57	31/60	45/60	51/56
Eosinophilic material/lumen	2/57	4/60	12/60*	11/56*
Sperm stasis	5/57	5/60	11/60	14/56
Periarteritis nodosa	5/57	10/60	19/60*	16/56*
Epididymes:				
Oligospermia	11/55	9/33	7/14	36/55*
Degenerated seminal product	8/55	7/33	3/14	22/55*
Seminal vesicle:				
Decreased secretory product	1/56	11/13*	17/20*	23/52*
Atrophy	0/56	4/13*	6/20*	10/52*

*p<0.05; chi square analysis

As described in section 4.1.2.8.1, an increase in interstitial cell tumours of the testes in the mid and high dose males at the 12 and 24 months was observed in this study. It is therefore possible that the effects observed on the testes described above may be secondary to an effect of the Leydig cell tumours. Of the effects noted in the study, atrophy in seminiferous tubules is often observed adjacent to large tumours, especially interstitial cell tumours. Also, atrophy in the seminal vesicles is commonly observed in association with testicular atrophy.

It should also be considered that the effects noted in the male reproductive system are mainly observed in animals at 24 months and, therefore, may be secondary to the natural ageing process of rats rather than a specific effect on the male reproductive system.

As described above, no effects on the male reproductive system was observed in the fertility study in male rabbits (Stauffer Chemical Company, 1982b). Therefore, overall, based on a weight of evidence, it is considered that there is no concern for male fertility.

The effects on male fertility have been investigated for the two structurally related substances, TCPP and TCEP. In a two-generation reproductive toxicity study with TCPP, no effects were observed on the male reproductive system. For TCEP, an effect on male reproductive organ weight was noted in mice and effects on sperm parameters were observed in mice and rats (reported in BAUA, 2006). The lack of a consistent effect on male fertility for these two substances indicates that a read-across from male fertility data on either substance to TCPP is not appropriate.

No evaluation of the female reproductive system was included in the two-year carcinogenicity study with TDCP. The effects on female fertility have been investigated for both TCPP and TCEP. In a two-generation reproductive toxicity study with TCPP, an increase in oestrus cycle length and a decrease in uterus weight were observed in treated females (reported in HSAA008a. In a continuous breeding study in mice with TCEP an impairment of fertility, seen as a decrease in the number of litters produced, was observed. However, in a cross-over mating trial, pregnancy and fertility indices were lower in treated male / control females only, indicating male mice are more sensitive to TCEP treatment than female mice (reported in BAUA, 2006). In a separate study investigating vaginal cytology in mice and rats following treatment with TCEP for 18 weeks, no effect on oestrus cyclicity was observed in mice. In rats, an increase in cycle length and variations in relative frequencies of oestrus stages were observed in the low and mid dose but not the high dose, and therefore the biological significance of the effect is questionable (reported in BAUA, 2006).

Given the inconsistent effects observed on the female reproductive system with TCEP and TCPP, it is not considered appropriate to read-across from female fertility data on either substance to address any possible effects on female fertility of TDCP. As there are no data available for effects of TDCP on female fertility, it is considered that there is a data gap for this particular endpoint in females.

Studies in humans

No studies are available.

Developmental toxicity

Studies in animals

TDCP was administered daily to 20 mated Sprague Dawley female rats/dose group by oral gavage from days 6-15 of gestation (Stauffer Chemical Company, 1978f). Doses administered (in corn oil) were 0, 25, 100 and 400 mg/kg/day. General observations were made daily, body weights measured on days 0, 6, 11, 15 and 19 of gestation. All surviving females were sacrificed on day 19 and the dams and foetuses examined grossly. Numbers of corpora lutea, implantations, resorptions, live foetuses and dead foetuses were noted. One third of the foetuses were examined by serial whole body sectioning using Wilson's technique. The remaining foetuses were eviscerated, fixed and examined for skeletal abnormalities using alizarin red staining.

Pregnancy rates were unaffected by treatment. The mean number of corpora lutea and implantation sites and the implantation efficiencies of the treated animals surviving to day 19 of gestation were similar to or exceeded control values.

Results indicated embryotoxicity in the high dose group. In this group, the rate of resorptions was statistically significantly increased when compared to controls (14.4 % compared to 6.7 %). The foetal viability index for the high dose group was statistically significantly lower than control. No increase was seen at the low or mid doses.

The authors considered that data for mean weight and crown-rump length from two of the 100 mg/kg/day litters should be removed, as they appeared to be of an older gestation age. When this was done, there was a slightly lower mean foetal weight (2.21 g) and crown-rump length (3.18 cm) for the 400 mg/kg/day litters when compared to controls (2.42 g and 3.35 cm) respectively, although these did not reach statistical significance. The finding of increased incidence of dilated lateral ventricles of the brain was slight and within the historical control range. There was considerable evidence of retarded skeletal development in the high dose group; incomplete ossification of intraparietal and supraoccipital, nonossified hyoid and nonossified centres in the sternebrae, nonossified centre of the sacral and caudal portions of the vertebrae, nonossified arches of the sacral vertebrae and incomplete ossification of the pubis, and nonossified centres in the metacarpels and metatarsels. Such findings are consistent with the reduced foetal weight, length and viability at this dose level and indicate developmental retardation which may be related to the maternal toxicity seen at 400 mg/kg/day. The finding of increased incidence of foetuses with angulated ribs at 400 mg/kg/day may have been related to treatment but is of unknown biological significance (no historical control data for this effect was included in the report). A NOAEL of 100 mg/kg/day can be derived for developmental toxicity, based on the statistically significant increased resorptions and the decreased foetal viability index at 400 mg/kg/day.

There were three mortalities at the highest dose. These may have been caused by intubation errors, as findings at necropsy were not considered indicative of treatment-related effects. Clinical signs of toxicity were marked in most animals of the high dose and consisted of urine stains (19/20), hunched appearance (20/20), salivation (18/20) and alopecia (7/20). Rough coat (3/20), bloody crust around the nose (3/20), thinness (2/20) and depression (1/20) were noted in number of high dose animals also. Some clinical signs were also noted in the mid dose group and these may have been treatment-related (alopecia (1/20), hunched appearance (3/20), rough hair coat (1/20) and urine stains (5/20)). There was a significant body weight loss in mid and high dose animals from days 6-11 of treatment. These treated animals lost 15.6 g and 28.9 g, respectively, when compared to untreated animals who gained 22.1 g during this period. From days 11-15, mean weight gain of mid and low dose groups was not different from control, while mean weight gains were reduced in the 400 mg/kg/day group (50% of control). The overall mean weight gain from days 0-19 was significantly reduced ($p<0.05$) at 400 mg/kg/day (56% of controls). The mean weight gain in low and mid dose animals was not different to that of untreated animals. Mean food consumption was significantly reduced to 84.8% at 100 mg/kg/day (days 7-11) and at 400 mg/kg/day to an average of 45% throughout treatment. There were no specific findings at necropsy, which were indicative of a treatment-related effect. A NOAEL for systemic maternal effects of 100 mg/kg/day can be derived from this study.

In a developmental study (Tanaka *et al*., 1981), in which only the abstract of the study is in English, groups of 15-24 female Wistar rats were dosed orally with 0, 25, 50, 100, 200 and 400 mg/kg/day TDCP in olive oil during days 7 through 15 of gestation. At the highest dose level, 11 out of 15 dams died and toxic symptoms included piloerection, salivation and

haematuria. At this dose level, maternal body weight gain and food consumption were significantly reduced when compared to control values. Body weight gain was reduced by 17 %. Maternal kidney weight was significantly increased in the mid and high dose groups when compared to controls (absolute kidney weights were increased by 8.7 % and 35.5% in the mid and high dose groups and the relative weights were increased by 12.2 % and 65.3 %, respectively).

At 400 mg/kg/day, a significant increase in foetal death occurred. As indicated above, 11 out of the 15 dams dosed at this level died. One of the remaining dams had total dead implants. The remaining 3 dams had live foetuses. The number of live foetuses from this treatment group was 22 compared to a total of 194 in the control group (all other treatment groups were comparable to the controls). The number of dead foetuses in the high treatment group was 26 compared to 6 in the control group. The number of dead foetuses in the other treatment groups was comparable to controls. There was no evidence of an adverse effect of TDCP on skeletal development of the foetuses at any dose level. In postnatal examination performed at dose levels of 200 mg/kg/day and below, there was no change in the performance of the offspring in functional tests such as open field, water maze, rota rod, inclined screen, pain reflex and preyer's reflex examinations. From this study, a NOAEL of 200 mg/kg/day can be derived for both maternal and developmental toxicity based on effects observed at 400 mg/kg/day.

Studies in humans

No studies are available.

Summary of toxicity for reproduction

With regard to effects upon fertility, no information is available in humans.

A negative result was obtained in a fertility study carried out in male rabbits in which animals were dosed daily with concentrations of TDCP up to 200 mg/kg/day for twelve weeks and then mated with two females during the last week of treatment. Mating, fertility, pregnancy parameters, sperm analysis and the male reproductive tract were unaffected by treatment.

In a 2-year carcinogenicity study in rats, an evaluation was made of the male reproductive system. Only control and high dose animals were evaluated at 12 months, and no significant differences were noted at this time point. Effects were noted in the testes, epididymis and seminal vesicles in all animals at 24 months, with a trend for higher incidence in the treated groups.

As described in section 4.1.2.8.1, an increase in interstitial cell tumours of the testes in the mid and high dose males at the 12 and 24 months was observed in this study. Therefore, it is possible that the effects observed on the testes may be secondary to an effect of the Leydig cell tumours. It should also be considered that the effects noted in the male reproductive system are only observed in animals at 24 months and, therefore, may be secondary to the natural ageing process of rats rather than a specific effect on the male reproductive system.

In addition to these points, as indicated above, no effect on the male reproductive system and no effects on fertility were observed in the fertility study in male rabbits. Therefore, based on a weight of evidence, it is considered that there is no concern for male fertility.

No evaluation of the female reproductive system was included in the 2-year carcinogenicity study with TDCP. In reproductive toxicity studies with the structurally similar substances,

TCEP and TDCP, inconsistent effects were observed on the female reproductive system. Therefore, it is not considered appropriate to read-across from data on either substance to address the possible effects of TCPP on female fertility. Therefore, it is considered that there is a data gap for female fertility.

In relation to developmental effects, there are no data available in humans. In a developmental study in rats, a dose of 400 mg/kg/day significantly increased the rate of resorptions compared to controls. At this high dose there was also evidence of retarded skeletal development. All of this was accompanied by significant maternal toxicity at this high dose. There was no evidence of embryotoxicity in the absence of maternal effects. The NOAEL for developmental toxicity was 100 mg/kg/day, based on the statistically significant increased resorptions and the decreased foetal viability index at 400 mg/kg/day.

In a second developmental study on rats, the highest dose of 400 mg/kg/day resulted in the deaths of 11 out of 15 of the dams with a reduction in live foetuses and a significantly high incidence of foetal deaths. No observations were noted at 200 mg/kg/day.

An overall NOAEL of 100 mg/kg/day can be derived for developmental toxicity based on the statistically significant increased resorptions and the decreased foetal viability index at 400 mg/kg/day seen in the first developmental study reported. This NOAEL is taken forward to risk characterisation in preference to the NOAEL of 200 mg/kg identified in the second study described, as only the abstract from the second study is available in English and therefore, full details of the study are not available to the Rapporteur.

A NOAEL of 100 mg/kg/day is derived for maternal toxicity, based on the clinical signs of toxicity and statistically significant decrease in mean body weight noted in animals dosed at 400 mg/kg/day in the first study reported.

Risk characterisation [16]

General aspects

This section provides an overview of the occupational use, exposure and toxicological profile of TDCP.

Occupational exposure to TDCP may occur during the:

Manufacture of TDCP
2. Manufacture of flexible PUR foam
   slabstock foams
   moulded foams
3. Cutting of flexible PUR foam
4. Production of foam granules and rebonded PUR foam
5. Manufacture of automotive parts

[16] Conclusion (i) There is a need for further information and/or testing.
Conclusion (ii) There is at present no need for further information and/or testing and no need for risk reduction measures beyond those which are being applied already.
Conclusion (iii) There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

TDCP is a liquid at room temperature with a low vapour pressure of 5.6 x 10^{-6} Pa at 25 ^{0}C and a calculated saturated vapour pressure (SVC) of 1 μg/m^3 at 21 ^{0}C. Exposure to TDCP will be in the form of inhalation and by skin contact.

The sole use of TDCP is as a flame retardant. The main downstream use of TDCP is in the production of flexible polyurethane foam. The flame retardant is not chemically reacted, but physically bound within the matrix and therefore has the potential for migration.

The TDCP manufacturing process is mostly carried out in a closed system, with transfers done using closed lines. The process is mostly computer controlled, thus minimising worker exposure to the substance during its manufacture. The closed system is breached only for sampling and maintenance. Monitoring for operator dermal and inhalation exposure during TDCP manufacturing was carried out by industry by the two EU production plants.

During blending of the manufactured substance and drumming, worker exposure can potentially occur. In addition, during the manufacture and subsequent use of flexible polyurethane foam, there is the potential for worker exposure to TDCP.

For the purposes of risk characterisation, two types of worker exposure are considered. 'Typical' exposure covers the circumstances in which most workers are exposed and is based on normal industry working practice. 'Reasonable worst case' (RWC) exposures are intended to cover exposure situations where adequate control is lacking. RWC exposures are not considered as extreme incidents, but rather higher end exposures which are reasonably foreseeable.

TDCP inhalation exposures did not vary greatly across the industry sectors, and all were relatively low. The highest reasonable worst case inhalation exposure was found during the manufacture of TDCP, at 5.6 μg/m^3. During the production of TDCP, the typical inhalation exposure (8 hr TWA) is 2.8 μg/m^3.

TDCP dermal exposures varied across the industry sectors. The highest dermal exposure was during the production of PUR foam, with a reasonable worst-case exposure of 32 mg/day during the manufacture of moulded foam. The highest typical dermal exposure was found during the production of TDCP, at 10.5 mg/day.

No oral or inhalation toxicokinetic studies, either in animals or humans, are available. Animal data indicate that TDCP is rapidly and extensively absorbed after oral administration and therefore 100% absorption by the oral route is assumed. An *in vitro* percutaneous absorption study determined the percentage dermal penetration through human skin at three doses. The mean total absorption was found to be 15.4 %, 10.7 % and 6.0 %, for doses 0.003, 0.01 and 0.12 mg/cm^2, respectively. Based on these results, 15 % dermal absorption is assumed for scenarios where there is exposure to "neat" TDCP and 30 % dermal absorption is assumed for scenarios 3, 4 and 5, where there is exposure due to handling of foam containing TDCP. Using the default values in the TGD, 100 % absorption by the inhalation route is assumed

TDCP was extensively distributed, metabolised and excreted following oral, dermal and i.v. administration. No accumulation in the body is expected.

Assessment of available data indicates that TDCP is of low acute toxicity via the oral, inhalation and dermal routes.

TDCP produces only minimal dermal and eye irritation and any mild effects are fully reversible. The lack of any substantial skin or eye irritation and the lack of irritation observed

in the acute inhalation studies suggests that TDCP would be unlikely to produce significant respiratory tract irritation.

Evidence from sensitisation studies indicates that TDCP does not possess significant skin sensitisation potential.

With respect to repeated dose toxicity, in a 2-year carcinogenicity study in which rats were fed diets containing TDCP to achieve dose levels of up to 80 mg/kg/day, there was evident systemic toxicity. The most significant effects were histological abnormalities identified in the kidneys and testicular effects in the treated animals. Based on these effects, a LOAEL of 5 mg/kg/day was derived from the study.

In mutagenicity studies, evidence was provided to suggest that TDCP is a bacterial cell mutagen *in vitro*. Positive responses were seen in the Ames mutation assay, following metabolic activation only. In mammalian cell studies, TDCP caused mutation in mouse lymphoma cells in the presence of metabolic activation. *In vivo*, TDCP was not clastogenic in a mouse micronucleus assay and was negative in an *in vivo*/*in vitro* unscheduled DNA synthesis assay. Negative results were also obtained in a second *in vivo* mouse micronucleus assay and in an *in vivo*/*in vitro* urine mutagenicity study. Therefore, TDCP is not considered to be genotoxic *in vivo.*

In a 2-year carcinogenicity study in which groups of 60 male and 60 female rats were fed diets containing TDCP to achieve dose levels of 0, 5, 20 and 80 mg/kg/day, there was a significant increase in the incidence of renal cortical tumours and testicular interstitial cell tumours in animals dosed with 20 and 80 mg/kg/day. A LOAEL of 5 mg/kg/day is taken from this study, based on the hyperplasia of the convoluted tubule epithelium observed in all male animals at 24 months. Hyperplasia is often considered as a pre-neoplastic lesion, which can lead to tumour formation. Based on the available data, it is not unreasonable to assume that the tumours observed have developed through hyperplastic changes.

In an epidemiology study carried out in a TDCP manufacturing plant, there was no evidence of a carcinogenic effect of TDCP.

As stated above, there is some evidence to suggest that TDCP is mutagenic *in vitro.* However, *in vivo* mutagenicity studies were negative, indicating that *in vivo* TDCP is non-genotoxic. This indicates that TDCP may be a threshold carcinogen.

TDCP is classified as Carc. Cat. 3 R40 “Limited evidence of a carcinogenic effect” based on the results of the carcinogenicity study.[17].

In a fertility study carried out in male rabbits, mating, fertility, pregnancy parameters, sperm analysis and the male reproductive tract were unaffected by TDCP. In the 2 year carcinogenicity study described above, effects were noted on the male reproductive organs. The effects were mainly observed in animals at 24 months and therefore could considered to be secondary to the natural ageing process of rats rather than a specific effect on the male reproductive system. In addition, an increased incidence of Leydig cell tumours was observed in mid and high dose animals at 12 and 24 months and so the effects observed on the male reproductive system may be secondary to this carcinogenic effect. Therefore, based on a weight of evidence, it is considered that there is no concern for male fertility and conclusion (ii) is drawn.

[17] Commission Working Group on the Classification and Labelling of Dangerous Substances Meeting on the Health Effects of Pesticides, Existing Chemicals & New Chemicals November 14-18, 2005

No data is available on the effects on the possible effect of TDCP on female fertility. Therefore, it is considered that there is a data gap for female fertility. In considering this data gap, and the possible requirement for a female fertility study to address this endpoint, the available data for TDCP was reviewed. In the chronic toxicity study with TDCP, a LOAEL of 5 mg/kg was derived for repeated dose toxicity and carcinogenicity and brought forward to risk characterisation for both of these endpoints. Given the low LOAEL derived from this study, it is considered possible that any risk to female fertility will already be covered by the LOAEL for repeated dose toxicity and carcinogenicity, and the subsequent conclusions drawn for both of these endpoints.

This consideration is further supported by a study by Janer *et al.*, (2007), where a retrospective analysis of the relationship between NOAEL's for effects on fertility, obtained from two-generation reproductive toxicity studies, and those for repeated dose toxicity, obtained from subchronic studies, was conducted. The results showed that on average there was no, or only a small difference (less than two-fold), in the NOAEL's between the two types of studies. This supports the view that the endpoint for female fertility is likely to be already covered by the LOAEL derived from the chronic study and any risk for female fertility will be addressed within the risk characterisation for repeated dose toxicity and carcinogenicity. Therefore, a conclusion (i) "on hold" is drawn for the endpoint of female fertility.

In a developmental study, there was no evidence of embryotoxicity in the absence of maternal effects. Significant maternal toxicity was seen at 400 mg/kg/day. The NOAEL for developmental effects is determined to be 100 mg/kg/day, based on increased resorptions and decreased foetal viability at 400 mg/kg/day.

Workers

The total number of persons in the EU occupationally exposed to TDCP through all exposure scenarios is unknown.

Occupational exposure to TDCP occurs primarily by the dermal and inhalation exposure. Ingestion is not considered for workers in this risk assessment. Exposure levels used for the manufacture and use of TDCP have been derived from measured data supplied by industry.

To make a comparison between exposure data and data from the toxicological studies for each end-point, total body burdens have been calculated (inhalation, dermal and both combined) for workers for the worst-case and typical inhalation and dermal exposure scenarios.

Scenario 1: Manufacture of TDCP

With regard to TDCP production, the reasonable worst-case inhalation exposure is 5.6 $\mu g/m^3$. Using default values of a 70 kg worker inhaling 10 m^3 of air per 8-hour day and assuming 100 % absorption, the inhalation body burden is 8×10^{-4} mg/kg. For dermal exposure in this scenario, the reasonable worst-case exposure is 0.1 mg/cm^2/day. Using default values of a 70 kg worker with 210 cm^2 of exposed skin and assuming 15 % dermal absorption, the dermal body burden is 4.5×10^{-2} mg/kg. Combining the two values gives a calculated total body burden of 4.6×10^{-2} mg/kg for this scenario.

The typical inhalation exposure for this scenario is 2.8 $\mu g/m^3$. Using the default values stated above, the inhalation body burden is 4×10^{-4} mg/kg. For dermal exposure in this scenario, the

typical exposure is 5 x 10^{-2} mg/cm^2/day, leading to a dermal body burden of 2.3 x 10^{-2} mg/kg. Combining the two values gives a calculated total body burden of 2.3 x 10^{-2} mg/kg.

Scenario 2(a) Manufacture of flexible PUR foam: slabstock foams

Regarding the manufacture of flexible polyurethane foam (slabstock foam), the reasonable worst-case inhalation exposure is 5.1 μg/m^3. Using default values of a 70 kg worker inhaling 10 m^3 of air per 8-hour day and assuming 100 % absorption, the inhalation body burden is 7.3 x 10^{-4} mg/kg. For dermal exposure in this scenario, the reasonable worst-case exposure is 7 x 10^{-2} mg/cm^2/day. Using default values of a 70 kg worker with 420 cm^2 of exposed skin and assuming 15 % dermal absorption, the dermal body burden is 6.3 x 10^{-2} mg/kg. Combining the two values gives a calculated total body burden of 6.4 x 10^{-2} mg/kg for this scenario.

The typical inhalation exposure for this scenario is 0.62 μg/m^3. Using the default values stated above, the inhalation body burden is 8.9 x 10^{-5} mg/kg. For dermal exposure in this scenario, the typical exposure is 2 x 10^{-3} mg/cm^2/day, leading to a dermal body burden of 1.8 x 10^{-3} mg/kg. Combining the two values gives a calculated total body burden of 1.9 x 10^{-3} mg/kg.

Scenario 2(b): Manufacture of flexible PUR foam: moulded foams

Regarding the manufacture of flexible polyurethane foam (moulded foam), the reasonable worst-case inhalation exposure is 4.8 μg/m^3. Using default values of a 70 kg worker inhaling 10 m^3 of air per 8-hour day and assuming 100 % absorption, the inhalation body burden is 6.9 x 10^{-4} mg/kg. For dermal exposure in this scenario, the reasonable worst-case exposure is 7.5 x 10^{-2} mg/cm^2/day. Using default values of a 70 kg worker with 420 cm^2 of exposed skin and assuming 15 % dermal absorption, the dermal body burden is 6.8 x 10^{-2} mg/kg. Combining the two values gives a calculated total body burden of 6.9 x 10^{-2} mg/kg for this scenario.

The typical inhalation exposure for this scenario is 0.63 μg/m^3. Using the default values stated above, the inhalation body burden is 9 x 10^{-5} mg/kg. For dermal exposure in this scenario, the typical exposure is 1.5 x 10^{-3} mg/cm^2/day, leading to a dermal body burden of 1.4 x 10^{-3} mg/kg. Combining the two values gives a calculated total body burden of 1.5 x 10^{-3} mg/kg.

Scenario 3: Cutting of flexible PUR foam

With regard to the scenario of machine cutting of flexible PUR foam, the reasonable worst-case inhalation exposure is 4.1 μg/m^3. Using default values of a 70 kg worker inhaling 10 m^3 of air per 8-hour day and assuming 100 % absorption, the inhalation body burden is 5.9 x 10^{-4} mg/kg. For dermal exposure in this scenario, the reasonable worst-case exposure is 7.1 x 10^{-3} mg/cm^2/day. Using default values of a 70 kg worker with 420 cm^2 of exposed skin and assuming 30 % dermal absorption, the dermal body burden is 1.3 x 10^{-2} mg/kg. Combining the two values gives a calculated total body burden of 1.4 x 10^{-2} mg/kg for this scenario.

The typical inhalation exposure for this scenario is 1.9 μg/m^3. Using the default values stated above, the inhalation body burden is 2.7 x 10^{-4} mg/kg. For dermal exposure in this scenario, the typical exposure is 9.8 x 10^{-4} mg/cm^2/day, leading to a dermal body burden of 1.8 x 10^{-3} mg/kg. Combining the two values gives a calculated total body burden of 2.1 x 10^{-3} mg/kg.

Scenario 4: Production of foam granules and rebonded PUR foam

Regarding the exposure scenario of the production of rebonded foam, the reasonable worst-case inhalation exposure is 4.6 μg/m^3. Using default values of a 70 kg worker inhaling 10 m^3

of air per 8-hour day and assuming 100 % absorption, the inhalation body burden is 6.6 x 10^{-4} mg/kg. The reasonable worst case dermal exposure is 1.7 x 10^{-3} mg/cm^2/day. Using default values of a 70 kg worker with 420 cm^2 of exposed skin and assuming 30 % dermal absorption, the dermal body burden is 3.1 x 10^{-3} mg/kg. Combining the two also gives a reasonable worst-case body burden of 3.8 x 10^{-3} mg/kg.

The typical inhalation exposure for this scenario is 0.59 μg/m^3, which gives a body burden of 8.4 x 10^{-5} mg/kg. The typical dermal exposure is 5.5 x 10^{-4} mg/cm^2/day, leading to a dermal body burden of 1 x 10^{-3} mg/kg. This leads to a total typical body burden of 1 x 10^{-3} mg/kg.

Scenario 5: Manufacture of automotive parts

With regard to occupational exposure during the manufacture of automotive parts, the reasonable worst-case inhalation exposure is 4.6 μg/m^3. Using default values of a 70 kg worker inhaling 10 m^3 of air per 8-hour day and assuming 100 % absorption, the inhalation body burden is 6.6 x 10^{-4} mg/kg. For dermal exposure in this scenario, the reasonable worst-case exposure is 7.1 x 10^{-3} mg/cm^2/day. Using default values of a 70 kg worker with 420 cm^2 of exposed skin and assuming 30 % dermal absorption, the dermal body burden is 1.3 x 10^{-2} mg/kg. Combining the two values gives a calculated total body burden of 1.4 x 10^{-2} mg/kg for this scenario.

The typical inhalation exposure for this scenario is 1.9 μg/m^3. Using the default values stated above, the inhalation body burden is 2.7 x 10^{-4} mg/kg. For dermal exposure in this scenario, the typical exposure is 9.8 x 10^{-4} mg/cm^2/day, leading to a dermal body burden of 1.8 x 10^{-3} mg/kg. Combining the two values gives a calculated total body burden of 2.1 x 10^{-3} mg/kg.

Table 4.49 below gives a summary of all dermal and inhalation exposures for TDCP

Table 4.49 Summary of dermal and inhalation exposure values for all TDCP exposure scenarios

Scenario	Inhalation body burden worst-case (mg/kg)	Dermal body burden worst-case (mg/kg)	Combined worst case body burden (mg/kg)	Inhalation body burden typical case (mg/kg)	Dermal body burden typical case (mg/kg)	Combined typical case body burden (mg/kg)
1	8 x 10^{-4}	4.5 x 10^{-2}	4.6 x 10^{-2}	4 x 10^{-4}	2.3 x 10^{-2}	2.3 x 10^{-2}
2 (a)	7.3 x 10^{-4}	6.3 x 10^{-2}	6.4 x 10^{-2}	8.9 x 10^{-5}	1.8 x 10^{-3}	1.9 x 10^{-3}
2 (b)	6.9 x 10^{-4}	6.8 x 10^{-2}	6.9 x 10^{-2}	9 x 10^{-5}	1.4 x 10^{-3}	1.5 x 10^{-3}
3	5.9 x 10^{-4}	1.3 x 10^{-2}	1.4 x 10^{-2}	2.7 x 10^{-4}	1.8 x 10^{-3}	2.1 x 10^{-3}
4	6.6 x 10^{-4}	3.1 x 10^{-3}	3.8 x 10^{-3}	8.4 x 10^{-5}	1 x 10^{-3}	1 x 10^{-3}
5	6.6 x 10^{-4}	1.3 x 10^{-2}	1.4 x 10^{-2}	2.7 x 10^{-4}	1.8 x 10^{-3}	2.1 x 10^{-3}

The exposure scenarios referred to by numbers in the above table are:

Manufacture of TDCP
Manufacture of flexible PUR foam
    slabstock foams
    moulded foams
Cutting of flexible PUR foam
Production of foam granules and rebonded PUR foam
Manufacture of automotive parts

Acute toxicity

No significant signs of toxicity were seen in experimental animals via the oral, inhalation and dermal routes and so **conclusion (ii)** is drawn for this end-point for all exposure scenarios.

Irritation and corrosivity

TDCP is not a skin or eye irritant and is considered unlikely to be a respiratory irritant and therefore **conclusion (ii)** is drawn for this end-point, for all exposure scenarios.

Sensitisation

Skin

Based on available data, TDCP is not considered to be a skin sensitiser. Therefore, **conclusion (ii)** is drawn for this end-point, for all exposure scenarios.

Respiratory tract

No data are available on the respiratory sensitisation potential of TDCP. There is currently no validated test method available to identify respiratory sensitisers. As TDCP is produced in a closed system, and has a low vapour pressure, it is expected that exposure of the respiratory tract will be low. TDCP is not suspected to be a respiratory sensitiser in humans as no specific cases of suspected respiratory sensitisation in the workplace have been reported. **Conclusion (ii)** is drawn for this end-point, for all exposure scenarios.

Repeated dose toxicity

In relation to repeated dose toxicity, a LOAEL of 5 mg/kg/day was derived from a 2-year study in which rats were dosed with TDCP at concentrations of up to 80 mg/kg/day. This LOAEL was based on hyperplasia of the convoluted tubule epithelium observed in all male animals at 24 months in the kidney and the testicular effects observed at this dose. Assuming 100% absorption by the oral route, this leads to an internal body burden of 5 mg/kg/day.

The minimal MOS for repeated dose toxicity is 150. This is established by taking into account an interspecies factor of 10 (4 for metabolic size differences * 2.5 for sensitivity differences), an intraspecies factor of 5, and a factor of 3 to take account of the use of a LOAEL rather than a NOAEL.

For scenario 1, TDCP production, with respect to inhalation exposure, the body burden for reasonable worst-case exposure was 8×10^{-4} mg/kg. When this is compared with the internal body burden of 5 mg/kg/day, the MOS value is 6,250. With respect to dermal exposure, the body burden for reasonable worst-case exposure is 4.5×10^{-2} mg/kg. This gives a MOS of 111. The total body burden for reasonable worst case for this scenario is 4.6×10^{-2} mg/kg and so results in a MOS of 109. For this scenario, the typical body burden for inhalation exposure is 4×10^{-4} mg/kg, which when compared with the internal body burden results in a MOS of 12,500. For dermal exposure, the typical body burden is 2.3×10^{-2} mg/kg, leading to a MOS of 217. The combined typical body burden is also 2.3×10^{-2} mg/kg, which also gives an MOS of 217.

When the MOSs are compared to the minimal MOS of 150, there is concern for this scenario for the reasonable worst case dermal and combined exposure. Therefore, **conclusion (iii)** is drawn. The MOS for the reasonable worst case combined exposure is also below the minimal MOS. However, it is the dermal exposure, rather than the inhalation exposure which is driving the conclusion (iii) for the combined exposure. There is no concern for the typical dermal exposure or inhalation exposures.

For scenario 2a, the manufacture of flexible slabstock foam, with respect to the reasonable worst case inhalation exposure, the body burden is 7.3×10^{-4} mg/kg, which when compared with the internal body burden of 5 mg/kg/day results in a MOS value of 6,849. With respect to dermal exposure, the body burden for reasonable worst-case exposure is 6.3×10^{-2} mg/kg, leading to a MOS of 79. The total body burden for reasonable worst-case for this scenario is 6.4×10^{-2} mg/kg, resulting in a MOS of 78. For this scenario, the typical body burden for inhalation exposure is 8.9×10^{-5} mg/kg, and when compared with the internal body burden gives a MOS value of 56,180. The typical dermal body burden is estimated to be 1.8×10^{-3} mg/kg, leading to a MOS of 2,778. The total typical body burden is 1.9×10^{-3} mg/kg, leading to an MOS of 2,632.

When the MOSs are compared to the minimal MOS of 150, there is some concern for this scenario. There is concern only for dermal reasonable worst-case exposure. Therefore, **conclusion (iii)** is drawn. The MOS for reasonable worst case combined exposure is also below the minimal MOS. However, it is the dermal exposure, rather than the inhalation exposure, which is driving the conclusion (iii) for the combined exposure. There is no concern for inhalation exposure alone or for typical exposure by either route of exposure.

For scenario 2b, manufacture of moulded PUR foam, with respect to inhalation exposure, the body burden for reasonable worst-case exposure is 6.9×10^{-4} mg/kg. When this is compared with the internal body burden the MOS is 7,246. With respect to dermal exposure, the body burden for reasonable worst-case exposure is 6.8×10^{-2} mg/kg. This gives a MOS of 74. The total body burden for reasonable worst case for this scenario is 6.9×10^{-2} mg/kg, resulting in a MOS of 72. For this scenario, the typical body burden for inhalation exposure is 9×10^{-5} mg/kg, which when compared to the internal body burden results in a MOS value of 55,556. The typical dermal body burden is estimated to be 1.4×10^{-3} mg/kg, leading to a MOS of 3,571. The total typical body burden is 1.5×10^{-3} mg/kg, which gives a MOS of 3,333.

When the MOSs are compared to the minimal MOS of 150, there is some concern for this scenario. There is concern only for dermal reasonable worst-case exposure. Therefore, **conclusion (iii)** is drawn. The MOS for reasonable worst case combined exposure is also below the minimal MOS. However, it is the dermal exposure, rather than the inhalation exposure, which is driving the conclusion (iii) for the combined exposure. There is no concern for inhalation exposure alone or for typical exposure by either route of exposure.

For scenario 3, machine cutting of flexible foam, the body burden for reasonable worst-case inhalation exposure is 5.9×10^{-4} mg/kg. When this is compared with the internal body burden, the MOS value is 8,475. With respect to dermal exposure, the body burden for reasonable worst-case exposure is 1.3×10^{-2} mg/kg, leading to a MOS of 385. The total body burden for reasonable worst-case is 1.4×10^{-2} mg/kg, resulting in a MOS of 357. The typical inhalation body burden is 2.7×10^{-4} mg/kg, which leads to a MOS value of 18,519 when compared with the internal body burden. The typical dermal body burden is estimated to be 1.8×10^{-3} mg/kg, leading to a MOS of 2,778. The total typical body burden is 2.1×10^{-3} mg/kg, which gives a MOS of 2,381.

When the MOSs are compared to the minimal MOS of 150, there is no concern for this scenario and therefore, **conclusion (ii)** is drawn.

Regarding scenario 4, the production of foam granules and rebonded foam, the reasonable worst case inhalation body burden is 6.6 x 10^{-4} mg/kg, which when compared with the internal body burden results in a MOS value of 7,576. The reasonable worst case dermal body burden is 3.1 x 10^{-3} mg/kg, leading to a MOS of 1,613. The combined reasonable worst case body burden is 3.8 x 10^{-3} mg/kg, resulting in a MOS of 1,316. The typical inhalation body burden is 8.4 x 10^{-5} mg/kg. This leads to a MOS of 59,524. The typical dermal and combined body burdens are 1 x 10^{-3} mg/kg, leading to a MOS of 5,000 in both cases.

When the MOSs are compared with the minimal MOS of 150, there is no concern for this scenario and therefore, **conclusion (ii)** is drawn.

For scenario 5, the manufacture of automotive parts, the body burden for reasonable worst-case inhalation exposure is 6.6 x 10^{-4} mg/kg. When this is compared with the internal body burden of 5 mg/kg/day, the MOS value is 7,576. With respect to dermal reasonable worst case exposure, the body burden is 1.3 x 10^{-2} mg/kg, leading to a MOS of 385. The total body burden for reasonable worst-case for this scenario is 1.4 x 10^{-2} mg/kg, resulting in a MOS of 357. For the typical exposures, the inhalation body burden is 2.7 x 10^{-4} mg/kg. This gives a MOS value of 18,519. The dermal body burden is estimated to be 1.8 x 10^{-3} mg/kg, leading to a MOS of 2,778. The combined body burden is 2.1 x 10^{-3} mg/kg, which gives a MOS of 2,381.

When the MOSs are compared to the minimal MOS of 150, there is no concern for this scenario and therefore **conclusion (ii**) is drawn.

Tables 4.50 and **4.51** below summarise the MOSs and conclusions for repeated dose toxicity for worst case and typical exposure, respectively.

Table 4.50 MOS values and conclusions for repeated dose toxicity of TDCP – Reasonable worst case exposure

Minimal MOS :150									
Scenario	**Inhalation**			**Dermal**			**Combined**		
	Body burden (mg/kg)	**MOS**	**Concl.**	**Body burden (mg/kg)**	**MOS**	**Concl**	**Body burden (mg/kg)**	**MOS**	**Concl**
1.Manufacture of TDCP	8 x 10^{-4}	6,250	(ii)	4.5 x 10^{-2}	111	**(iii)**	4.6 x 10^{-2}	109	**(iii)**
2a.Manufacture of flexible PUR foam: Slabstock	7.3 x 10^{-4}	6,849	(ii)	6.3 x 10^{-2}	79	**(iii)**	6.4 x 10^{-2}	78	**(iii)**
2b.Manufacture of flexible PUR foam: Moulded	6.9 x 10^{-4}	7,246	(ii)	6.8 x 10^{-2}	74	**(iii)**	6.9 x 10^{-2}	72	**(iii)**
3.Cutting of flexible PUR foam	5.9 x 10^{-4}	8,475	(ii)	1.3 x 10^{-2}	385	(ii)	1.4 x 10^{-2}	357	(ii)
4.Production of foam granules & rebonded foam	6.6 x 10^{-4}	7,576	(ii)	3.1 x 10^{-3}	1,613	(ii)	3.8 x 10^{-3}	1,316	(ii)
5.Manufacture of automotive parts	6.6 x 10^{-4}	7,576	(ii)	1.3 x 10^{-2}	385	(ii)	1.4 x 10^{-2}	357	(ii)

Table 4.51 MOS values and conclusions for repeated dose toxicity of TDCP – Typical Exposure

Minimal MOS :150									
Scenario	**Inhalation**			**Dermal**			**Combined**		
	Body burden (mg/kg)	**MOS**	**Concl.**	**Body burden (mg/kg)**	**MOS**	**Concl**	**Body burden (mg/kg)**	**MOS**	**Concl**
1.Manufacture of TDCP	4×10^{-4}	12,500	(ii)	2.3×10^{-2}	217	(ii)	$2.3x\ 10^{-2}$	217	(ii)
2a.Manufacture of flexible PUR foam: Slabstock	8.9×10^{-5}	56,180	(ii)	1.8×10^{-3}	2,778	(ii)	1.9×10^{-3}	2,632	(ii)
2b.Manufacture of flexible PUR foam: Moulded	9×10^{-5}	55,556	(ii)	1.4×10^{-3}	3,571	(ii)	1.5×10^{-3}	3,333	(ii)
3.Cutting of flexible PUR foam	2.7×10^{-4}	18,519	(ii)	1.8×10^{-3}	2,778	(ii)	2.1×10^{-3}	2,381	(ii)
4.Production of foam granules & rebonded foam	8.4×10^{-5}	59,524	(ii)	1×10^{-3}	5,000	(ii)	1×10^{-3}	5,000	(ii)
5.Manufacture of automotive parts	2.7×10^{-4}	18,519	(ii)	1.8×10^{-3}	2,778	(ii)	2.1×10^{-3}	2,381	(ii)

Mutagenicity

In relation to mutagenicity, studies have suggested that TCPP is a bacterial cell mutagen. In mammalian cells, positive results were also seen. However, TDCP was negative in an *in vivo* mouse micronucleus test and an *in vivo*/*in vitro* UDS assay, both studies having been conducted in accordance with OECD guidelines. Negative results were also obtained in a second *in vivo* micronucleus study and in an *in vivo*/*in vitro* mutagenicity test. Therefore, *in vivo*, TDCP is considered to be non-genotoxic and a **conclusion (ii)** is drawn for this endpoint for all exposure scenarios.

Carcinogenicity

In a 2-year carcinogenicity study, there was a significant increase in the incidence of renal cortical tumours and testicular interstitial cell tumours in animals dosed with 20 and 80 mg/kg/day. A LOAEL of 5 mg/kg/day is taken from this study, based on the hyperplasia of the convoluted tubule epithelium observed in the kidneys of all male animals at 24 months. Hyperplasia is often considered as a pre-neoplastic lesion, which can lead to tumour formation. Based on the available data, it is not unreasonable to assume that the tumours observed in the 2-year carcinogenicity study developed through hyperplastic changes. Assuming 100% oral absorption, this gives an internal body burden of 5 mg/kg/day.

The minimal MOS for carcinogenicity is 150. This is established by taking into account an interspecies factor of 10 (4 for metabolic size differences * 2.5 for sensitivity differences), an intraspecies factor of 5, and a factor of 3 to take account of the use of a LOAEL rather than a NOAEL.

As the internal body burden here is the same as that for the repeated dose toxicity section above, the calculated MOSs will also be the same. Therefore, it is not proposed to repeat the calculations scenario-by-scenario here.

The MOS values calculated in section 4.1.3.2.4 (Repeated dose toxicity) are presented in **Tables 4.52** and **4.53**, below. The conclusions for carcinogenicity when the MOSs are compared with the minimal MOS of 150 are also presented.

For scenarios 1 (manufacture of TDCP), 2a (manufacture of flexible slabstock PUR foam) and 2b (manufacture of flexible moulded PUR foam), when the MOSs are compared with the minimal MOS of 150, there is concern only for the reasonable worst case dermal exposure. Therefore, **conclusion (iii)** is drawn for the reasonable worst case dermal exposure for all three scenarios. The MOS for the reasonable worse case combined exposures are also below the minimal MOS. However, it is the dermal exposure, rather than the inhalation exposure, which is driving the conclusion (iii) for the combined exposure in each case. For all three scenarios, there is no concern for inhalation exposure alone, or the typical exposure by either route.

For scenarios 3 (cutting of flexible PUR foam), 4 (production of foam granules and rebonded foam) and 5 (manufacture of automotive parts), **conclusion (ii)** is drawn for the reasonable worst case and typical exposures.

Tables 4.52 and **4.53** below summarise the MOSs and conclusions for carcinogenicity for worst case and typical exposures, respectively.

Table 4.52 MOS values and conclusions for carcinogenicity of TDCP – Reasonable worst case exposure

Minimal MOS :150									
Scenario	**Inhalation**			**Dermal**			**Combined**		
	Body burden (mg/kg)	**MOS**	**Concl.**	**Body burden (mg/kg)**	**MOS**	**Concl**	**Body burden (mg/kg)**	**MOS**	**Concl**
1.Manufacture of TDCP	8×10^{-4}	6,250	(ii)	4.5×10^{-2}	111	**(iii)**	4.6×10^{-2}	109	**(iii)**
2a.Manufacture of flexible PUR foam: Slabstock	7.3×10^{-4}	6,849	(ii)	6.3×10^{-2}	79	**(iii)**	6.4×10^{-2}	78	**(iii)**
2b.Manufacture of flexible PUR foam: Moulded	6.9×10^{-4}	7,246	(ii)	6.8×10^{-2}	74	**(iii)**	6.9×10^{-2}	72	**(iii)**
3.Cutting of flexible PUR foam	5.9×10^{-4}	8,475	(ii)	1.3×10^{-2}	385	(ii)	1.4×10^{-2}	357	(ii)
4.Production of foam granules & rebonded foam	6.6×10^{-4}	7,576	(ii)	3.1×10^{-3}	1,613	(ii)	3.8×10^{-3}	1,316	(ii)
5.Manufacture of automotive parts	6.6×10^{-4}	7,576	(ii)	1.3×10^{-2}	385	(ii)	1.4×10^{-2}	357	(ii)

Table 4.53 MOS values and conclusions for carcinogenicity of TDCP – Typical exposure

Minimal MOS :150									
Scenario	**Inhalation**			**Dermal**			**Combined**		
	Body burden (mg/kg)	**MOS**	**Concl.**	**Body burden (mg/kg)**	**MOS**	**Concl**	**Body burden (mg/kg)**	**MOS**	**Concl**
1.Manufacture of TDCP	4×10^{-4}	12,500	(ii)	2.3×10^{-2}	217	(ii)	$2.3x\ 10^{-2}$	217	(ii)
2a.Manufacture of flexible PUR foam: Slabstock	8.9×10^{-5}	56,180	(ii)	1.8×10^{-3}	2,778	(ii)	1.9×10^{-3}	2,632	(ii)
2b.Manufacture of flexible PUR foam: Moulded	9×10^{-5}	55,556	(ii)	1.4×10^{-3}	3,571	(ii)	1.5×10^{-3}	3,333	(ii)
3.Cutting of flexible PUR foam	2.7×10^{-4}	18,519	(ii)	1.8×10^{-3}	2,778	(ii)	2.1×10^{-3}	2,381	(ii)
4.Production of foam granules & rebonded foam	8.4×10^{-5}	59,524	(ii)	1×10^{-3}	5,000	(ii)	1×10^{-3}	5,000	(ii)
5.Manufacture of automotive parts	2.7×10^{-4}	18,519	(ii)	1.8×10^{-3}	2,778	(ii)	2.1×10^{-3}	2,381	(ii)

Toxicity for reproduction

Effects on fertility

There is no concern for male fertility and therefore **conclusion (ii)** is drawn for effects on male fertility for all exposure scenarios.

With respect to effects on female fertility, there are no data available Therefore, it is considered that there is a data gap for female fertility. As discussed in section 4.1.3.1, it is considered that the endpoint for female fertility is likely to be already covered by the low LOAEL of 5 mg/kg derived from the chronic toxicity study with TDCP and any risk for female fertility will be addressed within the risk characterisation for repeated dose toxicity and carcinogenicity. Therefore, a **conclusion (i) "on hold"** is drawn for effects on female fertility for all exposure scenarios.

Developmental toxicity

In a developmental study in rats, a dose of 400 mg/kg/day significantly increased the rate of resorptions compared to controls. Based on this, a NOAEL of 100 mg/kg/day was derived for developmental effects. Assuming 100% absorption by the oral route, this leads to an internal body burden of 100 mg/kg/day.

The minimal MOS for development is 50. This is established by taking into account an interspecies factor of 10 (4 for metabolic size differences * 2.5 for sensitivity differences), an intraspecies factor of 5.

For scenario 1, manufacture of TDCP, the body burden for reasonable worst-case inhalation exposure is 8×10^{-4} mg/kg. When this is compared with the internal body burden of 100

mg/kg/day the MOS value is 125,000. With respect to dermal exposure, the body burden for reasonable worst-case exposure is 4.5 x 10^{-2} mg/kg. This gives a MOS of 2,222. The total body burden for reasonable worst case is 4.6 x 10^{-2} mg/kg, resulting in a MOS of 2,174. The typical body burden for inhalation exposure is 4 x 10^{-4} mg/kg, which when compared with the internal body burden results in a MOS of 250,000. The body burdens for the typical dermal and combined exposures are 2.3 x 10^{-2} mg/kg, leading to a MOS of 4,348 for both.

When these MOSs are compared to a minimal MOS of 50, it is concluded that these MOSs are sufficient and there are no concerns for developmental toxicity for this scenario and so **conclusion (ii)** is drawn.

For scenario 2a, the manufacture of flexible slabstock foam, with respect to inhalation exposure, the body burden for reasonable worst-case is 7.3 x 10^{-4} mg/kg. When this is compared with the internal body burden of 100 mg/kg/day, it results in a MOS value of 136,986. The body burden corresponding to the reasonable worst-case dermal exposure is 6.3 x 10^{-2} mg/kg, leading to a MOS of 1,587. The total body burden for reasonable worst-case for this scenario is 6.4 x 10^{-2} mg/kg, resulting in a MOS of 1,563. The typical body burden for inhalation exposure is 8.9 x 10^{-5} mg/kg, and when compared with the internal body burden gives a MOS value of greater than 1,000,000. The typical dermal body burden is estimated to be 1.8 x 10^{-3}, leading to a MOS of 55,556. For the combined exposure, the body burden is estimated to be 1.9 x 10^{-3}, leading to a MOS of 52,632.

When the MOSs are compared to the minimal MOS of 50, there is no concern for this scenario and so a **conclusion (ii)** is drawn.

For scenario 2b, manufacture of moulded PUR foam, the body burden with respect to the reasonable worst case inhalation exposure is 6.9 x 10^{-4} mg/kg. When this is compared with the internal body burden, the MOS is 144,928. The body burden for reasonable worst-case dermal exposure is 6.8 x 10^{-2} mg/kg. This gives a MOS of 1,471. The total body burden for reasonable worst case for this scenario is 6.9 x 10^{-2} mg/kg, resulting in a MOS of 1,449. The typical body burden for inhalation exposure is 9 x 10^{-5} mg/kg. This gives a MOS value of greater than 1,000,000 when compared with the internal body burden of 100 mg/kg/day. The typical dermal body burden is estimated to be 1.4 x 10^{-3} mg/kg, leading to a MOS of 71,429. The total typical body burden is 1.5 x 10^{-3} mg/kg, which gives a MOS of 66,667.

When the MOSs are compared with the minimal MOS of 50, there is no concern for this scenario. Therefore, **conclusion (ii)** is drawn for this scenario.

For scenario 3, cutting of flexible foam, with respect to the reasonable worst case exposures, the inhalation body burden is 5.9 x 10^{-4} mg/kg. When this is compared with the internal body burden, it results in a MOS value of 169,492. The dermal body burden is 1.3 x 10^{-2} mg/kg, leading to a MOS of 7,692. The total body burden is 1.4 x 10^{-2} mg/kg, resulting in a MOS of 7,143. The typical body burden for inhalation exposure is 2.7 x 10^{-4} mg/kg, leading to a MOS of 370,370. The typical dermal body burden is estimated to be 1.8 x 10^{-3} mg/kg, resulting in a MOS of 55,556. The total typical body burden is 2.1 x 10^{-3} mg/kg, which gives a MOS of 47,619.

When these MOSs are compared to the minimal MOS of 50, it is concluded that they are sufficient and there are no concerns for developmental toxicity for this scenario and so **conclusion (ii)** is drawn.

Regarding scenario 4, the production of foam granules and rebonded foam, the reasonable worst-case inhalation body burden is 6.6 x 10^{-4} mg/kg, which when compared with the

internal body burden results in a MOS value of 151,515. The dermal worst-case body burden is 3.1 x 10^{-3} mg/kg, leading to a MOS of 32,258. The combined worst case body burden is 3.8 x 10^{-3} mg/kg, resulting in a MOS of 26,316. The typical inhalation body burden is 8.4 x 10^{-5} mg/kg. This leads to a MOS of greater than 1,000,000. The typical dermal and combined body burdens are both 1 x 10^{-3} mg/kg, leading to a MOS of 100,000 for both.

When the MOSs are compared with the minimal MOS of 50, there is no concern for this scenario and so **conclusion (ii)** is drawn.

For scenario 5, the manufacture of automotive parts, with respect to the reasonable worst case exposures, the inhalation body burden is 6.6 x 10^{-4} mg/kg. When this is compared with the internal body burden of 100 mg/kg/day, the MOS value is 151,515. The inhalation body burden is 1.3 x 10^{-2} mg/kg, leading to a MOS of 7,692. The total combined body burden is 1.4 x 10^{-2} mg/kg, resulting in a MOS of 7,143. The typical body burden for inhalation exposure is 2.7 x 10^{-4} mg/kg. This gives a MOS value of 370,370. The typical dermal body burden is estimated to be 1.8 x 10^{-3} mg/kg, leading to a MOS of 55,556. The combined typical body burden is 2.1 x 10^{-3} mg/kg, which gives a MOS of 47,619.

When the MOSs are compared to the minimal MOS of 50, there is no concern for this scenario and so **conclusion (ii)** is drawn.

Tables 4.54 and **4.55** below summarise the MOSs and conclusions for developmental toxicity for worst case and typical exposure, respectively.

Table 4.54 MOS values and conclusions for developmental toxicity of TDCP – Reasonable worst case exposure

Minimal MOS :50									
Scenario	**Inhalation**			**Dermal**			**Combined**		
	Body burden (mg/kg)	**MOS**	**Concl.**	**Body burden (mg/kg)**	**MOS**	**Concl**	**Body burden (mg/kg)**	**MOS**	**Concl**
1.Manufacture of TDCP	8 x 10^{-4}	125,000	(ii)	4.5 x 10^{-2}	2,222	(ii)	4.6 x 10^{-2}	2,174	(ii)
2a.Manufacture of flexible PUR foam: Slabstock	7.3 x 10^{-4}	136,986	(ii)	6.3 x 10^{-2}	1,587	(ii)	6.4 x 10^{-2}	1,563	(ii)
2b.Manufacture of flexible PUR foam: Moulded	6.9 x 10^{-4}	144,928	(ii)	6.8 x 10^{-2}	1,471	(ii)	6.9 x 10^{-2}	1,449	(ii)
3.Cutting of flexible PUR foam	5.9 x 10^{-4}	169,492	(ii)	1.3 x 10^{-2}	7,692	(ii)	1.4 x 10^{-2}	7,143	(ii)
4.Production of foam granules & rebonded foam	6.6 x 10^{-4}	151,515	(ii)	3.1 x 10^{-3}	32,258	(ii)	3.8 x 10^{-3}	26,316	(ii)
5.Manufacture of automotive parts	6.6 x 10^{-4}	151,515	(ii)	1.3 x 10^{-2}	7,692	(ii)	1.4 x 10^{-2}	7,143	(ii)

Table 4.55 MOS values and conclusions for developmental toxicity of TDCP – Typical exposure

Minimal MOS :50									
Scenario	**Inhalation**			**Dermal**			**Combined**		
	Body burden (mg/kg)	**MOS**	**Concl**	**Body burden (mg/kg)**	**MOS**	**Concl**	**Body burden (mg/kg)**	**MOS**	**Concl**
1.Manufacture of TDCP	4×10^{-4}	250,000	(ii)	2.3×10^{-2}	4,348	(ii)	2.3×10^{-2}	4,348	(ii)
2a.Manufacture of flexible PUR foam: Slabstock	8.9×10^{-5}	>1,000,000	(ii)	1.8×10^{-3}	55,556	(ii)	1.9×10^{-3}	52,632	(ii)
2b.Manufacture of flexible PUR foam: Moulded	9×10^{-5}	>1,000,000	(ii)	1.4×10^{-3}	71,429	(ii)	1.5×10^{-3}	66,667	(ii)
3.Cutting of flexible PUR foam	2.7×10^{-4}	370,370	(ii)	1.8×10^{-3}	55,556	(ii)	2.1×10^{-3}	47,619	(ii)
4.Production of foam granules & rebonded foam	8.4×10^{-5}	>1,000,000	(ii)	1×10^{-3}	100,000	(ii)	1×10^{-3}	100,000	(ii)
5.Manufacture of automotive parts	2.7×10^{-4}	370,370	(ii)	1.8×10^{-3}	55,556	(ii)	2.1×10^{-3}	47,619	(ii)

Summary of risk characterisation for workers

With respect to worker scenarios 1 (manufacture of TDCP), 2a (manufacture of flexible PUR foam – slabstock) and 2b (manufacture of flexible PUR foam – moulded), the MOS for reasonable worse case dermal exposures for repeated dose toxicity and carcinogenicity are below the minimal MOS and therefore **conclusion (iii)** is drawn. There is no concern for the typical dermal exposures or inhalation exposure for these exposure scenarios. A conclusion (ii) is drawn for the remaining scenarios (worker scenarios 3, 4 and 5) for these endpoints.

A **conclusion (i) "on hold"** is drawn for effects on female fertility for all exposure scenarios.

A **conclusion (ii)** is drawn for all other endpoints for all worker exposure scenarios.

Consumers

The current use pattern provided by industry indicates that most of the TDCP produced in the EU is used in the production of flexible polyurethane foam in Europe. Most of the TDCP used in flexible foam is for the automotive industry, with some used in furniture. Consumers do not come into direct contact with these foams. The foam is only used in ways in which it is enclosed and therefore it is concluded that exposure to consumers is negligible.

For exposure to TDCP due to its release from flexible PUR foam, the end-points of concern are repeated dose toxicity, mutagenicity, carcinogenicity and reproductive toxicity.

Ageing studies that have been carried out have indicated that flame retardants are retained within PUR foam. Therefore, consumer exposure to flame retardants from these foams is expected to be very low. From the chamber tests that were performed on a similar substance,

TCPP, a RWC inhalation exposure value of 3.8 μg/m^3 24 hour TWA is used for risk characterisation. This is to allow for people, particularly elderly people, who spend a large proportion of their time indoors in a room with PU foam-containing furniture. A typical exposure value of 2.8 μg/m^3 is used for risk characterisation, on the basis of a consumer spending 18 out of 24 hours in rooms where there is PU foam-containing furniture. A RWC dermal body burden is taken as 0.0011 mg/kg. A value for RWC oral ingestion for children of 0.2 μg/kg/day, assuming a bodyweight of 9.1 kg is taken forward (taken from BAUA, 2006).

It is worth noting that the work ongoing to monitor the release of fire retardant from foam over years rather than hours, seems to indicate that the loss of fire retardant is negligible, in which case exposure would be negligible. The values taken forward for risk characterisation may therefore be an over-estimate.

The reasonable worst-case inhalation exposure is 3.8 μg/m^3. Using default values of a 70 kg person inhaling 20 m^3 of air per 24-hour day and assuming 100% absorption, the inhalation body burden is 1 μg/kg. The typical exposure of 2.8 μg/m^3 leads to an inhalation body burden of 0.6 μg/kg, assuming a 70 kg person inhales 0.75 x 20 m^3 in 18 hours.

Acute toxicity

As with the worker section above, **conclusion (ii)** is drawn for consumers in relation to acute toxicity.

Irritation and corrosivity

TDCP is not a skin or eye irritant and is considered unlikely to be a respiratory irritant. Therefore, as with the worker section above, **conclusion (ii)** is drawn for consumers for this endpoint.

Repeated dose toxicity

Based on hyperplasia of the kidney convoluted tubule epithelium and the testicular effects observed in all treated males rats in a 2-year study, a LOAEL of 5 mg/kg/day was derived for repeated dose toxicity. Assuming 100 % absorption by the oral route, this leads to an internal body burden of 5 mg/kg/day.

The minimal MOS for repeated dose toxicity for consumers is 300. This is established by taking into account an interspecies factor of 10 (4 for metabolic size differences * 2.5 for sensitivity differences), an intraspecies factor of 10, and a factor of 3 to take account of the use of a LOAEL rather than a NOAEL.

Regarding potential inhalation exposure to TDCP due to its release from flexible PUR foam, the body burden for reasonable worst-case exposure was 1 μg/kg. This gives a MOS value of 5000. When this MOS is compared to the minimal MOS of 300, it is concluded that this MOS is sufficient and there are no concerns for repeated dose toxicity to consumers for this scenario and so **conclusion (ii)** is drawn

Regarding potential dermal exposure due to the release of TDCP from flexible PUR foam, the reasonable worst-case body burden is taken as 0.0011 mg/kg, leading to a MOS of 4545.

Given this MOS, a **conclusion (ii)** can be drawn for dermal exposure for consumers for this scenario.

For children, the oral route is also considered. A RWC oral ingestion of 0.2 μg/kg (assuming a body weight of 9.1 kg) has been taken from the TCEP risk assessment report (BAUA, 2006). When this is compared to the internal body burden of 5 mg/kg, the MOS is 25,000. It is considered that this MOS is sufficient, and so there is no concern for exposure of children via the oral route i.e. **conclusion (ii)** is drawn.

Mutagenicity

As with the worker section above, **conclusion (ii)** is drawn for consumers with respect to mutagenicity.

Carcinogenicity

In a 2-year carcinogenicity study, there was a significant increase in the incidence of renal cortical tumours and testicular interstitial cell tumours in animals dosed with 20 and 80 mg/kg/day. A LOAEL of 5 mg/kg/day is taken from this study, based on the hyperplasia of the convoluted tubule epithelium observed in the kidney of all male animals at 24 months. Assuming 100 % oral absorption, this gives an internal body burden of 5 mg/kg/day.

The minimal MOS for carcinogenicity is 300. This is established by taking into account an interspecies factor of 10 (4 for metabolic size differences * 2.5 for sensitivity differences), an intraspecies factor of 10, and a factor of 3 to take into account the use of a LOAEL rather than a NOAEL.

Regarding potential inhalation exposure to TDCP due to its release from flexible PUR foam, the body burden for reasonable worst-case exposure was 1 μg/kg. This gives a MOS value of 5,000. When this MOS is compared to the minimal MOS of 300, it can be concluded that this MOS is sufficient and there are no concerns for consumers in relation to carcinogenicity and so **conclusion (ii)** is drawn.

Regarding potential dermal exposure due to the release of TDCP from flexible PUR foam, the reasonable worst-case body burden is taken as 0.0011 mg/kg, leading to a MOS of 4545. Given this MOS, a **conclusion (ii)** can be drawn for dermal exposure for consumers for this scenario.

For children, the oral route is also considered. A RWC oral ingestion of 0.2 μg/kg (assuming a body weight of 9.1 kg) has been taken from the TCEP risk assessment report (BAUA, 2006). When this is compared to the internal body burden of 5 mg/kg the MOS is 25,000. It is considered that this MOS is sufficient, and so there is no concern for exposure of children via the oral route i.e. **conclusion (ii)** is drawn.

Toxicity for reproduction

Effects on fertility

As with the worker section above, **conclusion (ii)** is drawn for consumers with respect to effects on male fertility.

With respect to effects on female fertility, there are no data available. Therefore, it is considered that there is a data gap for female fertility. As with the worker section above, it is considered that the endpoint for female fertility is likely to be already covered by the low LOAEL of 5 mg/kg derived from the chronic toxicity study with TDCP and any risk for female fertility will be addressed within the risk characterisation for repeated dose toxicity and carcinogenicity. Therefore, a **conclusion (i) "on hold"** is drawn for effects on female fertility.

Developmental toxicity

In a developmental study in rats, a significant increase in the rate of resorptions and significantly lower foetal viability index were observed. A NOAEL of 100 mg/kg/day was derived for developmental effects. Assuming 100 % absorption by the oral route, this leads to an internal body burden of 100 mg/kg/day.

The minimal MOS for development is 100. This is established by taking into account an interspecies factor of 10 (4 for metabolic size differences * 2.5 for sensitivity differences), and an intraspecies factor of 10.

Regarding potential inhalation exposure to TDCP due to its release from flexible PUR foam, the body burden for reasonable worst-case exposure was 1 μg/kg. This gives a MOS value of 100,000. When this MOS is compared to the minimal MOS of 100, it is concluded that this MOS is sufficient and there are no concerns for developmental toxicity to consumers and so **conclusion (ii)** is drawn.

Regarding potential dermal exposure due to the release of TDCP from flexible PUR foam, the reasonable worst-case body burden is taken as 0.0011 mg/kg, leading to a MOS of 90,909. Given this MOS, a **conclusion (ii)** can be drawn for dermal exposure for consumers for developmental toxicity.

Summary of risk characterisation for consumers

Conclusion (ii) is drawn for consumers for all exposure scenarios for all endpoints except effects on female fertility, for which a **conclusion (i) "on hold"** is drawn.

Humans exposed via the environment

Regional exposure

Repeated dose toxicity

A LOAEL of 5 mg/kg/day was derived from a 2-year study. This was based on hyperplasia of the kidney convoluted tubule epithelium and testicular effects observed in all male animals at 24 months. Assuming 100 % absorption by the oral route, this leads to an internal body burden of 5 mg/kg/day.

The minimal MOS for repeated dose toxicity is 300. This is established by taking into account an interspecies factor of 10 (4 for metabolic size differences * 2.5 for sensitivity differences),

an intraspecies factor of 10, and a factor of 3 to take account of the use of a LOAEL rather than a NOAEL.

From section 4.1.1.3, the total daily human exposure to TDCP from regional sources is 1.52 x 10^{-5} mg/kg/day. This leads to a MOS of 328,947. This MOS is considered sufficient and thus a **conclusion (ii)** is drawn for repeated dose toxicity for regional exposure.

Mutagenicity

As with the worker section above, **conclusion (ii)** is drawn for regional exposure of man via the environment with respect to mutagenicity.

Carcinogenicity

A risk characterisation for regional exposure is carried out for the carcinogenicity end-point using the LOAEL of 5 mg/kg/day from the 2 year carcinogenicity study as a starting point. Assuming 100 % absorption by the oral route, this leads to an internal body burden of 5 mg/kg.

The minimal MOS for carcinogenicity is 300. This is established by taking into account an interspecies factor of 10 (4 for metabolic size differences * 2.5 for sensitivity differences), an intraspecies factor of 10 and a further factor of 3 to take into account the use of a LOAEL rather than a NOAEL.

From section 4.1.1.3, the total daily human exposure to TDCP from regional sources is 1.52 x 10^{-5} mg/kg/day. This leads to a MOS of 328,947. This MOS is considered sufficient and thus a **conclusion (ii)** is drawn for carcinogenicity for regional exposure.

Reproductive toxicity

Effects on fertility

As with the consumer section above, **conclusion (ii)** is drawn for effects on male fertility and **conclusion (i) "on hold**" is drawn for effects on female fertility.

Developmental toxicity

From a developmental toxicity study in rats, a NOAEL of 100 mg/kg/day was derived for developmental effects. Assuming 100 % absorption by the oral route, this leads to an internal body burden of 100 mg/kg/day.

The minimal MOS for development is 100. This is established by taking into account an interspecies factor of 10 (4 for metabolic size differences * 2.5 for sensitivity differences) and an intraspecies factor of 10.

The total daily human exposure to TDCP from regional sources is 1.52 x 10^{-5} mg/kg/day. This leads to a MOS of >6,000,000, and so a **conclusion (ii)** is drawn for developmental toxicity for regional exposure.

Local exposure

Repeated dose toxicity

A LOAEL of 5 mg/kg/day was derived for repeat dose toxicity. Assuming 100 % absorption by the oral route, this leads to an internal body burden of 5 mg/kg/day.

The minimal MOS for repeated dose toxicity is 300. This is established by taking into account an interspecies factor of 10 (4 for metabolic size differences * 2.5 for sensitivity differences), an intraspecies factor of 10, and a factor of 3 to take account of the use of a LOAEL rather than a NOAEL.

From section 4.1.1.3, the highest continuous local exposure is for confidential use E1b and is estimated to be 6.99 x 10^{-4} mg/kg/day (which is taken from **Table 4.42**).

Comparing the local exposure value of 6.99 x 10^{-4} mg/kg/day to an internal body burden of 5 mg/kg leads to a MOS of 7,153. When this is compared to a minimal MOS of 300 there is no concern, and so **conclusion (ii)** is drawn for repeated dose toxicity for local exposure.

Mutagenicity

As with the worker section above, **conclusion (ii)** is drawn for regional exposure of man via the environment with respect to mutagenicity.

Carcinogenicity

A risk characterisation for local exposure is carried out for the carcinogenicity end-point using the LOAEL of 5 mg/kg/day from the 2-year carcinogenicity study as a starting point. Assuming 100 % absorption by the oral route, this leads to an internal body burden of 5 mg/kg.

The minimal MOS for carcinogenicity is 300. This is established by taking into account an interspecies factor of 10 (4 for metabolic size differences * 2.5 for sensitivity differences), an intraspecies factor of 10, and a factor of 3 to take into account the use of a LOAEL rather than a NOAEL.

From section 4.1.1.3, the highest continuous local exposure is for confidential use E1b and is estimated to be 6.99 x 10^{-4} mg/kg/day (taken from **Table 4.42**).

Comparing the local exposure value of 6.99 x 10^{-4} mg/kg/day to an internal body burden of 5 mg/kg leads to a MOS of 7,153. When this is compared to a minimal MOS of 300, there is no concern, and so **conclusion (ii)** is drawn for carcinogenicity for local exposure.

Reproductive toxicity

Effects on fertility

As with the consumer section above, **conclusion (ii)** is drawn for effects on male fertility and **conclusion (i) "on hold**" is drawn for effects on female fertility.

Developmental toxicity

A NOAEL of 100 mg/kg/day was derived for developmental effects. Assuming 100% absorption by the oral route, this leads to an internal body burden of 100 mg/kg/day.

The minimal MOS for development is 100. This is established by taking into account an interspecies factor of 10 (4 for metabolic size differences * 2.5 for sensitivity differences), an intraspecies factor of 10.

From T**able 4.42**, for confidential use E1b, the highest continuous local exposure is estimated to be 6.99 x 10^{-4} mg/kg/day.

Comparing the local exposure value of 6.99 x 10^{-4} mg/kg/day to an internal body burden of 100 mg/kg leads to a MOS of 143,061. When this is compared to a minimal MOS of 100, there is no concern, and so **conclusion (ii)** is drawn for developmental toxicity for local exposure**.**

Summary of risk characterisation for exposure via the environment

Tables 4.56 and **4.57** below summarise the MOSs and conclusions for regional and local exposures to TDCP.

Table 4.56 MOSs and conclusions for regional exposure to TDCP

End-point	Exposure mg/kg/day	mMOS	MOS	Conclusion
Repeated Dose	1.52x 10^{-5}	300	328,947	(ii)
Mutagenicity	1.52 x 10^{-5}			(ii)
Carcinogenicity	1.52 x 10^{-5}	300	328,947	(ii)
Fertility - Male		-		(ii)
Fertility - Female		-		(i) "on hold"
Development	1.52 x 10^{-5}	100	>6,000,000	(ii)

Table 4.57 MOSs and conclusions for local exposure to TDCP

End-point	Exposure mg/kg/day	mMOS	MOS	Conclusion
Repeated Dose	6.99 x 10^{-4}	300	7,153	(ii)
Mutagenicity				(ii)
Carcinogenicity	6.99 x 10^{-4}	300	7,153	(ii)
Fertility - Male		-	-	(ii)
Fertility - Female		-	-	(i) "on hold"
Development	6.99 x 10^{-4}	100	6.99 x 10^{-4}	(ii)

Combined exposure

The combined exposure to TDCP is the sum of all the specific sources (occupational exposure, consumer exposure and indirect exposure via the environment) and by all routes of exposure (oral, dermal and inhalation). Therefore, a worst case estimate for this combined

exposure would be the sum of the RWC estimates, for inhalation and dermal exposures, for the three populations; i.e. workers, consumers and man exposed via the environment.

Consumers may be exposed to TDCP indirectly from flexible foam used in upholstery and bedding. Exposure is also possible indirectly *via* environmental sources. In calculating the combined exposures, the RWC exposures have been used, and these are presented in **Table 4.58**, below.

Table 4.58 Combined regional and local exposure to TDCP (excluding occupational exposure)

Source of exposure	Exposure	Body burdens (mg/kg/day)
Consumer		
Release of TCPP from flexible polyurethane foam		
Inhalation	0.0038 mg/m^3	0.001
Dermal	0.0011 mg/kg	0.0011
Man via the environment		
Local exposure	6.99 x 10^{-4} mg/kg/day	6.99 x 10^{-4}
Regional exposure	1.52 x 10^{-5} mg/kg/day	1.52 x 10^{-5}
Combined local		2.8 x 10^{-3}
Combined regional		2.1 x 10^{-3}

As discussed in section 4.1.1.4, occupational exposures are not included in the combined exposure calculation. As can be seen from **Table 4.49** in section 4.1.3.2., the body burdens for the reasonable worst case and typical occupational exposures are significantly higher than those for consumers or for indirect exposure via the environment. Therefore, the occupational exposure value would dominate the combined exposure estimate, resulting in conclusion (iii)'s being drawn, as per those for the worker risk characterisation. It is therefore considered more appropriate to exclude occupational exposure from the combined exposure risk characterisation.

Repeated dose toxicity

In relation to repeated dose toxicity, a LOAEL of 5 mg/kg/day was derived from a 2-year study, in which hyperplasia of the convoluted tubule epithelium and testicular effects were observed in all male animals. Assuming 100 % absorption by the oral route, this leads to an internal body burden of 5 mg/kg/day.

The minimal MOS for repeated dose toxicity is 300. This is established by taking into account an interspecies factor of 10 (4 for metabolic size differences * 2.5 for sensitivity differences), an intraspecies factor of 10, and a factor of 3 to take account of the use of a LOAEL rather than a NOAEL.

From **Table 4.58**, above, the body burden for the combined local exposure is 2.8 x 10^{-3} mg/kg. When this is compared with the internal body burden of 5 mg/kg/day, the MOS is 1,786. There are no concerns for the combined local exposure and so **conclusion (ii)** is drawn.

The body burden for the combined regional exposure is 2.1 x 10^{-3} mg/kg, which gives a MOS of 2,381. There are no concerns for the combined regional exposure and so **conclusion (ii)** is drawn also for repeated dose toxicity.

Mutagenicity

As with the worker section above, **conclusion (ii)** is drawn for regional exposure of man via the environment with respect to mutagenicity.

Carcinogenicity

A risk characterisation for local exposure is carried out for the carcinogenicity end-point using the LOAEL of 5 mg/kg/day from the 2-year carcinogenicity study as a starting point. Assuming 100 % absorption by the oral route, this leads to an internal body burden of 5 mg/kg.

The minimal MOS for carcinogenicity is 300. This is established by taking into account an interspecies factor of 10 (4 for metabolic size differences * 2.5 for sensitivity differences), an intraspecies factor of 10, and a factor of 3 to take into account the use of a LOAEL rather than a NOAEL.

From **Table 4.58**, above, the body burden for the combined local exposure is 2.8 x 10^{-3} mg/kg. When this is compared with the internal body burden of 5 mg/kg/day, the MOS is 1,786. There are no concerns for the combined local exposure and so **conclusion (ii)** is drawn.

The body burden for the combined regional exposure is 2.1 x 10^{-3} mg/kg, which gives a MOS of 2,381. There are no concerns for the combined regional exposure and so **conclusion (ii)** is also drawn.

Reproductive toxicity

Effects on fertility

As with the consumer section above, **conclusion (ii)** is drawn for effects on male fertility and **conclusion (i) "on hold**" is drawn for effects on female fertility.

Developmental toxicity

From a developmental study in rats, a NOAEL of 100 mg/kg/day was derived for developmental effects. Assuming 100 % absorption by the oral route, this leads to an internal body burden of 100 mg/kg/day.

The minimal MOS for development is 100. This is established by taking into account an interspecies factor of 10 (4 for metabolic size differences * 2.5 for sensitivity differences), an intraspecies factor of 10.

From **Table 4.58**, above, the body burden for the combined local exposure is 2.8 x 10^{-3} mg/kg. When this is compared with the internal body burden of 100 mg/kg/day, the MOS is 35,714. There are no concerns for the combined local exposure and so **conclusion (ii)** is drawn for developmental toxicity.

The body burden for the combined regional exposure is 2.1 x 10^{-3} mg/kg, which gives a MOS of 47,619. There are no concerns for the combined regional exposure and so **conclusion (ii)** is also drawn.

HUMAN HEALTH (PHYSICO-CHEMICAL PROPERTIES)

Exposure assessment

Exposure potentially occurs in the workplace during the manufacture of TDCP and during the manufacture of PUR foam containing TDCP.

Effects assessment: Hazard identification

Explosivity

Explosive properties have not been tested. Based on its chemical structure and the known synthetic route of manufacture via an exothermic chemical reaction, there is no indication that the substance is thermodynamically unstable. The DCS test used for boiling point measurement of TDCP showed no exotherms. The substance's structure does not contain any of the more commonly known endothermic groups such as azides, cyano-, dienes, peroxide or chlorate. Therefore, TDCP is not expected to possess explosive properties.

Flammability

Based on the known chemical and physical properties of TDCP and its chemical structure, it is not expected to produce flammable gases in contact with water or damp air.

Oxidizing potential

Oxidising properties have not been tested. By reference to the structural formula, it can be seen that TDCP contains highly electronegative atoms of chlorine; however, the fact that these elements are only bonded to carbon and/or hydrogen renders it unlikely that this will confer oxidising properties on the substance.

Risk characterisation

TDCP gives no reason for concern to human health in relation to its physico-chemical properties. There is no need for further information and/or testing (**conclusion (ii)**).

RESULTS [18]

INTRODUCTION

The conclusions from the risk characterisation processes are brought together and summarised below.

ENVIRONMENT

Environment

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

Conclusion (ii) applies at the regional scale in all compartments and to all current local life cycle stages. TDCP does not meet all of the PBT criteria (it meets the screening criteria for P or vP).

It is understood that the life cycle stages associated with Confidential Use C (i.e. C1a, C1b and C2) are no longer relevant in Europe, on the basis of industry information. Should it be the case that supply for Use C resumes in future, conclusion (i) or (iii) would apply for some compartments and some life cycle stages.

The Rapporteur has no reason to anticipate significant tonnage increases in the near future, based on industry information and general research.

The main area of uncertainty is the assumption regarding limited availability of TDCP for release from foams. This is discussed in section 3.1 and will affect all life cycle stages associated with foam production, processing and use (local life cycle stages A1a, A1b, A2, B1, B2, I1 and J1, and the regional background). The sensitivity of the risk assessment to this uncertainty has been considered, as follows. While the exact level of availability is uncertain, it would be very unlikely to be as high as 40%, which is the level that applies for the related substance TCPP (which is well supported by experimental evidence). Taking this as the worst case, PEC/PNEC ratios could potentially be (in most cases) four times higher for TDCP foam-related life cycle stages. It is clear that even in this worst case, no additional risks would be identified for these local life cycle stages. The Rapporteur has no reason to anticipate significant tonnage increases in the near future, based on industry information and general research.

[18] Conclusion (i) There is a need for further information and/or testing.
Conclusion (ii) There is at present no need for further information and/or testing and no need for risk reduction measures beyond those which are being applied already.
Conclusion (iii) There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

HUMAN HEALTH

Human health (toxicity)

Workers

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

A conclusion (i) "on hold" applies to effects on female fertility for all worker exposure scenarios.

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

Conclusion (ii) applies to all worker exposure scenarios for the endpoints acute toxicity, irritation, sensitisation, mutagenicity, effects on male fertility and developmental toxicity.

Conclusion (ii) applies to typical dermal exposure and inhalation exposures, both reasonable worst case and typical, during the manufacture of TDCP (worker scenario 1), manufacture of flexible PUR foam – stabstock (worker scenario 2a), and manufacture of flexible PUR foam – moulded (worker scenario 2b) in relation to repeated dose toxicity and carcinogenicity.

Conclusion (ii) also applies to all other worker exposure scenarios (worker scenarios 3, 4 and 5) for both reasonable worst case and typical exposures in relation to repeated dose toxicity and carcinogenicity.

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

Conclusion (iii) applies to reasonable worst case dermal exposure during the manufacture of TDCP (worker scenario 1), manufacture of flexible PUR foam – stabstock (worker scenario 2a) and manufacture of flexible PUR foam – moulded (worker scenario 2b) in relation to repeated dose toxicity and carcinogenicity.

Consumers

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

A conclusion (i) "on hold" applies to effects on female fertility for all consumer exposures.

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

Conclusion (ii) applies to all consumer exposure scenarios for the endpoints acute toxicity, irritation, sensitisation, repeated dose toxicity, mutagenicity, carcinogenicity, effects on male fertility and developmental toxicity.

Humans exposed via the environment

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

A conclusion (i) "on hold" applies to effects on female fertility for both regional and local exposures.

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

Conclusion (ii) applies to both regional and local exposures for the endpoints acute toxicity, irritation, sensitisation, repeated dose toxicity, mutagenicity, carcinogenicity, effects on male fertility and developmental toxicity.

Combined exposure

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

A conclusion (i) "on hold" applies to effects on female fertility for combined exposure.

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

Conclusion (ii) applies to combined exposure for the endpoints acute toxicity, irritation, sensitisation, repeated dose toxicity, mutagenicity, carcinogenicity, effects on male fertility and developmental toxicity.

Human health (risks from physico-chemical properties)

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

Conclusion (ii) applies to all endpoints.

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ABBREVIATIONS

ADI	Acceptable Daily Intake
AF	Assessment Factor
ASTM	American Society for Testing and Materials
ATP	Adaptation to Technical Progress
AUC	Area Under The Curve
B	Bioaccumulation
BBA	Biologische Bundesanstalt für Land- und Forstwirtschaft
BCF	Bioconcentration Factor
BMC	Benchmark Concentration
BMD	Benchmark Dose
BMF	Biomagnification Factor
bw	body weight / *Bw*, *b.w.*
C	Corrosive (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
CA	Chromosome Aberration
CA	Competent Authority
CAS	Chemical Abstract Services
CEC	Commission of the European Communities
CEN	European Standards Organisation / European Committee for Normalisation
CMR	Carcinogenic, Mutagenic and toxic to Reproduction
CNS	Central Nervous System
COD	Chemical Oxygen Demand
CSTEE	Scientific Committee for Toxicity, Ecotoxicity and the Environment (DG SANCO)
CT_{50}	Clearance Time, elimination or depuration expressed as half-life
d.wt	dry weight / dw
dfi	daily food intake
DG	Directorate General
DIN	Deutsche Industrie Norm (German norm)
DNA	DeoxyriboNucleic Acid
DOC	Dissolved Organic Carbon
DT50	Degradation half-life or period required for 50 percent dissipation / degradation
DT90	Period required for 50 percent dissipation / degradation
E	Explosive (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
EASE	Estimation and Assessment of Substance Exposure Physico-chemical properties [Model]
EbC50	Effect Concentration measured as 50% reduction in biomass growth in algae tests

EC	European Communities
EC10	Effect Concentration measured as 10% effect
EC50	median Effect Concentration
ECB	European Chemicals Bureau
ECETOC	European Centre for Ecotoxicology and Toxicology of Chemicals
ECVAM	European Centre for the Validation of Alternative Methods
EDC	Endocrine Disrupting Chemical
EEC	European Economic Communities
EINECS	European Inventory of Existing Commercial Chemical Substances
ELINCS	European List of New Chemical Substances
EN	European Norm
EPA	Environmental Protection Agency (USA)
ErC50	Effect Concentration measured as 50% reduction in growth rate in algae tests
ESD	Emission Scenario Document
EU	European Union
EUSES	European Union System for the Evaluation of Substances [software tool in support of the Technical Guidance Document on risk assessment]
F(+)	(Highly) flammable (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
FAO	Food and Agriculture Organisation of the United Nations
FELS	Fish Early Life Stage
FR	Flame retardant
GLP	Good Laboratory Practice
HEDSET	EC/OECD Harmonised Electronic Data Set (for data collection of existing substances)
HELCOM	Helsinki Commission -Baltic Marine Environment Protection Commission
HPLC	High Pressure Liquid Chromatography
HPVC	High Production Volume Chemical (> 1000 t/a)
IARC	International Agency for Research on Cancer
IC	Industrial Category
IC50	median Immobilisation Concentration or median Inhibitory Concentration
ILO	International Labour Organisation
IPCS	International Programme on Chemical Safety
ISO	International Organisation for Standardisation
IUCLID	International Uniform Chemical Information Database (existing substances)
IUPAC	International Union for Pure and Applied Chemistry
JEFCA	Joint FAO/WHO Expert Committee on Food Additives
JMPR	Joint FAO/WHO Meeting on Pesticide Residues
Koc	organic carbon normalised distribution coefficient

Kow	octanol/water partition coefficient
Kp	solids-water partition coefficient
L(E)C50	median Lethal (Effect) Concentration
LAEL	Lowest Adverse Effect Level
LC50	median Lethal Concentration
LD50	median Lethal Dose
LEV	Local Exhaust Ventilation
LLNA	Local Lymph Node Assay
LOAEL	Lowest Observed Adverse Effect Level
LOEC	Lowest Observed Effect Concentration
LOED	Lowest Observed Effect Dose
LOEL	Lowest Observed Effect Level
MAC	Maximum Allowable Concentration
MATC	Maximum Acceptable Toxic Concentration
MC	Main Category
MITI	Ministry of International Trade and Industry, Japan
MOE	Margin of Exposure
MOS	Margin of Safety
MW	Molecular Weight
N	Dangerous for the environment (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC
NAEL	No Adverse Effect Level
NOAEL	No Observed Adverse Effect Level
NOEL	No Observed Effect Level
NOEC	No Observed Effect Concentration
NTP	National Toxicology Program (USA)
O	Oxidizing (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
OECD	Organisation for Economic Cooperation and Development
OEL	Occupational Exposure Limit
OJ	Official Journal
OSPAR	Oslo and Paris Convention for the protection of the marine environment of the Northeast Atlantic
P	Persistent
pKa	negative log of the acid dissociation constant
PBT	Persistent, Bioaccumulative and Toxic
PBPK	Physiologically Based PharmacoKinetic modelling
PBTK	Physiologically Based ToxicoKinetic modelling

PEC	Predicted Environmental Concentration
pH	logarithm (to the base 10) (of the hydrogen ion concentration $\{H^+\}$
pKa	logarithm (to the base 10) of the acid dissociation constant
pKb	logarithm (to the base 10) of the base dissociation constant
PNEC	Predicted No Effect Concentration
POP	Persistent Organic Pollutant
PPE	Personal Protective Equipment
PUR	Polyurethane
QSAR	(Quantitative) Structure-Activity Relationship
R phrases	Risk phrases according to Annex III of Directive 67/548/EEC
RAR	Risk Assessment Report
RC	Risk Characterisation
RfC	Reference Concentration
RfD	Reference Dose
RNA	RiboNucleic Acid
RPE	Respiratory Protective Equipment
RWC	Reasonable Worst Case
S phrases	Safety phrases according to Annex III of Directive 67/548/EEC
SAR	Structure-Activity Relationships
SBR	Standardised birth ratio
SCE	Sister Chromatic Exchange
SDS	Safety Data Sheet
SETAC	Society of Environmental Toxicology And Chemistry
SNIF	Summary Notification Interchange Format (new substances)
SSD	Species Sensitivity Distribution
STP	Sewage Treatment Plant
T(+)	(Very) Toxic (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
TDI	Tolerable Daily Intake
TG	Test Guideline
TGD	Technical Guidance Document [1]
TNsG	Technical Notes for Guidance (for Biocides)
TNO	The Netherlands Organisation for Applied Scientific Research
UC	Use Category
UDS	Unscheduled DNA Synthesis
UN	United Nations
UNEP	United Nations Environment Programme
US EPA	Environmental Protection Agency, USA

UV	Ultraviolet Region of Spectrum
UVCB	Unknown or Variable composition, Complex reaction products of Biological material
vB	very Bioaccumulative
vP	very Persistent
vPvB	very Persistent and very Bioaccumulative
v/v	volume per volume ratio
w/w	weight per weight ratio
WHO	World Health Organization
WWTP	Waste Water Treatment Plant
Xn	Harmful (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
Xi	Irritant (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)

Appendix A: Life Cycle of TDCP - Supporting Information

Information in this appendix was originally presented in Section 2 of the risk assessment. For purposes of readability, it has been removed to this appendix to make section 2 more concise.

In general it is assumed that the reader has already studied the relevant section(s) of the main RAR. Sources cited in the text are referenced in full in the main reference list.

1 FLEXIBLE FOAM PRODUCTION

Slabstock foams[19]

Polyurethanes are step addition polymers made by reacting isocyanate compounds with compounds containing active hydrogen groups, usually hydroxyl groups, on the ends of long polyether or polyester chains. The isocyanate groups can also react with water to form carbon dioxide and this reaction is used as the principal source of gas for blowing the foam, as well as a source of heat for the expansion and curing of the foam. Other blowing agents may also be added to the foam, sometimes via formulation with the polyol. The density of the foam can be progressively reduced by increasing the water content of the formulation and adding sufficient isocyanate to react with it. This also leads to a stiffening of the polymer and so the density of the foam can be reduced without greatly reducing the load-bearing properties of the foam. However, the exothermic heat of reaction effectively limits the amount of water in the formulation to about 4.6-5.5 parts of water to 100 parts of the polyether polyol, depending on the scale of manufacture, rate of heat dissipation, amount of excess isocyanate present and many other factors.

Since the foam product is a good insulator, overheating of the foam can sometimes occur due to the heat release from reactions during its production and/or curing (for instance excess isocyanate in the foam could react with atmospheric moisture during curing, releasing heat). In some situations, the temperature of the interior of the foam can rise until the polyether chains begin to oxidise and produce more heat. In extreme cases, the foam may spontaneously ignite. The first sign of overheating is the formation of a yellow-brown discoloration in the centre of the foam. Typically, antioxidants are added to the polyether polyols used in flexible foam production to minimise these "scorch" effects (Woods 1982 in EC 2000). The most common type of halogenated flame retardants used in polyurethane foams appears to be halogenated phosphorus based chemicals. However, these types of flame retardant can contribute to scorch problems, particularly in some low density flexible foams.

Flexible polyurethane foams can be manufactured in continuous or batch processes, with cross-sections of up to about 2.2 m wide by 1.25 m high. In a typical process the initial ingredients (mainly water, isocyanate, polyether polyols and any other additive such as a flame retardant) are mixed together at around 20^{o}C and placed into a mould. There then follows an induction period ("cream time") before bubbles appear and the foam begins to rise. The maximum temperature in the system occurs 30 minutes to 1 hour after the end of the foam rise, with the internal temperature remaining near this maximum temperature for 1-8 hours, depending on the block size. In typical low density foam, the temperature of the interior could be around 160°C. The foam is then left to cure for around 48 hours (Woods

[19] The majority of the description of foam production presented in this section is taken from the risk assessment for pentabromodiphenyl ether (EC, 2000).

1982 in EC, 2000). The blocks may for example be up to 60 meters long or alternatively they may be cut down to lengths of about 2 metres (HMIP, 1995).

Slabstock foam is usually made by continuously metering all the foam reactants to a mixing head, where they are mechanically mixed and immediately applied to the bottom lining of a continuously moving trough formed by a horizontal bottom paper (or foil) and two vertical side papers (or foils). If the top of the foam is unrestrained, a continuous "domed" block is formed. As the final users usually require foam in sheets of uniform thickness, a domed top is often undesirable as it increases the amount of scrap foam during trimming. Several processes are used in order to reduce this effect such as: a) constraining the rise of the foam by using a paper or foil on the top of the mould; b) distributing the foam mixture onto a shaped base plate that allows foam to expand downwards; c) using a vertical process (Woods 1982 in EC 2000).

Continuous foaming machines can produce polyurethane foam at rates up to 500 kg/minute. The density of the foam produced is generally in the range 10-60 kg/m^3, with most being in the range 15-27 kg/m^3 (Woods 1982 in EC 2000).

The foaming section of the process is enclosed within a tunnel fitted with extraction for removal of di-isocyanate vapours and blowing agent emissions (HMIP, 1995).

Moulded foams

Moulded PUR products can be produced from TDI (toluene di-isocyanate) and also from mixture of TDI and MDI (methylene diphenyl di-isocyanate). In polyurethane moulding processes the catalysts and certain other additives may be premixed into the polyol and blowing agents may be added to the di-isocyanate stream. Alternatively, components may be fed separately into multi-component mixing heads (HMIP, 1995).

A PUR mould has to perform the following functions (BASF, undated):

receive and distribute the reaction mixture

maintain the correct reaction temperature and remove the heat of reaction

absorb the reaction pressure

seal against loss of material (flash)

vent air

locate inserts and reinforcing materials

Depending on the properties required in the PUR foam, moulding may be carried out with the application of heat or alternatively under ambient conditions (cold cure process). Industry has indicated that cold cure moulded foam does not contain flame retardants (pers. comm. 31st July 2002, producers and downstream users). Hot cure foams result in lower densities and a higher hardness than cold cured foams.

Hot cure foaming is believed to account for 20 % of flexible foam production and is used in the production of foams for automotive seating, aircraft seating and office furniture. The process is almost universally employed for the production of moulded automotive seating foams.

With hot cure moulding formulations the blowing is by carbon dioxide generated in situ by incorporation of water into the reaction mixture. With cold cure moulding carbon dioxide is

also the normal means of expansion but some formulations may also employ a volatile organic compound as a secondary blowing agent (HMIP, 1995).

Predetermined quantities of mixed reactants are automatically or manually dispensed discontinuously into moulds, which may be stationary or continuously circulating on a track (HMIP 1995 and BASF undated). The moulds are normally temperature conditioned prior to filling (HMIP, 1995) to around 40 ^{o}C.

After the mixture of reactants has been dispensed, the lid of the mould is closed and foaming takes place. Alternatively the mixture is injected into a closed mould with defined vents. With cold cure formulations the foam becomes tack free at ambient temperature. With hot cure moulding, the moulds are heated to temperatures typically in the range 150,^{o}C to 230,^{o}C (HMIP, 1995).

Moulding allows inserts and fabrics, for example, to be added at the moulding stage to form an integral part of the moulded product. Also, components containing more than one foam composition, such as car seat cushions, can be produced by dispensing different formulations into different parts of the mould (HMIP, 1995).

On completion of the curing cycle, the moulds are opened and the moulded shapes are removed for trimming and finishing. Some moulded items are subject to a crushing stage or vacuum treatment in order to break open the closed cells in the moulding. The crushing operations may lead to the release of volatile compounds such as amines from within the cell structure of the foam (HMIP, 1995).

After removal of the moulded article the mould is cleaned by removal of residual foam material from the lid and from vents, etc. The mould is then treated with a mould release agent such as a wax, which may be in organic solvent, or in aqueous dispersion (HMIP, 1995).

Polyether versus polyester foams

Slabstock foam exists in both polyether and polyester form, depending on the nature of the polyol used (i.e. polyetherols or polyesterols). Polyether foams differ from polyester because of their greater flexibility and their homogeneous density. Polyester foams are more brittle and generally more difficult to produce than polyether foams (EC, 1997).

There is a large variety of polyether and polyester foams that serve several applications. In general terms two main branches can be identified, being comfort polyether foam for the furniture and bedding industry, and technical foam (mainly in polyester form) for various industrial purposes (EC, 1997).

Polyether PU foam is a standard commodity product, sourced by customers depending on price (EC, 1997). Foam production plants are generally located close to their markets, as the product's high volume and low weight do not allow for economic transport over long distances (EUROPUR, 2002). Technical foam can be further subdivided into three categories, according to the complexity of the technology involved in the manufacturing process, uses and price differences (EC, 1997). These are reported in **Table A.1**

Table A.1 Categories of technical foam

Category	Application	Comments
Conventional ester	Mainly used in the clothing and packaging industry and automotive trims.	Does not require significant technology. Produced by nearly all ester foamers. Constitutes the larger part of the technical foam market.
Speciality ester	Generally used in consumer goods such as sponges, painting rolls and sealing uses in automobiles	Involves greater technology and know-how to produce.
Post-treated foams	Mainly used for filtration purposes (building, households and automotive) and acoustics.	Encompass both polyether and polyester foams. Foams undergo additional processes, such as reticulation, impregnation and densification. Post-treatment processes require substantial technology and know-how.
Source: EC 1997		

Industry reports that TDCP is used mainly in polyester foam (Pers. comm. 16th October 2001). Rhodia (2000) primarily recommends TDCP for use in polyester foams, but notes that it will also function in polyether and moulded products. For example it may be used at higher loading rates in polyether foams where scorch is observed with conventional diphosphate type additives.

2 RECYCLING OF PUR FOAMS

The European Diisocyanate and Polyol Producers Association (ISOPA) has produced a number of publications on PUR recycling and recovery. Two publications from the mid 1990s summarise the desirability and status of the various technologies at that time:

> Evaluating the Options (ISOPA 1994): describes PUR uses, identifies possible recycling options and evaluates these using a multi-criteria scoring and weighting technique. For a given use, options are rated as of high, average or low desirability or of no relevance; and
>
> Options in Practice (ISOPA 1995): reports on the extent to which the technology options for PUR recycling are available and used in practice. For a given use, identifies whether options are commercially available, developmental or still in a pilot stage.

A description of the range of PUR recycling options currently available is given in **Table A.2** Further information on recycling for automotive and furniture applications is given in sections 2.2.2.1.4, 2.2.2.1.5 and 2.2.2.1.6 of the main risk assessment report.

Table A.2 PUR recycling options

Option	Description
Re-use	Reusing the same piece of PUR for the same or a similar application. Some use across the range of applications e.g. second hand furniture, sale of cars seats by dismantlers, re-use of sandwich panels on building sites
Rebonding	Rebonding chopped flexible PUR foam into new products together with a polyol/di-isocyanate. Mainly for scrap foam generated during the cutting of slabstock foams. Used in office furniture, low-end grade furniture, sound insulation in cars, carpet backing, high-density mattresses. See section 2.2.2.1.4 of the main risk assessment report (ISOPA 2003, Bürgi, D., (BAG), (2002)).
Loose crumb	Flexible PUR foam is shredded but not reformed. Mainly for scrap foam generated during the cutting of slabstock foams. Main use in the EU is for garden furniture (see section 2.2.2.1.4 of the main risk assessment report, also ISOPA 2001a).
Adhesive pressing	PUR is granulated and blended with 5% to 10% polymeric MDI and formed into boards/mouldings at temperatures up to 200°C and under pressure (20 to 200 bar). Products are finished by sawing and sanding or by applying additional facings. Mainly for production trim from rigid block foam and panel production where composition is known. Also for production trim or used PUR from some automotive parts (e.g. thermoformable foam from headliners, flexible integral skin foam from steering wheels, flexible foam backed car carpets). Main applications are furniture in kitchens and sailing boats because virtually unaffected by water, also for flooring e.g. in gymnasiums which needs to have a certain elasticity (see ISOPA 2001b).
Use of particles	Oil binders: PU powder and larger particles obtained from cutting and shaping rigid foam for building and construction applications in the factory are used to absorb spilled liquids. Includes production of pressboards for use in windy conditions and hoses containing particles for use in containment of spills on water (see ISOPA 2001c). Insulating mortar: particles of rigid foam production scrap from building and construction applications are one of the main raw materials in insulating mortar used on construction sites for thermal and acoustic insulation (see ISOPA 2001c)
Regrind/ Powdering	PU foam scrap is ground into fine particles (0.05mm to 0.2 mm) and added as a filler to virgin systems in the production of PUR foam. Can be used for production trim or post consumer parts. Technologies in development (see ISOPA 2001d).
Chemolysis	PUR molecules are broken down into smaller building blocks for reassembly into polymers suitable for the production of further PUR products. Preferable to process feedstock of known composition to obtain consistent and predictable regenerated products, e.g. production waste. Hydrolysis: PUR reacted with water under pressure at elevated temperature. Process developed up to pilot plant stage. Aminolysis: PUR reacted with amines such as dibutylamine under pressure at elevated temperature. Process at the research stage. Glycosis: PUR reacted with diols at elevated temperatures (200°C) with cleavage of covalent bonds. Processes developed for a range of PUR inputs to pilot and commercial scales. Single phase glycosis is currently applied industrially. For flexible foams it yields polyols which can replace up to 90% of the virgin polyols in semi-rigid foams, bringing the recycled content of "old" foam in the "new" foam to 30% (see ISOPA 2001e)
Feedstock recycling	For PUR in mixed waste streams. Many of the developing technologies are uneconomic at present. Pyrolysis: mixed plastics heated in an inert atmosphere. Liquid and gaseous hydrocarbons formed used as feedstock in other petrochemical processes. Pilot plant in the UK. Gasification: In a two stage process, mixed plastics are heated, then combined with air or oxygen. Product can be used in refinery processes and in production of methanol, ammonia and oxo-alcohols. Likely to be of most interest to PUR. Hydrogenation: plastics treated with hydrogen under high temperature and pressure. Liquid and gaseous hydrocarbons formed are used in refineries and chemical plants. Existing plants for packaging waste streams. Trials for non-packaging waste streams. Steel industry: up to 35% of the heavy oil or coal dust used as a reducing agent in blast furnaces can be replaced with mixed plastics. Operational at a German furnace (see ISOPA 2001f)
Energy recovery	Incineration with energy recovery, mainly in the combustion of municipal solid waste (MSWC). New markets under development, e.g. in power stations where PUR is used as a co-fuel and substitute for coal, as a co-fuel in cement kilns and as a co-fuel for industrial boilers (see ISOPA 1996 and 2001g). MSWC varies across European from around 80% of MSW in Denmark to as low as 12 % in the UK. Option recommended for recovery of rigid foams from demolition (ISOPA 2001b)

Regardless of the recycling technology employed, two factors play a key role in determining the technical and commercial feasibility of recycling polyurethane materials (ISOPA, 2001h):

densification of low density, voluminous PUR foams, allowing for cost-effective transportation from collection point to recycling operation
size reduction of PUR articles (mattresses, car seats, insulation panels, etc.) making them suitable for treatment.

More than 100,000 tonnes of PUR is recycled and recovered each year (ISOPA undated 2), most via the rebonding of scrap from flexible foam production (see sections 2.2.2.1.2 and 2.2.2.1.4 of the main risk assessment report). The majority of PUR is collected as mixed plastic waste or as municipal waste (ISOPA, 1994).

ISOPA (1994) does not give figures for actual recycling levels in Europe and reported that "in the absence of a viable market, incineration with energy recovery … (was then) the most realistic and cost effective recycling option for PUR post consumer waste". Industry has confirmed that foam is still not recycled in large volumes in Europe (Pers. comm. 16/10/01).

The Rebonding Process – further information

Bonded foam, or rebond, is a moulded polyurethane product made from pieces of shredded flexible polyurethane foam, held together with a binder. Foam pieces from various sources - production trim and post-consumer waste - can be suitable for rebonding, although in practice production trim and cuttings are by far the most commonly processed (ISOPA, 2001a). Rebonding is not relevant to moulded foams as the foam is pre-formed and thus not cut.

Granulators and flock-mills are normally used to shred the foam into pieces approximately one centimetre in diameter. There are other technologies available to handle large foam pieces by cutting them into very thin strips, which can then be reduced into smaller pieces (ISOPA, 2001a). This type of process is deemed to be 'dust-free'. In the UK, modern equipment is of the 'turbine cutting' type, which produce particles of a controlled size and are designed to minimise production of dusts, which are in themselves a fire hazard. Some older types of equipment shred the foam by tearing, and produce more dust. This is commonly removed by air filters and disposed of to landfill; however, FR-containing foam is not processed by this method (Pers. comm. 29th April 2004).

The rebonding technologies used vary according to the market requirements and the final use of the rebond articles. Rebonding of polyurethane foam can be carried out through either batch or continuous moulding. The foam blocks are further processed to fabricate parts and articles, resulting in trim that in turn can be reused in the process. Rebonding is also applied in the moulding-to-final-shape technology which allows processors to optimise material use and cost (ISOPA, 2001a).

Use of Rebonded Foam – further information

A number of reports make reference to current levels of rebonding in Europe, and all provide different information:

more than 40,000 tonnes of bonded foam were produced in Europe in 1999, of which more than half was associated with flooring applications. A further 60,000 tonnes of scrap foam (production waste) was sent to the USA for carpet underlay. There is a trend towards lower export from Europe to the US (Mark and Kamprath, 2000);

world-wide, about 400,000 to 500,000 tonnes of foam is recycled on a yearly basis. In Europe that figure is of the order of about 60,000 tonnes (EURO-MOULDERS 2002);

an estimated 80,000 tonnes of PUR in the form of process trim is currently collected in Europe for further use (ISOPA 1994);

up to 50 000 tonnes of rebonded foam are processed each year in Western Europe (ISOPA 2001a); and

scrap foam is often recycled into carpet underlay (rebond), particularly in the United States. The EU is an exporter of scrap foam (around 40,000 tonnes/year) to the United States for this use (ENDS 1998 in EC 2000).

Overall, between 40,000 and 80,000 tonnes of scrap foam is rebonded in Europe each year with a further 40,000 to 60,000 tonnes shipped to the US. However, discussions with a UK cutter indicate that the situation at present is somewhat different, the US market being "pretty closed" at the current time. Some of this scrap foam will contain TDCP.

Scrap foam sent to the US is used to make 'rebond', a carpet padding used between carpet and hard flooring surfaces such as concrete and wood. The carpet rebond is not attached to the carpet, thus the padding (rebond) is a separate material from the carpet itself. Carpet is laid over the rebond to provide a cushion effect and helps in minimising carpet wear (RPA 2000). Scrap foam exported to the US will include some foam that contains TDCP. Traditionally in the EU foam-backed carpet (latex) and latex underlay is used. It is understood that carpet rebond is not imported into Europe and thus this will not affect exposure to TDCP in the EU.

3 AUTOMOTIVE USE: USE A

Production and use

TDCP is a useful flame retardant for automotive uses on the basis of its low volatility and hence low fogging potential. 'Fogging' is the condensation of substances used in vehicle interiors onto glass following volatilisation. It also offers good resistance to migration following moderate heat or humidity ageing (Rhodia, 2000).

TDCP is typically recommended for the production of flame retardant foam required to resist ignition from low intensity flame sources such as those described in Federal Motor Vehicles Safety Standard No. 302 (Rhodia, 2000). This is the accepted standard for the interiors of motor vehicles in the United States. This states that, for individual components, the rate of flame spread must not exceed 101.6 mm/min. This is a small-scale test regulated by the US Department of Transportation. This is also the standard recommended in the UK Society of Motor Manufacturers and Traders' (SMMT) TEC 811/1989 guideline. However, there is a UN standard which requires only 254 mm/min (RPA, 2000).

1n 1997 alone, more than 300,000 tonnes of PUR were used in applications in Western European cars. A typical car of 1,000 kilograms (kg) total weight contains 100 kg of plastics, of which about 15 kg are PUR. The main applications for PUR are: seat foam (7 kg per car), cushion overlay (fabric backing), carpet backing, door panels, sound absorption and vibration dampening, dashboards, steering wheels, bumpers, energy absorbers, headliners, airbag covers and window encapsulation (ISOPA and EURO-MOULDERS, undated). However, not all PUR car parts will be treated with flame retardants.

Industry reports that TDCP is also used in seats used in public transport. A range of different flame retardants are used in such seats, with transport companies having their own standards.

In this regard TDCP is not used in the London Underground as this organisation has a policy of no halogens in order to minimise smoke production (Pers. comm., 16th October 2001).

ISOPA data indicate that 100 foamers/moulders are involved in the production of automotive products from PUR foam in Europe each year, consuming 365,000 tonnes of polyurethane (ISOPA undated), however, only three or four European producers of moulded foam use flame retardants (pers. comm. 31st July 2002, producers and downstream users).

End of Life – Current Situation

The following discussion of current and future levels of recycling of automotive PUR is taken from Mark and Kamprath (2000) unless stated otherwise. This study presents data on conditions in Germany but indicates that other countries in Europe e.g. the Netherlands have somewhat similar economic and market conditions.

Most cars at the end of life are delivered either to car dealers, where old cars are traded for new ones, or they may be delivered directly to an officially recognised dismantler or scrap dealer. At present very little dismantling takes place across the EU. The current situation in Germany and in many other countries, where there is no external funding for dismantling from the consumer, means that parts removal is not cost effective. Therefore only batteries and well functioning spare parts tend to be removed from cars.

Only in the Netherlands and Italy are small amounts of plastics and PUR currently removed from cars, with activities in the Netherlands being subsidised by the first owner of the car. For example in 1998, Auto Recycling Netherlands recovered 2,200 tonnes of PUR from the dismantling of seats (3 % of the 70,000 tonnes of PUR available for recovery). This material was sent for recycling. Some scrap is used in the production of new parts for cars. For example, in the BMW 5, recycled polyol from glycolysed scrap is used in the manufacture of the warm air duct (Clausius, undated).

In the vast majority of cases therefore, PUR seats remains in the end of life vehicle (ELV), which is sold to shredders for further processing. There are around 50 shredders in Germany.

After separation of the metal fraction of the shredded hulk, about 200 kg of ASR (automotive shredder residue) remains at the shredder site. The total ASR volume in Europe is a minimum of 1.5 million tons per year out of 6.7 million ELVs in Europe and about 200,000 tons out of 1.3 million ELVs in Germany.

Most ASR is currently disposed to landfill. There are many potential recovery operations for ASR but only recovery in municipal solid waste combustion (MSWC) is currently in use. Less than 70,000 tons of ASR (just under 5 % of the 1.5 million tons per year) is used for energy recovery. This involves waste combustion to generate medium pressure steam (40 bar) used to drive a turbine for electricity generation, or to provide medium to low pressure steam in district heating and industrial processes. An alternative source (pers. comm. 11th February 2003) suggests that incineration of ASR for energy recovery is widespread, and that it is only disposed of to landfill 'in exceptional cases'.

End of Life – Future Situation

The recycling and recovery of polyurethane and other car components is the subject of the End of Life Vehicles Directive 2000/53/EC. This came into force on 18th September 2000 and was to be transposed by Member States by March 2002. The Directive is intended to reduce the amount of waste arising from the scrapping of vehicles. It targets overall re-use,

recycling and recovery rates at 85 % by average weight per vehicle by 2006 and 95% by 2015, and to increase the rate of re-use and recycling over the same period to at least 80% and 85% respectively by average weight per vehicle and year. Another requirement of the Directive is for vehicle manufacturers to design products and cars with recycling and re-use in mind: expressed in the so called Type Approval of new vehicles as from 2005 (EURO-MOULDERS, 2002), with the need for a minimum 95 % of components of new vehicles to be reusable/recoverable (pers. comm. 11th February 2003).

The result of this is that systems will need to be set up to ensure that end of life vehicles (ELVs) are collected into approved dismantling chains and that improved treatment standards will be established for vehicle dismantlers and scrap dealers to meet (EURO-MOULDERS, 2002).

PUR seating is one of the large plastic parts in an ELV and it can be relatively easily dismantled. Thus it is one of the key targets for legislators and environmental authorities for dismantling (Mark and Kamprath, 2000). Future options for the recovery of automotive PUR are:

as a fuel in the production of cement or lime, or in the steel industry

rebonding

regrind/powdering

chemical recycling, e.g. glycosis

feedstock recycling, e.g. gasification

recovery in municipal solid waste combustion (MSWC)

All bar the last two options require dismantling of the seat cushions.

Removal of the cover textiles, plastics and large metallic parts inside the seat module and shredding the foam to 5-10 cm pieces would allow use in cement kilns for secondary firing as a fuel replacement[20]. This option is not currently cost effective due to current low fuel prices. For use for primary flame fuel firing in cement kilns, the seat foam would need to be shredded to form <2 cm fluff. This would require total dismantling of the seats and full separation of non-PUR materials. This option would be more costly and not economic as cement producers do not have lower gate fees for primary fuel versus secondary fuel replacement.

The use of PU seating as a fuel in steel (pig iron) furnaces is being seriously considered in Japan and studied in North America. However, the relatively high gate fee and additional treatment cost compared to MSWC make this route less attractive than that option.

Rebonding (see section 2.2.2.1.4 of the main risk assessment report) is widely used for scrap foam from slabstock foam production. It is estimated foam from ELVs could produce 70,000 to 80,000 tonnes of foam each year, which would double the size of the current EU market. The current market is not considered large enough to absorb this additional tonnage.

In laboratory tests, new moulded foam seats have been made containing 15 % to 20 % reground/powdered foam and exhibiting excellent processing characteristics. The investment

[20] It is assumed that the small steel wires inside the foam cushion would not need to be removed on the basis that tyres are used for secondary firing with the steel cord left inside the tyre.

cost of the first generation equipment limits the operational potential of this technology to slabstock (ISOPA, 1991d). This option is not operating on a commercial scale.

The polyols resulting from glycolyis although of similar costs to virgin materials are not suitable for seat production and can only be used in the production of rigid PUR foams. PUR from ELVs would generate around 200,000 tons of recycled polyols each year, about 50% of the current market for polyols in systems[21] for rigid foam production in 1999 (IAL, 2000). Thus the market is not big enough by far to take this additional input. More generally, viable chemical recycling routes for mixtures of PUR materials from ELV's seats do not exist at present at sufficiently large scale.

ASR can be used as an input for gasification plants that produce methanol via synthesis gas treatments (EURO-MOULDERS, 2002).

Use in MSWC does not require pre-treatment of waste as incinerators can take ASR. Alternatively bales of seat foam can be dropped into the bunker as they are delivered from the dismantler after the baling wires are cut.

The lowest cost option for the future disposal of an ELV is reported to be shredding followed by fuel substitution. Other favourable routes depend on regional circumstances. In general terms, seat dismantling is currently uneconomic and contaminants in the PUR from shredder residue prevent the use of other options such as rebonding. Also, because of the various qualities of the ELV PUR foams used for many years (10 to 15 years) in cars, special and costly cleaning and treatment methods would need to be found to produce recyclates with acceptable and stable characteristics (EURO-MOULDERS, 2002).

As fuel substitution in cement kilns is not currently economic, MSWC is at present the only viable option. It is however viewed by legislators as inferior to other material recycling or recovery routes.

All other use scenarios are described in detail in the Confidential Annex.

[21] See the TCPP risk assessment for a discussion of polyols and systems for rigid foam production.

Appendix B: A new assessment of the release of flame retardants from polyurethane foam

Authors: Peter Fisk, Louise McLaughlin, Ros Wildey

This report was prepared by Peter Fisk Associates, largely under contract to the Environment Agency, as part of three environmental risk assessments being carried out under the ESR programme. Some parts were conducted independently by Peter Fisk Associates.

1 Introduction

The context of this report is the Existing Substances Regulation (ESR) risk assessments of the substances TCPP, TDCP and V6; its purpose is to review measured data supplied by industry and from the literature, which can help assessment of the rates of release of substances from a polyurethane (PUR) matrix. There are several complex areas of application of the data for the environmental risk assessment. There are various laboratory or simplified tests of release, and taken together at face value they do not reach an immediately obvious consistent set of conclusions. Therefore, in order to aid interpretation it has been necessary to develop a mathematical model of how fast additives are lost from polymer matrices, applied to polyurethane in particular. In order to achieve this objective it has been necessary to draw upon a somewhat wider set of source literature than that on PUR alone.

The proposed areas of application for the model are discussed below. The starting point of this study is the description of flame retardant releases in the Emission Scenario Document (ESD) for Plastics Additives (OECD, 2004).

The draft ESR risk assessments contain much of the background, and that is not repeated here. Losses from foam are relevant to the following processes identified to date:

Foam production and storage
Foam processing, recycling
In-service loss
Waste remaining in the environment
Release from foam within landfills (where degradation of the polymer may also be important).

The above life cycle stages are also described in the ESR assessments of several brominated diphenyl ethers, although the extent of information now available, and the higher tonnages of the present substances in use means that the present treatment and these older ones are not identical, although broadly compatible.

The structure of this document in the subsequent sections is:

Review of measured data
A new mathematical model
Conclusions for the ESR RAR developments.

Some of the more detailed data and arguments are developed in Sections 2 and 3. The key findings for the current risk assessments are given in Section 4.

Whilst the models developed are based on a number of assumptions, and there are developments that would be necessary for a more complete picture, the work brings together several studies into a consistent whole, sufficient for the present purpose.

The authors are grateful for useful comments from Environment Agency and industry reviewers, and from Professor Gary Stevens of the University of Surrey.

2 SUMMARY OF MEASURED DATA

Polyurethane foams intended for use in construction or furniture are frequently treated with flame retardants (FRs), including TCPP and TDCP. Typical applications of this type of foam are insulation panels, one or two-component spray foams for professional or consumer use (e.g. for *in situ* application to roofs or as fillers), mattresses and upholstery foam, including for automotive applications.

During the storage, handling, service life, recycling and disposal of such foams, it is possible that the FR may be released due to diffusion through the polymer, followed by volatilisation or washing from its surface. For the purposes of risk assessment, it is important to quantify these releases in order to determine exposure to both humans and the environment. The main focus of this document is the environment, although the emission rates described could be used to estimate human exposure.

Several studies have been published relating to both flame retardant levels in indoor environments and the measurement of releases from various polymers, including polyurethane. Details of some key studies relevant to releases of TCPP and TDCP from foam are summarised in Section 2.1, and the results are discussed in Section 2.2. A brief review of studies relating to indoor measurements is given in Section 2.3.

When a fresh piece of foam is used in a study, such variables as air flow rate, foam size, chamber size affect concentrations measured in the air and on the walls of the chamber, and remaining in the foam. There might typically be a rapid loss rate as the outer surface of the foam loses flame retardant and as the receiving environment becomes saturated; thereafter the rate may slow. These factors are explored in more detail through this report.

2.1 MEASURED RELEASES FROM FOAM

2.1.1 BAM study

Researchers at the Federal Institute for Materials Research and Testing (BAM), funded by the Federal Environmental Agency in Germany, conducted chamber tests on different types of polyurethane foams, circuit boards and computer equipment (UBA, 2003). Sample materials were placed in either glass or stainless steel chambers under conditions that modelled real-life situations. Clean, dust-free air was passed through the chamber at a rate equivalent to 0.5 air exchanges per hour, at a temperature of 23 °C and relative humidity of 50 %. Sample sizes were selected such that the emitting surface area to chamber volume ratio modelled typical use patterns.

Emissions of TCPP to air were sampled via a pre-purified polyurethane foam plug fitted to the chamber air outlet. The foam plugs were extracted with acetone using ultrasonication and

analysis by GC-MS was used to determine TCPP concentrations in the extract. In addition, at the end of some tests the chamber walls were rinsed with acetone and any losses of TCPP due to sink effects (condensation onto the chamber walls) were determined by GC-MS. The limit of detection was reported as 17 pg/µl and the limit of determination 55 pg/µl.

Three types of foam were tested, namely rigid insulation foam, rigid assembly foam and flexible furniture foam. Assembly foam is that which is used for adhesive/filling uses, referred to in the RARs as 1K. Within each group, other conditions such as foam density, FR (flame retardant) loading rate, ratio of emitting surface area to chamber surface area (source to sink ratio), and coverings were varied. TCPP was detected in all cases and the findings are summarised in **Table B.1**. Note that it appears that **Table B.1** contains original FR % b.w. concentrations that may have been supplied by manufacturers rather than determined by BAM for the sample sets they actually used. If this is the case there will be uncertainty in relating the release rates to the notional original concentrations. It was found that the air concentrations increased at the start of the tests, then reached a plateau air concentration or decreased slightly before the steady state concentration was reached. This concentration profile may be explained by the sink effect, where a certain time is required before equilibrium between air and the chamber walls is reached, or it may be due to migration of TCPP to the foam surface. A plateau air concentration also reflects saturation of the vapour phase, with a dynamic equilibrium between TCPP in the air on the surface of foam, and on the walls of the chamber.

Results were calculated as area-specific emission rates (SER), either on the basis of the equilibrium air concentration and area-specific air flow rate, or using the total amount of TCPP detected from both the air and chamber walls. Where there is close agreement between the two results, the test system is considered to be in equilibrium.

The observed emission rates were 0.3 to 0.7 µg $m^{-2}h^{-1}$ for insulation foams, 40 to 70 µg $m^{-2}h^{-1}$ for assembly foams, 36 to 77 µg$m^{-2}h^{-1}$ for upholstery foams and 12 ng $m^{-2}h^{-1}$ for a mattress.

Due to the variation in sample types and conditions used in the experiments, it is not possible to make direct quantitative comparisons between them. However, the researchers reached the following conclusions:

> In the test with insulation foams, a distinct sink effect was noted, with 25 and 33% of the total emitted TCPP being found on the chamber walls at the end of the test. Increasing the source to sink ratio was shown to reduce this effect since the measured equilibrium air concentration was higher when the source to sink ratio was increased for the Insulation I foam sample (PIR insulation foam welded in polyethylene foils, density 30 g/l). The higher concentrations in air are approaching theoretical upper limits based on the vapour pressure (202 000 ng/m^3), so it is not surprising that there would be some condensation onto any available surface.
>
> The increased emission of TCPP from the insulation foam with the smaller density is due to an increased interface between the polymer phase and air.

Table B.1 Results of BAM 2003

+ Based on total emission measured from PUR plug and walls of test vessel.

Sample	Density (g/l)	% TCPP *	Area-specific air flow rate ($m^3 m^{-2} h^{-1}$) Q	Source:Sink ratio (m^2/m^2)	Maximum Air Conc (ng/m^3)	Time to reach maximum (days)	Eqbm Air Conc (ng/m^3) Ceq	Time to reach equilibrium (days)	Overall Area-specific emission rate+ ($\mu g\ m^{-2} h^{-1}$)	Area-specific emission rate Ceq.q ($\mu g m^{-2} h^{-1}$)	Sink effect (%)
Insulation I	30	5	1.243	0.28	800	~37	480	~50	0.70	0.60	25
Insulation I	30	5	1.243	0.40	1800	~35	780	50 – 60			
Insulation II	80	2.5	1.243	0.28	250	~35	170	~50	0.35	0.21	33
Assembly I	20	14	5.12	0.067	15000	~12	3000	~75	40	16	NR
Assembly II	25	14	5.12	0.037	15000	~12	3000		NR	NR	NR
Assembly III Smooth New	NR	18	5.12	0.037	10000 - 15000	~10	10000 - 15000	~10	NR	50	NR
Assembly III Smooth Old	NR	18	5.12	0.037	9500	~10	9500	~10	70	50	NR
Assembly III Sawn New	NR	18	5.12	0.037	10000 - 15000	~10	10000 - 15000	~10	NR	70	NR
Assembly III Sawn Old	NR	18	5.12	0.037	26500	~10	26500	~10	130	140	NR
Upholstered stool	NR	9	1.24	0.40	45000	100	41000	150	28	36	NR
Mattress	NR	2	1	0.21	100	10	10	20	NR	0.012	NR
Upholstery foam	27	2	1.1	0.13	70000	< 5	70000	< 5	NR	77	NR

*Nominal values based on manufacturing information for the foam samples.

NR – Not reported.

Insulation I: PIR insulation foam welded in polyethylene foils, density 30 g/l

Insulation II: PIR insulation foam welded in polyethylene foils, density 80 g/l

Assembly I: B2 PUR assembly foam with sawn surface, density 20 g/l

Assembly II: B2 PUR frame foam with sawn surface, density 25 g/l

Assembly III: I-C-PUR express pistol foam in aluminium form and either left smooth or cut off to give sawn surface. Tested immediately and after storage for 6 months

Upholstered stool: Upholstery foam covered with fabric

Mattress: Soft PUR foam inside fabric fleece and textile cover

Upholstery foam: Polyether-based PUR foam, uncovered

In addition to the higher TCPP content, the markedly increased polymer/air interface in the assembly foams results in substantially higher emission rates than for insulation foams. This effect of increased surface area was further demonstrated by testing a one component assembly foam with both a smooth and sawn surface. When new, there was no significant difference between the two. However, after storage for six months, emissions were greater for the sawn foam. No explanation was given for the difference between new and aged foams.

The presence of upholstery fabric appeared to increase the time required for the system to reach equilibrium, and was considered to be the reason for the difference in emission rate between the upholstered stool and the uncovered foam. No explanation was offered for the significantly lower emission rate from the mattress, but the same effect can be assumed to operate.

Further chamber tests were conducted using computer equipment, two typical workstations comprising a PC, keyboard, mouse and a single printer and monitor. Test conditions were the same as for the foam tests. TCPP was detected in emissions from one of the workstation tests at levels comparable to the other flame retardants present. The presence of TCPP was contrary to the manufacturer's data and was attributed to an unknown source of contamination, possibly packaging.

2.1.2 Elastogran study

In this test, a concrete plate was covered with a 10 cm thick layer of a rigid, closed-cell two-component spray foam, intended for indoor insulation purposes, containing 9% TCPP. The sample was placed in a test chamber with a surface area to volume ratio of 1.4 m^2/m^3, and the test conditions were 23°C, 50% relative humidity and 0.5 per hour air exchange rate, as for the mattress test. Volatile emissions were collected on Tenax TA and analysed by GC-MS. The limit of detection was reported as 1 $\mu g/m^3$. TCPP was not detected.

2.1.3 EUROPUR study

Chamber tests were conducted on behalf of industry, provided to the authors via Elastogran, sponsored by EUROPUR (EUROPUR 2001, later published in Cellular Polymers, 22 (4), 2003, although that later reference has not been reviewed). Three types of flexible PUR foam used in mattresses were tested. The samples were 2000 x 1000 x 120 mm of full depth foam (i.e. no springs), were uncovered and were reported to contain TCPP at the high end of the typical level for this application (reported to be 2.5 – 14 %, 7 – 8 % on average, based on industry data collected for the risk assessment of TCPP).

The mattresses were placed in a 3.2 m^3 test chamber at 23°C and relative humidity of 50%, with an air exchange rate of 0.5 per hour. Volatile emissions were collected on Tenax TA absorbent and analysed by GC-MS. The limit of detection was reported as 2 $\mu g/m^3$. Results are summarised in **Table B.2**.

The CME 33 mattress gave a measured steady state air concentration of approximately 16 $\mu g/m^3$ after 48 hours, while the measured air concentration from the HR mattress was continuing to decline at the end of the 160 hour measurement period, indicating that steady state had not been reached.

Table B.2 Summary results of EUROPUR (2001)

Mattress Type	Air Concentration (µg/m³)				
	24h	48h	72h	120h	160h
HR[1]	6.0	22	25	19	10
CME 33[2]	9.1	16	16	19	17
CMHR[3]	1.8	1.7	2	<1	<1

[1]HR = High resilience foam, 36 kg/m³, 1.5% TCPP
[2]CME = Combustion modified ether, 33 kg/m³.
[3] CMHR = Combustion modified high resilience foam, 35 kg/m³

2.1.4 BRMA study

A study of long-term flame retardant retention in foams was organised by the British Rubber Manufacturers' Association (BRMA, 1998 – 2005). Over a period of nearly eight years, six monthly samples of two flexible foams manufactured by Company A (containing TDCP) and Company B (containing TCPP) were analysed for total phosphorus and total chlorine content. Details of the method of analysis are available but not reported here.

A further test was carried out with separate foam samples, aged at 80°C for only 100 hours.

The pieces of foam were cushion-sized (47 cm x 47 cm x 20 cm) and stored uncovered in a general factory area, supported underneath. The results of the two test series are summarised in **Table B.3**.

Table B.3 Summary results of BRMA trial

Time (months)	Company A (TDCP)		Company B (TCPP)	
	% P	% Cl	% P	% Cl
0	0.75	2.6	0.40	1.3
80°C for 100 h	0.74	2.5	-	-
6	-	-	0.39	1.7
12	0.74	2.5	0.41	1.4
18	0.75	2.7	0.40	1.2
24	0.70	2.7	0.39	1.3
30	0.72	2.7	0.37	1.3
36	0.71	2.6	0.39	1.3
42	0.73	2.6	0.40	1.2
48	0.72	2.6	0.40	1.2
54	0.74	2.5	0.41	1.2
60	0.73	2.4	0.42	1.2
78*			0.44	1.42
84*			0.45	1.42
90			0.44	1.48

* Change of analytical laboratory

The conclusion in each test report, on the basis of these results, is that flame retardant retention in the foams is very good. Whilst this is evidently true, the method used is insufficiently sensitive to detect small losses and there is no need to convert the concentrations into total TCPP, at least at this point. The % P and % Cl values show, relative to time 0, a range from a loss of <1.5 % of TCPP /year to a gain of 1 %/year, so it is not possible to apply the values with confidence. The overall data set suggests very low losses. It is an important study in that it is both long term and used direct analysis of foam of typical size.

2.1.5 Consortium-sponsored study

On behalf of an industry consortium, a program of research has been undertaken by the Polymer Research Centre at the University of Surrey and the Bolton Research Institute (Univ. of Surrey, 2005). The purpose of this research was to develop realistic exposure models for the release of flame retardants from products, suitable for use in human health and environmental risk assessment. Phase 1 of the research, examining flame retardant release from foams, was published in February 2005.

Releases were measured using several methods under a variety of conditions relevant to human and environmental exposure:

Weight loss following thermal ageing at room temperature, 40 °C and 60 °C.
Analysis of flame retardant content following solvent extraction of foam aged at 60 °C.
Analysis of flame retardant emissions in aqueous media designed to model dermal absorption (contact blotting tests) and chewing (head over heels tests).
Measurement of volatile emissions during thermal ageing in sealed vials.
Measurement of particle size distribution in the pounding test using samples of aged and un-aged foams.

Experiments 1, 2, 4 and 5 are relevant for estimation of volatile releases during storage and service life for the purposes of risk assessment. Experiment 3 (not discussed herein) could have relevance to contact of foam with any liquid medium. Experiment 5 (pounding tests) could be used to assess the loss of particulates due to wear and tear during service-life.

Three types of foam were tested:

A combustion modified (CM) ether foam containing 8.47% by weight TCPP
A combustion modified high resilience (CMHR) foam containing 5.2 % by weight TCPP
An FR ether foam containing 5.5 % by weight TDCP.

Melamine was also present in the TCPP-containing foams.

2.1.5.1 Experiment 1: Thermal ageing

Samples sizes of 100 x 100 x 50 mm ('large') and 50 x 50 x 15 mm ('small') were aged for up to six weeks in:

an air-conditioned laboratory at 20°C and 75% relative humidity;
temperature controlled ovens at 40 and 60°C and ambient relative humidity;

an environmental chamber at 60°C and 75% relative humidity.

The bulk density of the foam tested was ~32 kg/m^3. The oven volumes were 150 or 350 litres, with 10 or 4.3 air changes per hour (considered by the authors to be a relatively fast rate). The foam was positioned on wire with enough space for free air movement to all surfaces. The results are summarised in **Table B.4**.

Table B.4 Percentage weight loss after ageing time of six weeks

	CM Ether Foam – TCPP		CMHR Foam - TCPP		FR Ether Foam – TDCP	
	Large	Small	Large	Small	Large	Small
20°C	0.11	0.26	0.02	0.18	0.11	0.18
40°C	0.44	1.86	0.52	1.47	0.17	0.24
60°C	3.21	7.12	2.18	3.99	0.16	0.17

Rates of loss are higher for the CM ether foam, reflecting the higher FR content. For foams containing TCPP, emissions increase with temperature and were found to obey an Arrhenius relationship; the size of the temperature effect suggests a higher activation energy than would be true for diffusion alone. The dimensions of the foam tested are also important, with higher percentage losses for the smaller block of foam. Results for TDCP were less predictable, but were in general lower than for TCPP, although the difference was small at ambient temperature.

Release rates in the environmental chamber at 75 % relative humidity were lower than for the corresponding oven test. The report attributes this to the higher relative humidity inhibiting diffusion of hydrophobic additives. However, there is no evidence to support this, and other factors, such as different test chamber volumes or air-exchange rates could have contributed.

The result at 20°C is the one of most relevance to the ESR risk assessment.

2.1.5.2 Experiment 2: Solvent extraction of flame retardant from aged foam

Foam samples ('large') were aged at 60 °C for 6 weeks. After ageing, small pieces of foam were cut from the block, extracted and analysed for residual flame retardant. Ten samples were analysed for each foam type.

The flame retardant content of aged foams was determined by extraction into toluene using Soxhlet extraction (over a period of 8 hours). Extracts were analysed by GC-MS. The extraction procedure was validated by spiking a piece of foam without flame retardant with known quantities of TCPP or TDCP. No description of how the spiked samples were prepared is given in the report. Recoveries are reported as 100 – 105.5 % for TCPP and 100 – 111 % of TDCP. However, analysis of un-aged foam samples gave results of 82.6 % of nominal for CM ether foam with TCPP, 102.6 % of nominal for CHMR foam with TCPP and 30 % of nominal for FR ether foam with TDCP. No explanation is given for the low yield of TDCP. It seems possible that the FR could be strongly bonded into the foam in some way, although evidently not irreversibly.

Results were expressed as percentage of flame retardant lost, and as the equivalent weight loss for the piece of foam. Actual weight loss after ageing was also recorded. The results are summarised in **Table B.5**.

Table B.5 Results of FR extraction for thermally aged samples (six weeks, 60°C)

Foam Type	Analytical data		Measured % weight loss of foam
	% of FR lost	Equivalent % weight loss of foam	
CM Ether Foam - TCPP	38.6 39.5	3.3	3.14
CMHR Foam - TCPP	47.6 47	2.4	2.01
FR Ether Foam - TDCP	24.0 13	1.88 0.86	0.36

There is reasonable agreement between the measured weight loss and the flame retardant loss, indicating that most of the observed weight loss is due to flame retardant emission. However, it is expected that a concentration gradient would develop over time, as flame retardant diffuses through the foam block. Since only small pieces of foam were analysed, the part of the block from which they were cut could affect the concentration of flame retardant remaining. Since samples were taken from the inner part of the block, overall losses from the whole block could be underestimated, although because of redistribution within the block this is not a major issue.

Variation in the recovered flame retardant for replicate samples was 40.7 – 64.4 % for CM ether foam, 40.2 – 93.1 % for CMHR foam and 16.6 – 33.9 % for FR ether foam.

The results of Experiment 2 seem to confirm those from Experiment 1, although TDCP loss rates were higher in Experiment 2.

2.1.5.3 Experiment 4: Measurement of volatile emissions during thermal ageing

Samples of foam were placed in septum sealed glass vials and stored in temperature-controlled ovens at 60°C, 40°C and room temperature for a period of 4 months. Headspace samples were collected using a syringe and analysed by GC-MS and sample weight loss was also recorded. The results obtained are summarised in **Table B.6**.

Table B.6 Volatile emissions from thermally aged foam in sealed vessels for 4 months

Temperature	CM Ether Foam		CMHR Foam		FR Ether Foam	
	Weight loss (%)	TCPP Released (% w/w)	Weight loss (%)	TCPP Released (% w/w)	Weight loss (%)	TDCP Released (% w/w)
60°C	1.4	0.26	0.8	0.3	0.2	0.064
40°C	0.06	0.11	0.4	0.059	0.4	0.023
Room temperature	-0.45	$<9.5 \times 10^{-5}$	-0.3	$<8.6 \times 10^{-5}$	-0.25	$<8.9 \times 10^{-5}$

The measured flame retardant release in this case is considerably lower than the recorded weight loss and in the case of room temperature samples, a slight weight increase was observed. The authors attribute this weight increase to possible water absorption. The weight loss at 40 and 60°C is also less than that measured in the first thermal ageing experiment.

The lack of flame retardant detected in the headspace of the vials is attributed to the enclosed nature of the vial leading to re-absorption to the foam. The lack of air flow through the vial means that air saturation would certainly have been reached, thus preventing any further

diffusion from the foam surface. The sample volume used was 50 cm^3 (20 mm x 50 mm x 50 mm) and the vial volume was 73 – 160 cm^3.

In experiments at room temperature no flame retardant was detected above the limit of detection of the analytical method. This is an important finding when considering potential releases from foam used in enclosed areas such as insulation panels.

2.1.5.4 Experiment 5: Pounding tests

This study will not be reviewed in detail. Two foam types, CM ether and CMHR, were subjected to pounding tests using un-aged and aged foams. The diameter of particles emitted from aged foam (30 nm to 0.1 μm) was typically smaller than for the un-aged foam (100 nm to 6.5 μm), and particle size decreased with increasing length of the test. From the available information, it is not possible to relate these results to typical conditions during service life. Further work is being undertaken to characterise the physical and chemical nature of the particles.

Volatile emissions of TCPP were not detected during the pounding tests. This implies a release rate of less than 36 and 10 μg/kg/h for unaged and aged foam respectively.

2.1.6 Losses from very small sized pieces of foam

2.1.6.1 Experimental details

A study (Hall, 2005) was commissioned by the industry to examine the loss of TCPP over time from small particles of polyurethane foam. This study is particularly important as a key to understanding the whole data set so is dealt with in some detail.

A small block of combustion modified polyether urethane foam was received from routine UK manufacture for GC-MS analysis to investigate the loss of TCPP over time. The foam was first analysed for the content of TCPP by extraction with dichloromethane. The foam was then blended into three different particle size ranges and 10 sets of 1 g of each range were weighed into Petri dishes. The samples were left in the open for different time periods of 0, 1, 3, 7, 10, 15, 30, 45, 60 and 90 days. After reaching the allotted time period the samples were analysed for the TCPP content.

The three particle size ranges were:

Dust (diameter less than 1 mm)
Small crumbs (diameter 3 mm to 1 cm)
Large crumbs (diameter 1 to 3 cm).

The crumbs were produced using a blending machine whilst the dust was produced by cooling the foam in liquid nitrogen prior to blending for 2 minutes.

The room where the samples were left measured 310 cm x 370 cm x 290 cm with an archway measuring 98 cm x 207 cm linking to a second room of 290 cm x 370 cm x 280 cm. This gives a total volume of 63 m^3 with a maximum sample loading of 27 g on day 0 reducing by 3 g at each of the sampling periods. There was no air flow monitoring of the room, however the air turnover is believed to be greater than total volume per day. Boards were placed up against the windows to stop light entering, which could affect the foam.

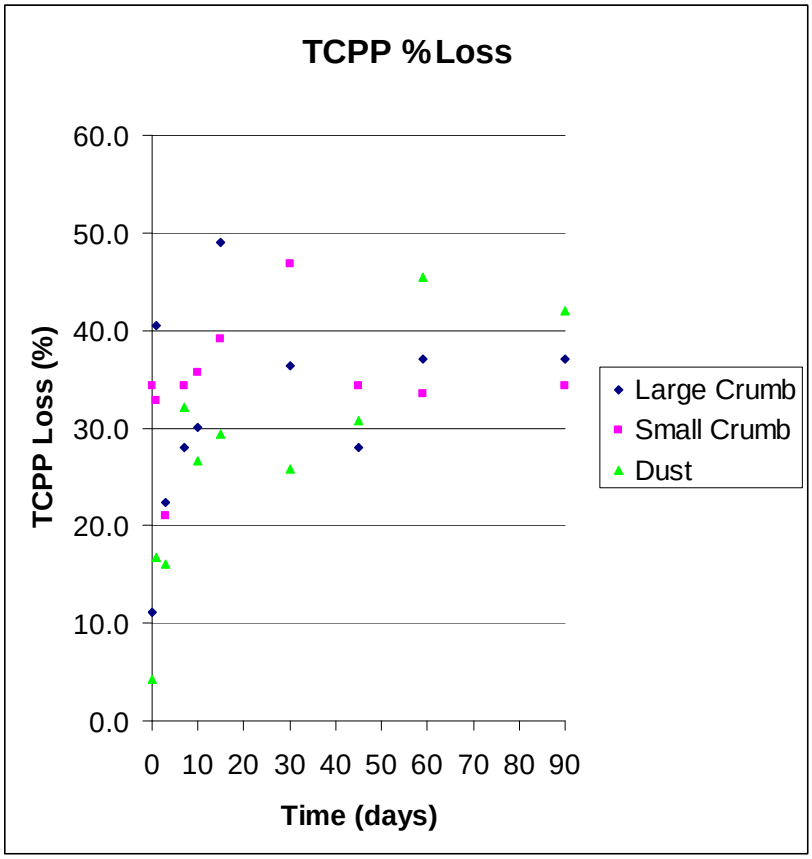
2.1.6.2 Results

Results of the study are presented in **Table B.7** and **Figure 2.1**.

Table B.7 Data for loss of TCPP from three sizes of foam particles

Time (days)	Large Crumb		Small Crumb		Dust	
	% TCPP	% loss	% TCPP	% loss	% TCPP	% loss
	14.3		14.3		14.3	
0	12.7	11.2	9.4	34.3	13.7	4.2
1	8.5	40.6	9.6	32.9	11.9	16.8
3	11.1	22.4	11.3	21.0	12.0	16.1
7	10.3	28.0	9.4	34.3	9.7	32.2
10	10.0	30.1	9.2	35.7	10.5	26.6
15	7.3	49.0	8.7	39.2	10.1	29.4
30	9.1	36.4	7.6	46.9	10.6	25.9
45	10.3	28.0	9.4	34.3	9.9	30.8
59	9.0	37.1	9.5	33.6	7.8	45.5
90	9.0	37.1	9.4	34.3	8.3	42.0

Figure B.1 Graph of loss of TCPP from three sizes of foam particles



2.1.6.3 Interpretation and conclusions

The experiments showed a TCPP loss from the particle size ranges of between 34 % and 42 % at the end of the 90 day period with the general trend being an initial loss of approximately 30 % over the first 10 days and subsequently a slower rate of loss to the final value. The greatest loss was observed in the dust size range with a final value of 42 %, for the large crumb sample a loss of 37.1 % was observed whilst the small crumb sample showed the least final value loss of 34.4 %. Despite some experimental variability, there is a clear trend associated with the results which indicates the dust range samples has a slightly higher rate of loss than the large and small crumbed samples.

There is an initial rapid loss followed by approach to a plateau at around 40% loss. The fact that the release reached a definite plateau, rather than merely slowing, supports the view that release of TCPP had stopped rather than being slowed or limited by some external factor. The rate of air turnover in the experimental system was unchanged and the lack of continued release therefore demonstrates that the plateau was not caused by any saturation effect. The initial rates correlate with particle size (discussed further in section 3). It is possible that rates over the first two days are as high as 20 % per day. Given that only 40 % of the TCPP is available, this could be seen as a loss of 50 % per day of that which is available to be lost.

It is necessary to consider whether there being an 'unavailable fraction' has a physicochemical explanation. It is possible that polar interactions between urethane functions and the flame retardant (FR) will exist. It is also possible that the FR could be physically entrapped. A recent paper, (Levchik *et al.*, 2005) shows that TDCP can react chemically with free NH_2 groups derived from decomposition of the isocyanates used to make PUR. The amount of these forms depends on the precise ingredients used to make the foam. This would be an essentially irreversible process. Therefore, it is reasonable that not all the TCPP was released from the particles used in the study.

2.2 DISCUSSION OF RESULTS

2.2.1 Large pieces of foam

From the information included in the two EUROPUR studies, it is possible to calculate area-specific release rates in the same manner as used by BAM.

For a piece of mattress foam with dimensions 2000 x 1000 x 120 mm, a surface area (A) of 2.72 m^2 was available for emission (i.e. one large face excluded). The chamber surface area was 13.12 m^2, its volume was 3.2 m^3 and the air exchange rate was 0.5 per hour, giving a volumetric air flow rate (V°) of 1.6 m^3h^{-1}. The area-specific air flow rate (q) is then calculated as:

$q = V°/A = 0.59\ m^3\ m^{-2}\ h^{-1}$

For the CME 33 foam, an equilibrium air concentration (Ceq) of approximately 16 $\mu g m^{-3}$ was attained, therefore the area-specific emission rate (SER) is calculated from:

$SER = Ceq \times q = 9.4\ \mu g\ m^{-2}h^{-1}$

From the BAM study, the SER for a piece of uncovered upholstery foam was determined to be 77 $\mu g\ m^{-2}\ h^{-1}$ under the similar test conditions in terms of temperature, humidity and area-specific air flow rate.

The mattress tested by BAM gave an area-specific emission rate of 12 ng m^{-2} h^{-1}, much lower than that measured by EUROPUR, although this mattress was covered which could have reduced emissions.

To illustrate how these emission rates can be used to estimate losses during service life, consider the emission rate of 5.44 µg $m^{-2}h^{-1}$. For a mattress with dimensions 2 x 1 x 0.12 m (one face excluded) the annual emission would be:

Normalised rate per unit area and time x Area x Time

2.72 m^2 x 5.44 µg $m^{-2}h^{-1}$ x 24 h/d x 365 d/y x 1E-09 kg/µg = 1.3E-04 kg/y or 130 mg/y

Assuming a foam density of 27 g/l (as the upholstery foam used in the BAM study), then the foam weight is 6.48 kg and assuming that the loading rate of TCPP is 10% (actual value not reported), this equates to an initial TCPP loading of 0.65 kg. A loss of 1.3E-04 kg/y is therefore equivalent to approximately 0.017% per year.

The highest emission measured by BAM was for an uncovered upholstery foam containing 2% TCPP, which gave an area-specific emission rate of 77 µg$m^{-2}h^{-1}$. The weight of a block of foam with the same dimensions as for the EUROPUR test is 6.48 kg, containing 0.13 kg TCPP. The annual emission is 3.18E-03 kg/y, equivalent to 2.4% per year.

The results of the Elastogran test on a closed-cell rigid insulation foam showed no emission of TCPP up to the detection limit of 1 µg/m^3. However, treating this upper limit as a worst case emission, the SER for this product can be calculated. The surface area to volume ratio is reported as 1.4 m^2/m^3 and the air exchange rate is 0.5 per hour, therefore:

q = 0.5/1.4 = 0.36 $m^3m^{-2}h^{-1}$

SER = Ceq x q = 0.36 x 1 = 0.36 µg$m^{-2}h^{-1}$

The foam tested had a density of 30 kg/m^3, was 10 cm thick (high for practical applications and considered an upper limit), and contained 9% TCPP. Assuming an emitting surface area (one face only) of 1 m^2, and hence a volume of 0.01 m^3, the weight of foam would be 0.3 kg, containing 0.027 kg TCPP. At an emission rate of 0.36 µg$m^{-2}h^{-1}$ the total amount release per year is 3.15 mg TCPP or around 0.01% per year.

The worst-case release from an insulation foam tested by BAM was 0.70 µg$m^{-2}h^{-1}$ for a foam of density 30 g/l and containing 5% TCPP. A block of the same dimensions as tested by EUROPUR would therefore contain 0.015 kg TCPP and the overall release would be around 0.04% per year.

Higher emission levels (up to 70 µg$m^{-2}h^{-1}$) were measured by BAM for assembly foams of density 20 – 25 g/l and containing 14 – 18% TCPP. However, it is not clear whether these samples were covered or uncovered, and the relevance of sawn surfaces in real applications is not known. Again assuming an emitting surface of 1 m^2 and a volume of 0.01 m^3, the block would contain 0.045 kg TCPP and the overall release would be around 1.4% per year.

These results are summarised in **Table B.8**, but should be treated with caution due to the variety of test conditions used.

Table B.8 Summary of annual release rates (excluding Surrey studies)

Sample	Study Reference	Estimated Annual Release (% per year)
Uncovered mattress foam	EUROPUR 2001	0.03
Uncovered upholstery foam	UBA 2003	2.4
Insulation foam (one side uncovered)	Elastogran 2002	0.01
Insulation foam (both sides covered)	UBA 2003	0.04
Assembly foam (sawn surface)	UBA 2003	1.4
Flexible cushion foam	BRMA 2001-2005	~0

The BAM and EUROPUR studies had generally similar conditions, although the latter had larger foam pieces and a larger chamber.

The research carried out on behalf of BRMA is based on the residual levels of flame retardant in foam, determined by measurement of total phosphorus and total chlorine, and reports that FR concentrations are stable over time.

The results of Experiment 1 at 20 °C from the University of Surrey study are of most relevance to the service-life of polymers. Over a 6 week period, losses of 0.02 - 0.11 and 0.18 - 0.26 % (by weight) were measured foam containing TCPP (large and small pieces respectively), while for foam containing TDCP, losses of 0.11 and 0.18% by weight were measured for large and small pieces respectively. The results of Experiment 2 suggest that this loss can be attributed mainly to release of flame retardant. **Table B.9** shows the equivalent flame retardant loss based on the assumption that the weight loss is due entirely to emission of TCPP or TDCP. However, extrapolating a 6-week experiment to an annual weight loss introduces some further uncertainty.

Table B.9 Results of University of Surrey Experiment 1 expressed as annual loss

Foam type	% FR	% loss (by weight, 6 weeks)	Equivalent % FR loss	% FR loss [1] (y)
CM Ether Large	8.47	0.11	1.3	11.3
CM Ether Small	8.47	0.26	3.1	26.9
CMHR Large	5.2	0.02	0.38	3.3
CMHR Small	5.2	0.18	3.5	30.3
FR Ether Large	5.5	0.11	2.0	17.3
FR Ether Small	5.5	0.18	3.3	28.6

[1] Assumes that the rate of loss will remain constant over the year – this assumption has not been tested.

In conclusion, the BAM, Elastogran and EUROPUR studies show estimated annual release rates in the range 0.01% to 2.4%, and one further study with the loss below the limit of detection. No unambiguous explanation for the evident variability is available, although various possibilities are explored. Significantly higher release rates were measured in the University of Surrey study, although this finding is consistent with the smaller dimensions of the pieces of foam tested and the high air-turnover rate used in the experiments. The loss rates

from the very small particles are considerably higher, again showing the importance of the size of the piece of foam.

2.2.2 Dust and loose crumb

The interpretation of these data for small foam pieces/particles will be returned to alongside the findings of Section 3.

2.3 FLAME RETARDANT LEVELS IN INDOOR ENVIRONMENTS

Separate to the model experiments described in Sections 2.1 and 2.2, a number of studies have been conducted measuring flame retardant levels in real indoor environments such as homes, offices, factories and automobiles. Concentrations have been measured in both air and dust.

These data are reported in the main RAR and are not reproduced here. They serve to show that TCPP and TDCP are widely found and underline the need to be able to explain realistically both the mechanisms by which the substances come to be found, and the concentrations.

2.4 APPLICATION TO ENVIRONMENTAL RISK ASSESSMENTS

2.4.1 Losses during curing and storage

After production, blocks of foam are routinely kept in storage at the production site until completely cool. By the same process of diffusion, it is reasonable to assume that local emissions of flame retardant could occur during this storage period. From information gained on a site visit to a major producer, it is known that foam tends to be stored in large warehouses with little air circulation. There is relatively little space between the blocks. Under those circumstances, it is very likely that the air around the blocks will be saturated with the additive, and thus there will be very little loss from the foam. This is very difficult to quantify.

2.4.2 Losses during service life

Service life losses are associated with diffusion through the polymer, followed by volatilisation or washing from the surface. It can reasonably be assumed, in the UK at least, that most domestic homes, offices, institutional or civic buildings will contain furnishings or insulation treated with TCPP and/or TDCP. From the studies reviewed, it can be concluded that losses from large pieces of foam during service life can occur.

2.4.3 Waste remaining in the environment

Waste remaining in the environment (WRITE) is dust and foam fragments generated by some form of physical attrition. It is also likely to be a very important contributor to measured environmental concentrations.

2.4.4 The importance of the receiving compartment

It is useful to summarise here factors that relate to this topic:

The ESD on Plastics Additives (OECD, 2004) does not discuss this other than to suggest a 50% split between air and water for service life losses.

The results and the models (discussed further in Section 3) show that the size of a piece of plastic or foam and the rate of air movement above it are very significant influences on the % emission rate, although it has less influence on the absolute rate, which is area dependent.

The new studies demonstrate a 'sink' effect, i.e. the receiving compartment properties are important. This makes modelling difficult because the number of possible physical locations of foam is enormous. The development of a generic containment model should be possible and subject to validation, but has not been attempted in the present study.

It could reasonably be assumed that in a closed compartment containing only PUR and air, should the air become saturated then the rate of emission from polymer will eventually equal the rate of redeposition (or readsorption)

Given the known vapour pressure of TCPP (and hence its saturated concentration in air), it can be calculated from the rate of release (obtained using the diffusion models described in section 3.2) that a closed compartment of 1 m^3 in contact with 1 m^2 of PUR would become saturated in about an hour and the rate of release will drop to zero if a release-readsorption equilibrium is established.

3 A MATHEMATICAL MODEL FOR LOSS OF FLAME RETARDANT FROM FOAM

Mathematical modelling of the rate of diffusion of non-polymer molecules within plastics has been used to aid interpretation of available data, support some very clear assertions (e.g. about the importance of the size of pieces of plastic) and to compare with measured rates.

For the purpose of clarity, modelling performed in this section assumes that all FR present in the plastic is available for release.

3.1 FUNDAMENTALS

There are several basic premises to the approach set out in the following sections:

A polymer is seen as a continuous matrix, not subject to physical or biological degradation. Such processes are important but are not the subject of the present text. Given the properties of foam, some adjustments will be needed. Foam is not a continuous matrix since it contains air cells, therefore the effective thickness of polymer is less than the thickness of the foam block itself. It is assumed that there is

no barrier to the migration of flame retardant through the air cells. The effective polymer thickness will be controlled by the cellular wall structure.
Additives are initially uniformly distributed through the polymer, without there being 'domains' of additive at very high concentration; and that redistribution occurs as a result of surface loss.
Additives are not chemically bound to the polymer, the only interactions being weak (non-specific physical interactions or weak hydrogen bonds). This assumption is critical, because if stronger forces such as strong hydrogen bonds are formed, then the basis of the diffusion model is flawed. However, studies of temperature dependence can give insights as to whether such bonding is occurring.
In the modelling, the concentration of an additive in the receiving compartment (usually air) is assumed to not be influential; however, this is an important factor, which is considered qualitatively. A containment model would need to be developed to account for this and is outside of the scope of this study.
A containment barrier model is also required for those cases where the foam is covered by a fabric or other layers that might constrain the additive at or close to the interface between the foam and the barrier, and prevent air flow over the surface. This is also dealt with by a quantitative estimation.

Under such conditions, an additive molecule at the surface of a polymer may evaporate from it or be washed from it. This process can continue, and, if the rate of escape from the surface is faster than the rate of diffusion (which there is every reason to believe is the case) then, in time, a concentration gradient near the surface of polymer can arise, of a scale much larger than molecular (microns to millimetres in size, perhaps).

Diffusion of solutes in liquid solution is known to depend primarily on molecular size, temperature, and viscosity of the solvent. The diffusion coefficient ***D*** is the primary descriptor of rate, as expressed in Fick's laws of diffusion. Fisk and Jonathan (1999) have provided a review of the prediction of diffusion coefficients in solution. In practice, diffusion in homogeneous solution can only be measured easily where a concentration gradient exists. At a boundary between phases (e.g. aqueous and non-aqueous immiscible solutions), molecules generally cross the interface freely, particularly where this partitioning process is favoured by the position of equilibrium and the relative concentrations in the two phases.

Considering polymers, the situation is more complicated because they are not very mobile, and therefore molecules can move less easily within the polymer than they can in solution. Nevertheless, many of the same principles apply. At the polymer-air interface, it could be envisaged that the additive could accumulate on the surface, but it may be assumed that where air is circulating freely, the concentration of the additive in air will be effectively zero, and that molecules of additive reaching the surface will evaporate rapidly. The consequence is that a diffusion gradient will be established within the polymer. A further uncertainty is that in cellular foams a different mechanism may exist due to the cellular structure and the establishing of a cellular-volume/external-atmosphere exchange mechanism (Note: this is akin to the cell wall acting as a gas/vapour transport membrane rather than a semi-infinite slab (as assumed herein, applying Fickian and Case I and Case II diffusion).

3.2 DEVELOPMENT OF THE MODEL

Sections 3.2.1 and 3.2.2 develop some simple equations that can readily be applied to the migration of additives in polymers. Sections 3.2.3 to 3.2.5 demonstrate the influence of

varying different parameters on the outputs of the model, while application of the model to scenarios relevant for polyurethane foams and comparison with measured data are discussed in Sections 3.3 and 3.4.

The mathematics of diffusion in solution and polymers is complex and so some major simplifications have to be made just to generate some practical numbers.

Migration of substances in polymers has received considerable attention in respect of studies for food contact approval, and whilst there are standard tests to meet regulatory targets, a reasonable body of more fundamental research has been carried out, and is still ongoing. This field of research is useful as a source of data, but it is beyond the present scope to review it. The equations used are similar, and the papers obtained contain measured diffusion coefficients.

Migration in polymers is sufficiently slow that it can be readily assumed that molecules that reach the polymer surface can volatilise or dissolve in any solvent there much faster than the diffusional rate (Fisk *et al*., 1999). It at least represents a reasonable worst case.

The sources of the equations used are such standard sources as Crank, 1975.

3.2.1 Initial rates

Fick's second law of diffusion deals with diffusion which is time-dependent, i.e. during the period between time zero and the establishment, if it occurs, of a steady state.

Consider a newly formed polymer containing evenly-distributed additive at concentration C_0. If the area of surface exposed to a sink for the substance is A, then Fick's second Law can be solved such that, for small amounts of loss (up to approx. 20%), the number of moles lost N is given by:

$$N = 2AC_0\left(\frac{Dt}{\pi}\right)^{0.5}$$

where D is the self-diffusion coefficient. This equation predicts that rate will slow with time, which is a consequence of the physical fact that the molecules near the surface will escape first, and then it takes more time for the deeper ones to reach the surface and escape. It also shows that the rate of loss is proportional to area and concentration, which seems entirely reasonable.

The diffusion coefficient represents the rate at which a molecule can diffuse through a medium. Diffusion coefficients depend on temperature, molecular size, and the viscosity of the solvent, and they can be predicted relatively easily (Fisk and Jonathan, 1999). Workers on diffusion in polymers give similar results (see Section 6, and in particular Reynier *et al*., 2001). Reynier *et al*. did not carry out an *ab initio* prediction, they simply sought correlation of some molecular size and shape parameters obtained from a molecular dynamics code with actual diffusion measurements in a single type of semicrystalline polypropylene at 40°C. The authors commented that these would not necessarily generalise to other conditions, or to other polymers. Such correlation approaches can however be very useful and could be constructed for PUR foams with appropriate experimental work.

3.2.2 Steady state rates

Eventually the initial rate of movement slows. The achievement, if it occurs, of a steady state implies that a linear concentration gradient is established over some depth ***L*** of the polymer. Again assuming that a single surface is exposed, with a concentration ***C*** in the interior of the polymer, then

$$\frac{N}{t} = \frac{ACD}{L}$$

This equation again shows that the rate of loss from the matrix is proportional to area and concentration.

Whether the initial rate model or the steady state model is most appropriate in the present context is explored below.

3.2.3 Application of the models

Application of the models requires a mixture of reasonable assumptions and measured values for the input data. These are described in **Table B.10**.

Table B.10 Input parameters for models

Constant	Meaning	Comment
A	Exposed area (m^2)	Reasonable assumptions can be made
C	Concentration of additive (%)	Usually known
t	Time scale (y)	Usually known
D	Diffusion coefficient (m^2/s)	Measurements for diffusion rates of additives in polymers are known, and a number of predictive methods are available (see Section 6)
L	Thickness of polymer over which a steady state is established (m)	This may well not be known; since it is only needed for the steady state equation, it may not be relevant.

3.2.4 Use of the Initial Rate Model

For the 'demonstration' calculations, the model was set up using the following parameters, reasonably representative of polymers but not intended to be specific.

Substance molecular weight: 300 g/mol

Temperature: 25°C

Diffusion coefficient: 3×10^{-15} m^2/s

Concentration of additive: 5%

Density of polymer: 1100 kg/m^3 – this assumes the bulk density to be consistent throughout.

These values were kept constant while the initial investigation was carried out.

3.2.4.1 Large flat pieces of plastic

3.2.4.1.1 Model outputs

The influence of surface area and timescale on the output of the initial rate model was investigated. To simplify calculations, it is assumed that only one surface is available for diffusion. This might be justified since during service life, the surfaces of polyurethane foam blocks are covered in some way e.g. by upholstery fabric in flexible foam for sofas or mattresses, or sandwiched between plastic or metal for rigid foam in construction applications.

For a piece of plastic with thickness 0.1 m, the surface area available for diffusion was varied from 0.0001 m^2 to 5 m^2 over timescales of 5, 10 and 20 years. The model outputs in grams are presented in **Table B.11**.

Table B.11 Amount of additive lost (grams) as a function of surface area and timescale

Timescale (y)	Surface area (m^2)									
	0.0001	**0.0005**	**0.001**	**0.005**	**0.01**	**0.1**	**1**	**2**	**3**	**5**
5	0.00427	0.0213	0.0427	0.213	0.427	4.27	42.7	85.4	128	2.13E+02
10	0.00604	0.0302	0.0604	0.302	0.604	6.04	60.4	121	181	3.02E+02
20	0.00854	0.0427	0.0854	0.427	0.854	8.54	85.4	171	256	4.27E+02

This demonstrates that the amount of substance released varies linearly with surface area and is dependent on the timescale considered. Expressed as a percentage loss averaged over time, as in **Table B.12** there is no dependence on surface area since the initial amount of additive present also varies linearly with surface area for a rectangular block.

Table B.12 Average annual percentage loss (thickness = 0.1 m)

Timescale (y)	Average percentage loss %/y
0.1	1.1
1	0.35
5	0.16
10	0.11
20	0.08

The magnitudes are discussed below. **Figure B.2** shows the total amount lost versus timescale for a 1 m^2 x 0.1 m block of foam, while **Figure B.3** shows annual percentage loss as a function of timescale. While the total amount lost clearly increases over time, this relationship is not linear, as the rate of loss decreases with time. This also means that when considering average annual losses, e.g. for regional risk assessment calculations for in-service loss, the expected lifetime of the product is an important consideration

For this initial rate model, the total amount of substance lost is independent of the thickness of the polymer block. **Table 3.4** shows the model outputs for a block with surface area 1 m^2 and varying thickness, over a 10-year timescale. Percentage loss is inversely proportional to

thickness, since the initial amount of additive present is dependent on thickness but the net amount lost remains constant.

Table B.13 Amount lost as a function of thickness
(surface area = 1 m^2, timescale = 10 years)

Thickness (m)	Total amount lost (g)	% lost over total time	Average percentage loss (%/y)
0.005	60.4	22	2.2
0.01	60.4	11	1.1
0.05	60.4	2.2	0.22
0.1	60.4	1.1	0.11
0.5	60.4	0.22	0.022

Figure B.2 Total amount lost as a function of timescale (surface area = 1 m^2)

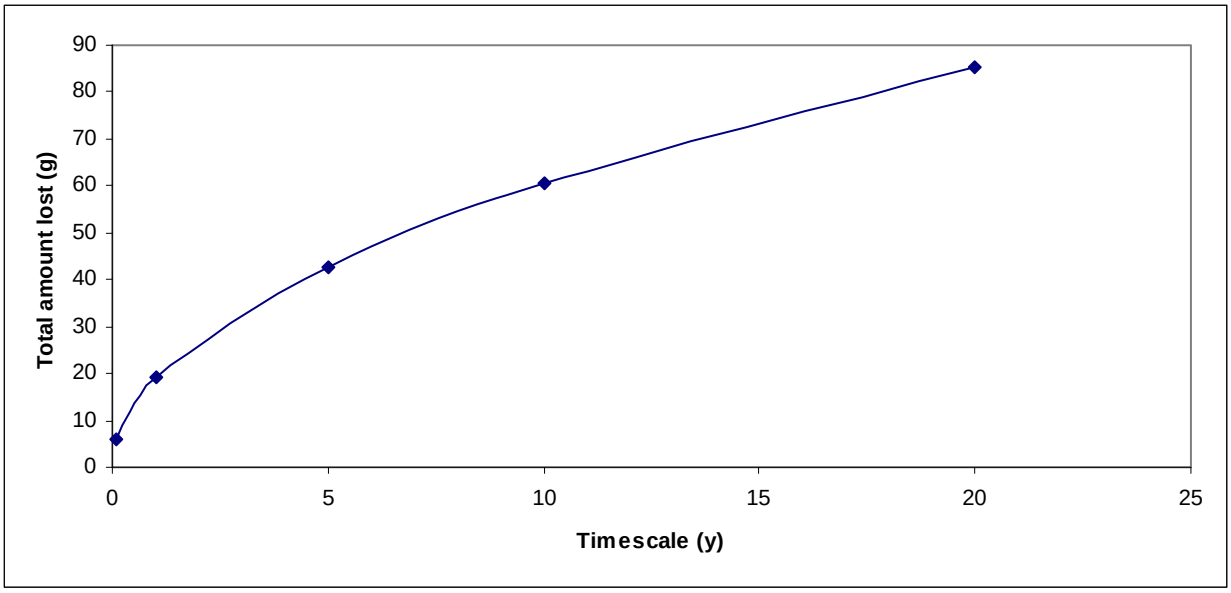
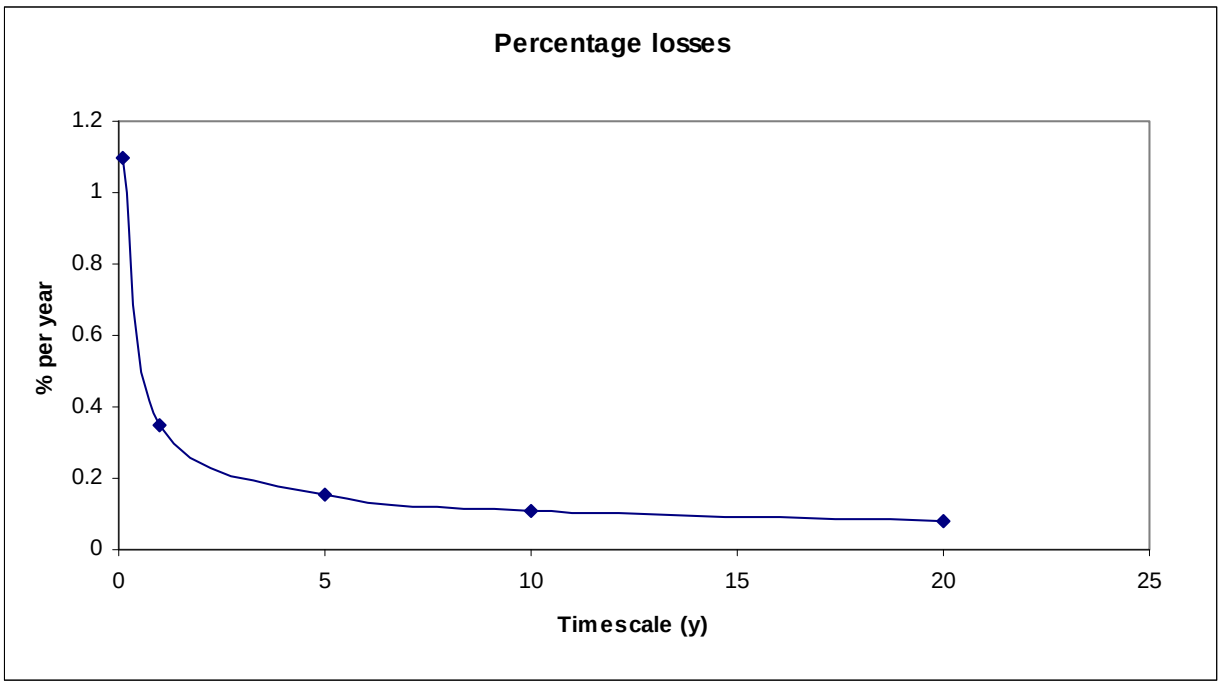


Figure B.3 Annual average percentage loss as a function of timescale (thickness = 0.1 m)



3.2.4.1.2 Applicability to polyurethane foams

Due to the nature of foams, the bulk density of a foam block is considerably lower than the density of the polymer itself. Typical flexible foams for use in furniture have a bulk density of 10 – 60 kg/m^3 (Woods, 1982). For the purposes of modelling, it can be assumed that there is no limitation to the diffusion of an additive through 'air cells' in the foam. Since it is already assumed that diffusion is occurring from one surface only, the "effective" thickness of polymer can therefore be determined if both densities are known and the available surface area remains constant:

Effective thickness = Actual thickness x (Bulk density of foam/Density of polymer)

As described in the risk assessment reports for TCPP, TDCP and V6, blocks of foam are stored on-site during the curing process. Curing time is typically 48 hours and temperatures can be as high as 150°C in the middle of a large block, although at the surface temperatures will be close to ambient. There is therefore potential for volatile emissions at this stage of the life-cycle.

3.2.4.2 Small particles

As well during the service life of polyurethane foam articles, losses due to diffusion should also be considered for two other scenarios. Waste remaining in the environment (WRITE) arises from physical abrasion of a polymer due to weathering and wear. For polyurethane foams, such losses may occur in addition to the in-service losses associated with use in furniture foam and result in small particles (e.g. 10-100 μm in size) of polymer collecting, for example, in dust. On this scale it could be assumed that no correction is required for bulk density of the foam.

A further life-cycle stage which may be of relevance is the production of rebonded or loose crumb foam from scrap foam produced as a result of cutting blocks into the required shapes. Scrap foam is shredded into pieces approximately 1 cm in diameter and, taking into account the correction for bulk density, there may be potential for significant volatile losses from these small pieces during the process. Once incorporated into rebonded foam or loose crumb furniture, it could be assumed that the diffusion behaviour is equivalent to that of a larger solid block.

In both cases, the assumption that diffusion occurs from only one surface is not valid, as the particles are likely to be approximately spherical. A correction for the increased surface area is therefore required.

For a spherical particle with diameter 100 μm, the surface area is calculated from $4\pi r^2$ and the volume is $4\pi r^3/3$ (r = radius = 50 μm), therefore the area is 3.14E-08 m^2 and the volume 5.24E-13 m^3. Inputting these values the model gives a percentage loss of 100% in less than a day, indicating that all additive would be lost over a very short timescale. Under conditions of low air movement, this loss may be ameliorated. The loss may seem surprising but reflects the small particle size. It should be borne in mind, however, that the model assumes a polymer that would have no specific interactions with any additive. Given that polyurethane is frequently used as an adsorbent in analytical chemistry, this assumption may be invalid.

The initial rate model is only strictly valid for up to about 20% loss of the substance from the polymer. At losses up to 50% the steady state model is therefore preferred because its parameters would reflect the physical reality of the concentration gradient present. If complete loss is predicted, this is outside the scope of both models but the results are still useful qualitatively, as an indication of the order of magnitude.

For a particle of 1 cm diameter, as applicable for producing rebonded or loose crumb foam, a correction for bulk density is required. The surface area available for emission remains at $4\pi r^2$ (3.14E-04 m^2), but the "effective" volume can be calculated by:

Effective volume = Actual volume x (Bulk density of foam/Density of polymer)

Assuming that the foam has a bulk density of 30 kg/m^3, the effective volume is therefore 1.43E-08 m^3 and the effective thickness is 1.5E-03 m. Inputting these values into the model with a timescale of 1 day gives an emission of over 100%. This indicates that volatile losses of additive during the production of rebonded foam could potentially be significant. Controls in these locations may not be so stringent as those in place at foaming locations where isocyanates are in use. However, it should be noted that typical industry practice is to carry out granulating processes within contained equipment, therefore actual rates of loss are anticipated to be much lower than the modelled results.

3.2.4.3 Impact of varying other parameters

To investigate the dependence of releases on parameters other than the dimensions of the piece of plastic, a fixed size of 1 m^2 surface area and 0.1 m thickness was used in the model with a 10 year timescale. Unless stated otherwise, other values used were as described in section 3.2.4.

3.2.4.3.1 Molecular weight

A number of measured diffusion coefficients in polymers are available, but a predictive equation is also available (Reynier *et al*., 2001). Predicted diffusion coefficients are dependent on the molecular weight (MW) of the additive according to the relationship:

$D\ (m^2/s) = 10^{(-7.83 - 0.0062MW)} /10000$

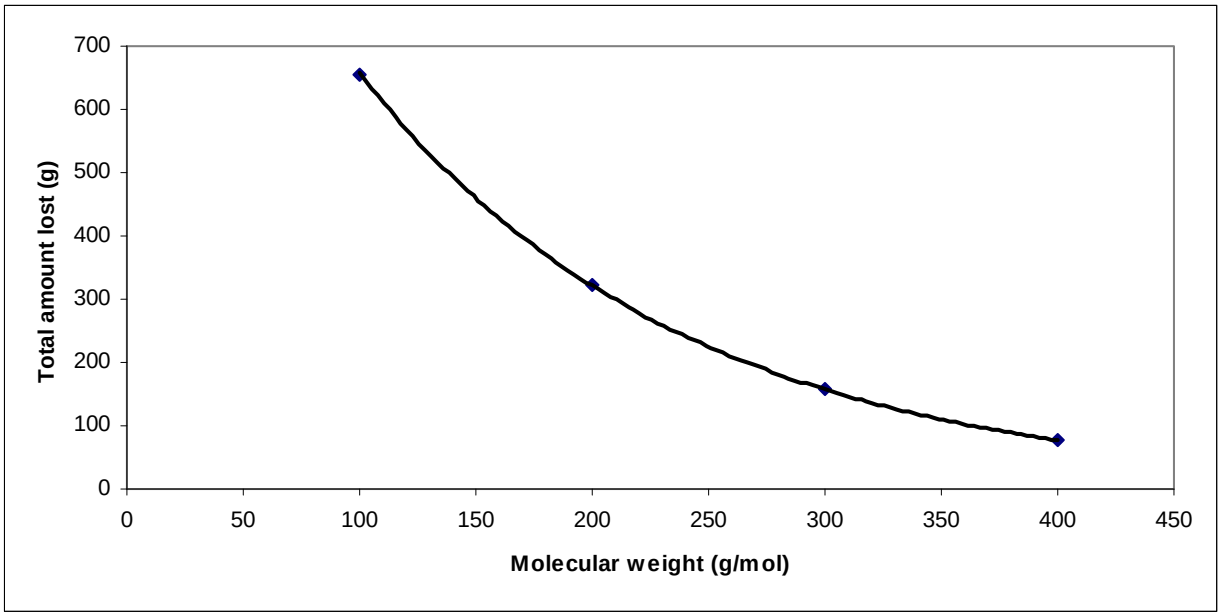
Using diffusion coefficients predicted by the model, releases for varying molecular weights are shown in **Table B.14** and **Figure B.4**.

Table B.14 Amount lost as a function of molecular weight

Molecular weight (g/mol)	Predicted diffusion coefficient (m^2/s)	Amount lost over 10 years (g)	Average annual loss (%)
100	3.548E-13	656	1.2
200	8.511E-14	322	0.585
300	2.042E-14	157	0.287
400	4.898E-15	77	0.14

It can therefore be seen that, as might be expected, the amount of additive lost increases exponentially with decreasing molecular weight. This approach is much less sensitive than the use of vapour pressure as a guide, as described in the ESD; vapour pressure changes very rapidly with changing molecular weight, whereas the diffusion model is less sensitive.

Figure B.4 Amount lost as a function of molecular weight



3.2.4.3.2 Temperature

Predicted diffusion coefficient, and hence release rate, is also dependent on temperature according to the relationship (many references, reviewed in Fisk and Jonathan, 1999):

$D\ (X°C) = [D\ (25°C) \times (X + 273)]/298$

This is shown in **Table B.15** and **Figure B.5**. The equation used here is only applicable at fixed viscosity of polymer (i.e. a thermoset polymer such as PUR, rather than a thermoplastic one).

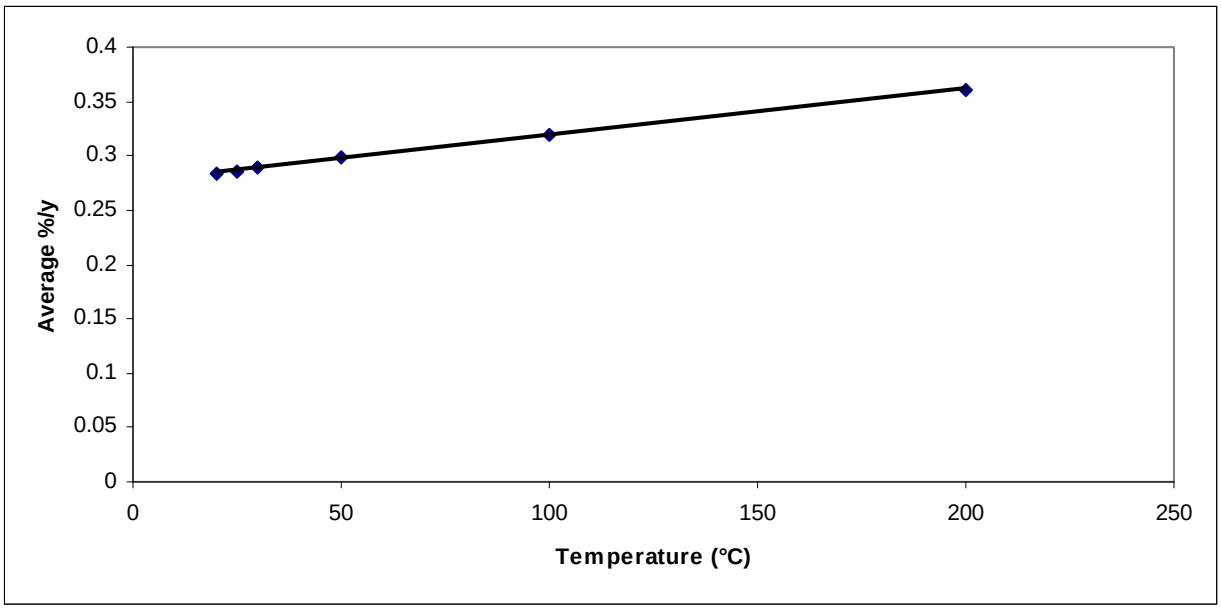
Table B.15 Amount lost as a function of temperature

Temperature (°C)	Predicted diffusion coefficient (m2/s)	Amount lost over 10 years (g)	Average annual loss (%)
20	2.007E-14	156	0.284
25	2.042E-14	157	0.286
30	2.076E-14	159	0.289
50	2.213E-14	164	0.298
100	2.556E-14	176	0.320

Although the difference made by temperature is small, this could become more significant for high or low-temperature applications.

The effect of temperature is small; this is a very useful result because the Plastics Additives ESD does not deal with this issue. For thermoplastics, the temperature dependence would be a little higher, since the viscosity of the polymer will change with temperature, but that is not described herein as it is not applicable to polyurethane foams.

Figure B.5 Amount lost as a function of temperature



3.2.5 Use of the Steady-state model

The initial rate model is only strictly valid for up to about 20% loss of the substance from the polymer. At losses up to 50% the steady state model is preferred on theoretical grounds. In some instances (very small particles) complete loss is predicted, which is outside the scope of both models but the results are still useful qualitatively, as an indication of the order of magnitude. The steady-state model refers to the point at which a linear concentration gradient

has been established within the polymer block. At this stage both surface area and thickness are important for determining the amount of substance lost, but expressed as a percentage per year, the rate of loss is dependent only on thickness.

The release rates predicted by the steady-state model are lower than the initial rate model. In the extreme scenario of very thick pieces of polymer, percentage loss values will be very low indeed, as shown in **Table B.16**

Table B.16 Percentage loss per year as a function of thickness (surface area 1m^2)

Thickness (m)	% per year
0.5	3.78E-05
1	9.46E-06

3.3 APPLICATION OF THE INITIAL RATE MODEL TO PUR FOAMS CONTAINING TCPP

3.3.1 Model Parameters

The initial rate model was tested for various scenarios relevant to the life cycle of TCPP. The following parameters were fixed in the model, which are representative of the properties of foams for which measured data are available, as described in Section 2.

Substance molecular weight: 328 g/mol

Concentration of additive: 5%

Density of polymer: 1100 kg/m^3

Bulk density of foam: 30 kg/m^3

The diffusion coefficient (3E-15 m^2/s) obtained from the literature was used.

3.3.2 Life cycle Stages

The outputs from the model are given in **Table B.17**.

3.3.2.1 Losses during curing

At foam production sites, large blocks of foam (typically with dimensions 60 x 2.2 x 1.25 m) are stored on-site while curing takes place. Temperatures in the interior can reach up to 150°C, but at the surface the temperature will be near ambient.

Inputs to the model were therefore as follows:

Surface area: 132 m^2

Thickness: 0.034 m (correcting for density)

Temperature: 25°C

Timescale: 2 days

3.3.2.2 Losses during service life

A typical application of PUR foam containing TCPP is in furniture such as sofas. Dimensions of a piece of such furniture foam could be, for example, 2 x 0.5 x 0.1 m. The temperature of a typical room is 23°C.

Inputs to the model were therefore as follows:

Surface area: 1 m^2

Thickness: 2.7E-03 m (correcting for density)

Temperature: 23°C

Timescale: 10 years

3.3.2.3 Waste Remaining in the Environment

Waste remaining in the environment (WRITE), for the present purpose, refers to small particles of foam produced from weathering and wear during service life, separate to volatile releases from the foam block itself. Volatile releases can also be expected from such particles. Applying the scenario to TCPP, the inputs were as follows:

Surface area:3.14E-08 m^2

Thickness: 50 µm

Volume: 5.24E-13 m^3.

Temperature: 23°C

Timescale: 1 day

3.3.2.4 Production of rebonded and loose crumb foam

The following inputs were used for TCPP:

Surface area: 3.14E-04 m^2

Thickness: 1.36E-04 m

Mass of additive present: 1.572E-05 kg

Temperature: 23°C

Timescale: 1 day

Table B.17 Releases of TCPP from typical life cycle stages

Lifecycle Stage	Percentage loss
Curing	0.076% in two days using initial rate model
In-service	1.3% per year before accounting for any covering, using steady state model
WRITE	100% loss in a few days (both models)
Rebonded foam	Maximum of 13% in one day predicted by initial rate model

These results are subject to a number of approximations and assumptions, and should not be over-interpreted.

3.4 COMPARISON OF MODEL WITH MEASURED VALUES

Table B.7 summarises the annual emissions derived from available studies in the literature.

An uncovered upholstery foam tested by EUROPUR in 2001 showed a measured release rate of 0.03% per year, whereas in a test by UBA in 2003, a release rate of 2.4% per year was measured. Since the exact dimensions of the foam tested by UBA are not known, it is not possible to directly compare the output from the model with this result. However, the result is not inconsistent with the model prediction of 1.3% per year for in-service loss.

In practice, some amelioration of the model results is to be expected since in practice, foams used in most applications are covered in some way e.g. upholstery fabric for furniture foams, steel panels for insulation foams.

Experiment 1 from the University of Surrey study is the one of most importance, because it included ambient conditions. Emission rates were found to be highly dependent on the dimensions of the piece of foam. Higher temperatures lead to higher diffusion rates and hence higher emissions. The results of this experiment were used to test the new model, as described below. It should be noted that during the air turnover period, the ovens used in this test may have become partially saturated.

For CM ether foam containing 8.47 % TCPP, density 32 kg/m^3, size 50 mm x 50 mm x 15 mm ('small'), the initial rate model at 20°C predicts 7.78 % loss over 6 weeks from one face of 50 mm x 50 mm, which should be multiplied by 3.2 for the whole surface area of the block, giving 24.9 % loss of TCPP, or 2.1 % of the total weight. The measured weight loss at this temperature is 0.26 %. Note: a factor of 8 difference may seem high but this may be due to containment effects.

For pieces of size 100 mm x 100 mm x 50 mm the initial rate model gives, at 20°C, 2.34% loss over 6 weeks from one face of 100 mm x 100 mm, which should be multiplied by 4 for the whole area, giving 9.36% loss of substance, or 0.79% of the total weight. The measured weight loss at this temperature is 0.11%.

Experiment 2 from this study indicates that the observed weight loss is mainly due to loss of flame retardant.

The data for loss from dust and foam show a plateau at around 40% loss, preceded by rapid (and hence facile) loss. The modelling predicts that all the FR should be lost very quickly. This suggests that 60% of the FR is unavailable to be lost from the foam to its surroundings.

The model seems to predict values of the right order of magnitude, and the relative rates for pieces of different sizes are dealt with well. The pieces used were all small relative to foam in actual use. Results are expressed in various forms in **Table B.18**; it must be borne in mind that these results do not reflect the loss that might occur with larger (or smaller) pieces.

Table B.18 Comparison of model predicted emissions with measured total weight loss (CM ether foam)

	Total Weight Loss (%)			
Temperature (°C)	**Predicted**		**Measured**	
	Small	Large	Small	Large
20	2.1	0.79	0.26	0.11
60		0.84	7.12	3.21
	TCPP Loss (%/d)			
Temperature (°C)	**Predicted**		**Measured**	
	Small	Large	Small	Large
20	0.59	0.22	0.07	0.031
60		0.24	2.0	0.90
	TCPP Loss (%/y)			
Temperature (°C)	**Predicted**		**Measured**	
	Small	Large	Small	Large
20	100	80.3	26.7	11.3
60		100	100	100

At 60°C the model predicts total weight loss of 0.84 % for a large piece of foam, while the measured data show a loss of 3.21%. This temperature dependence is much higher than expected for weak intermolecular forces, due to an activated process not accounted for in any diffusional model. The magnitude of the temperature dependence suggests some kind polar interaction with the polymer. Indeed, it is known that both substances adsorb moderately strongly to soil, which whilst being a very different medium, contains polar and non-polar domains just as polyurethane does. However, an irreversible chemical reaction is not implied by the data. The model predicts relatively small diffusional differences between TCPP and TDCP under conditions of high air turnover; this was found at 20°C. However, since air turnover is in fact important, then the lower loss rate of TDCP would be consistent with its lower vapour pressure, TDCP may also have a greater propensity than TCPP to associate with the PU foam.

3.5 CONCLUSIONS

3.5.1 Outcome of modelling

The modelling shows several important findings, the implications of which may need further work, not necessarily within the present project:

Loss rates from pieces of foam of dimensions 1 cm and below are predicted to be very fast, and, in a receiving compartment of sufficient size, complete loss can occur over a period of hours. The measured data show this to be correct, but modified for a value of around 60% of the FR which is not lost at all.

Loss rates from large thick pieces of plastic are predicted to be very much slower than the predicted values for flame retardants from the Plastics Additives ESD. However, even large blocks of foam contain a relatively small amount of polymer, and predicted rates are of the same order as measured values.

3.5.2 Comparison with Emission Scenario Document for Plastics Additives

The current Emission Scenario Document for Plastics Additives (OECD 2004) gives generic emission factors for losses of additives during the service life of plastic goods. For indoor service life, a default release of 0.05% to air over the service life for an additive of moderate volatility. Typical service life varies from 5 to 20 years depending on the application. For an additive with high volatility, the loss rate is increased by a factor of 5.

As demonstrated in Section 3.2, the total amount and percentage of additive lost through diffusion is dependent on the dimensions of the plastic, and the rate of loss is not constant during the service life of an article. While the default loss rates given in the ESD are within the range of values predicted by the model (e.g. **Table B.12**), there are grounds to suggest that a review is needed.

The Plastics Additives ESD approach to in-service loss does not take into account:

The concentration of additive in the polymer (although this will not change the rate when expressed as a % of initial concentration).
The mechanism of additive loss and the effect of containment.
The effect of polymer matrix type and structure on diffusion rates.
The relationship between molecular size and rate of diffusion.
Time-dependence of average annual release rates.
Time-temperature profile at different points in the life cycle.
Influence of the dimensions of the piece of plastic, which is probably the most important variable.
The significance of the air exchange rate, and the potential for saturation of the receiving air in contained situations – most practical situations are “contained”.
The presence of any fabric or other barrier at the surface.
The ESD sets a fixed rate of in-service loss, modified according to volatility. In practice, the key variable (D) is related to molecular size; volatility is also related to size.

4 DERIVATION OF RELEASE RATES FOR USE IN THE ENVIRONMENTAL RISK ASSESSMENTS

For application of the above findings for the purposes of risk assessment, a ‘reasonable worst case’ interpretation of the various sources has been applied.

Table B.19 sets out the basis of treatment of these releases to be used in the RAR. The rates presented in the table relate to TCPP. It must be noted that the % figures have all been multiplied by a fraction, representing that which is ‘available’ for release, i.e. is not very

strongly bound. This fraction is estimated to be 0.4 for TCPP (from the data) and 0.1 for TDCP and V6 (an estimate from a very limited amount of data).

Table B.19 Conclusions of the modelling related to life cycle stages in the risk assessment of TCPP, TDCP and V6

Application area	Conclusions
FLEXIBLE FOAM	
Foam production	It is considered that the only source of releases from large foam production sites will be from curing and storage (see below for more details). At small sites, a handling release is also included, in line with the published ESD. Additional releases associated with the generation of foam dusts due to cutting of foam blocks at the site must also be considered, since modelling now shows that FR contained in foam dusts will very rapidly be volatilised (see WRITE (Waste Remaining In the Environment) below). Since high levels of control are known to apply at these sites, it is considered adequate to assume that this release is negligible and contained within the curing/storage losses (see below).
Curing and storage at foam production sites	Rates of release to air are calculated from the in-service loss rate, and loss rates of 2.4% per year (worst-case emission from the BAM study) could apply. However, blocks are large and the air around them at the production site would probably be saturated for most of the time. The effect of air saturation on release rates is demonstrated in Experiment 4 of the University of Surrey study where at 60°C a release of 0.11% TCPP was measured over 4 months in a sealed vial, compared with 39.5% loss in 6 weeks in an oven test with air movement. The release rate of 2.4% is therefore considered to be too high for the conditions at the production site, and reduction by a factor of 100 is proposed. The proposed rate is therefore 0.024% to air, per year. This fraction applies to the fraction of product actually in storage at any one time, estimated in the RAR at 2.5%, giving an overall loss of **0.0006% per year to air**, for all sites. 50% is assumed to adsorb to surfaces and reach wastewater due to cleaning. While some internal parts of the foam blocks reach a high temperature during curing, this is not expected to have a significant influence on the release rate (as discussed in section 3.3.2.1). Correcting for availability, the release rates used in the risk assessment are: TCPP: 1.2E-04% to air and 1.2E-04% to wastewater TDCP: 3E-05% to air and 3E-05% to wastewater V6: 3E-05% to air and 3E-05% to wastewater
Further processing (i.e. at cutters' and furniture manufacturers' sites)	Cutters (termed 'converters' by the industry) and furniture manufacturers will store foam and cut it. The data and models indicate that there must be volatile losses from such locations. The same rate as for curing and storage at producers' sites should be applied for such stages. Additional releases associated with the generation of foam dusts must also be assessed, since modelling shows that FR contained in foam dusts will be volatilised very rapidly (see WRITE below). While it is known from consultation that dusts are collected at the point of cutting by extractors attached to the blade, it could still be the case that a small proportion of dusts and small pieces of foam are exposed to air and hence that some FR could be released on a local scale. A study has established that up to 0.1% of foam is lost as dust and non-recycled offcut pieces (EUROPUR, 2005), and it is herein assumed that 1% of this material is not collected by the extractor systems. These pieces of FR foam could then release FR into the workplace air and could reach the environment via air and also wastewater (via adsorption and cleaning). A release rate of **0.0005% to air and 0.0005% to water per year** is therefore proposed. Correcting for availability, the release rates used in the risk assessment are: TCPP: 2E-04% to air and 2E-04% to wastewater TDCP: 5E-05% to air and 5E-05% to wastewater V6: 5E-05% to air and 5E-05% to wastewater
In service loss for flexible foams (covered upholstery foams, mattresses, automotive	For uncovered foams, the % loss rate could be as high as 2.4%/year. However, given that the air surrounding the foam is likely to be slow moving, and the foam is covered in service by fabrics and upholstery, then it is proposed to reduce the rate by 10 x for each of these two

Application area	Conclusions
furnishing & sound insulation; including rebonded foam)	release-limiting factors. This is an estimate that is justified pragmatically on the basis of workplace monitoring data, and the fact that FR performance is not dramatically lost over time. An annual rate of release of **0.024% per year** to air is proposed for TCPP. For TDCP and V6, which have much lower volatility, a rate correction of ~25 is appropriate to allow for the slower rate of release at moderate air turnover, which is consistent with the ESD. Therefore the annual rate of release for TDCP and V6 is proposed as **0.001% per year**. Please note that this correction refers to *slower speed of release*, and is separate from the correction for lower *total amount available for release* for these substances compared with TCPP. Please refer to the discussions of different air turnover scenarios below the table. Correcting for availability, the release rates used in the risk assessment are: TCPP: 9.6E-03% to air TDCP: 1E-04% to air V6: 1E-04% to air
Loose crumb	The rate for loose crumb, used mainly in outdoor furnishing, with covering, is set to 0.24% for TCPP, 0.01% for TDCP and V6. Correcting for availability, the release rates used in the risk assessment are: TCPP: 0.096% to air TDCP: 1E-03% to air V6: 1E-03% to air
Recycling of flexible foams: loose crumb and rebonding	Both methods involve the generation of foam granules. Granule sizes are typically around 1 cm and therefore the model shows that losses of FR could be as high as 13% per day. However, the granulation and rebonding processes are contained within equipment, therefore rates of loss are anticipated to be much lower. Granulating machines are fitted with dust extraction equipment. Taking the same approach as for cutting at furniture manufacturing sites, it could be estimated that up to 0.1% of foam is lost as dust, and that 1% of this material is not collected by the extractor systems and could be released to the local air compartment. Releases are therefore 0.001% to air. Correcting for availability, the release rates used in the risk assessment are: TCPP: 4E-04% to air TDCP: 1E-04% to air V6: 1E-04% to air
RIGID FOAMS	
Rigid foam (production of panels)	As proposed in earlier work (Dec 03), it is considered that the only source of releases from large foam production sites will be from curing and storage (see below for more details). At small sites, a handling release is also included, in line with the published ESD. Additional releases associated with the generation of foam dusts due to cutting of panels at the site must also be considered, since modelling shows that FR contained in foam dusts will be volatilised very rapidly (see WRITE below). Since high levels of control are known to apply at these sites, it is considered adequate to assume that this release is negligible and contained within the curing losses (see below).
Curing and storage at foam production sites	Rates of release should now be calculated from the in-service loss rate of an uncovered foam. Loss rates of 2.4% per year could apply, equating to 0.0066% per day. However, blocks are large and the air around them would probably be saturated, as discussed previously for flexible foams, so this rate is estimated to be 100 x too high. The presence of facing panels will be an important additional retarding factor, say 10 x. The proposed rate is therefore 6.6E-06**%** to air per day. This fraction applies to the fraction of product actually in storage at any one time. This is not estimated in the RAR but could be around 1%, giving an overall loss of **2.4E-5% per year to air**, for all sites.

Application area	Conclusions
	Correcting for availability, the release rate used in the risk assessment is: TCPP: 4.8E-06% to air and 4.8E-06% to wastewater
1K foams – releases from foaming *in situ*	Release from foaming *in situ* (e.g. during building work) is based on the rate of release in service. Based on an uncovered foam (at the time of spraying) the loss rate should be as calculated for uncovered flexible foam, reduced by an estimated 10 x due to the enclosed nature of the application, giving 0.00066% per day. The formation of a 'skin' on spray foam may make this a slight over-estimate. Correcting for availability, the release rate used in the risk assessment is: TCPP: 0.096% to air
Spray foams – releases from foaming *in situ*	Release from foaming *in situ* (e.g. insulation of roofs) is based on the rate of release in service. Based on an uncovered foam (at the time of spraying) the loss rate should be as calculated for uncovered flexible foam, reduced by 10 x due to the large volume of the foam produced, giving 0.00066% per day. Correcting for availability, the release rate used in the risk assessment is: TCPP: 0.096% to air
In-service loss (sandwich panels; 1K foam; spray foam)	All of these foam types are in highly enclosed environments in service, and the rigidity of the foam would be a further retarding factor. Given the use in buildings where there will be very limited air circulation around the exposed foam and edges of panels, it is proposed to now set these rates of release to zero.
BOTH FOAM TYPES	
WRITE – weathering and wear in service, via abrasion and creation of small foam particles	The present approach is to assume complete release of the available fraction from small particles. The modelling suggests, however, that this will occur very rapidly, and dust reaching landfill will no longer contain the additive FR in a form that is available for release. Correcting for availability, the release rates used in the risk assessment are: TCPP: 0.8% to air TDCP: 0.2% to air V6: 0.2% to air
Release within landfill	It is not realistic to attempt to model losses from landfill. However, the Environment Agency has made measurements of TCPP and TDCP in leachate from a number of landfills, and these will be used to set up a general approach to releases.

TDCP and V6

The rates (before correction for the 'available' fraction) to be applied in the risk assessments for TDCP and V6 require further consideration. It should not be assumed that vapour pressure is a perfect indicator of volatility (it is a guide), because vapour pressure relates to the equilibrium of a vapour with an excess of the pure substance, e.g. as a liquid phase. Three scenarios can be identified:

> Where there is **very low air turn over**, all three substances will give saturation of the air and hence almost the same rate of loss, which would be very low, controlled by the air turn over. This applies to storage of foam.

Where there is **high turn over**, diffusion in the polymer controls and the rates for TDCP and V6 will be only very slightly lower than those of TCPP. This applies to small particles.

In the situation of **moderate air turn over** the air saturation is reached quickest for lower volatility, since it requires less substance, and hence the loss rate will be slower for TDCP and V6, although it is hard to estimate by how much. This applies to in service loss of flexible foam, including furniture and automotive foam. The ESD applies a factor of 25 x lower rate for TDCP and V6 relative to TDCP, for all stages; it seems appropriate to use this factor for these applications, although it is empirical.

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6 LITERATURE SEARCH RESULTS FOR DIFFUSION OF ADDITIVES IN POLYMERS

Diffusion coefficients of additives in polymers. I. Correlation with geometric parameters
Author
Reynier, Alain; Dole, Patrice; Humbel, Stephane; Feigenbaum, Alexandre
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Publication Source
Journal of Applied Polymer Science (2001), 82(10), 2422-2433
Identifier-CODEN
JAPNAB
ISSN
0021-8995
Publisher
John Wiley & Sons, Inc.
Abstract
Diffusion coeffs. of a broad range of mols. (mol. wt. 100-800 g/mol) were measured in polypropylene by solid/solid contact methods at 40°. The behaviors of the various mols. are compared to those of linear alkanes. The diffusion coeffs. are correlated to parameters describing size, shape, and flexibility of the mols. The concept of weighted fractionated vol. is introduced using mol. modeling. It enables the classification of the mols. according to modes of mol. displacement (crawling, jumps, or dual mode).
Document Type
Journal
Language
English
Accession Number
2001:725418 CAPLUS
Document Number
136:20534
Cited Reference or Reference
(1) Aitken, A; Transport and solubility of isomeric paraffins in rubber; Trans Faraday Soc 1955, V51, P116
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(3) Berens, A; Diffusion of organic vapors at low concentrations in glassy PVC, polystyrene, and PMMA; J Membrane Sci 1982, V10, P283
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ANSWER 2 ©2002 ACS

Title

Prediction of worst case migration: presentation of a rigorous methodology

Author

Reynier, A.; Dole, P.; Feigenbaum, A.

Organization

Securite et Qualite des Emballages Alimentaires, Institut National de la Recherche Agronomique, Reims,
51687, Fr.

Publication Source

Food Addit. Contam. (1999), 16(4), 137-152

Identifier-CODEN

FACOEB

ISSN

0265-203X

Publisher

Taylor & Francis Ltd.

Abstract

An improvement of the Piringer model, allowing the prediction of a worst case migration from packaging to food is presented here. The authors are proposing other consts. for the calcn. of the upperbound value of the diffusion coeff., using exptl. data detd. by a film to film method. Considering the plasticizing effects of food simulants, a model involving the variation of the diffusion coeff. vs. space and time must be used. Future fields of investigation are discussed: the relationship between diffusion coeffs. and the vol. of the migrant (instead of molar mass), and the variation of diffusion coeff.

Document Type

Journal

Language

English

Accession Number

1999:223301 CAPLUS

Document Number

131:18109

Cited Reference or Reference

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Search: polymer and volatil* NOT doctype: p NOT determination AND language: english AND migrat* AND
doctype: gr

Display from CAplus database

ANSWER 6 ©2002 ACS

Title

The **migration** of non-**volatile** additives from plastics: New concepts from further experiments with model systems

Author
Adcock, L. H.
Organization
PIRA, Leatherhead, UK
Publication Source
Lect. - Int. Symp. Migr., 4th (1983), 245-65 Publisher: Dtsch. Unilever GmbH, Hamburg, Fed. Rep. Ger.
Identifier-CODEN
51LFA6
Abstract
A review and discussion with no refs. on the **migration** of additives from polymers into food in the absence of **polymer** swelling.
Document Type
Conference; **General Review**
Language
English
Accession Number
1984:422051 CAPLUS
Document Number
101:22051
Search: polymer and (leach* or migrat*)* NOT doctype: p
Search: polymer and (leach* or migrat*) NOT doctype: p
Search: polymer and (leach* or migrat*) NOT doctype: p AND additive*
Display from CAplus database
ANSWER 20 ©2002 ACS
Title
Polymer additive migration to foods-a direct comparison of experimental data and values calculated from **migration** models for polypropylene
Author
O'Brien, Anthony; Cooper, Ian
Organization
PIRA International, Surrey, KT22 7RU, UK
Publication Source
Food Addit. Contam. (2001), 18(4), 343-355
Identifier-CODEN
FACOEB
ISSN
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Publisher
Taylor & Francis Ltd.
Abstract
To reduce the amt. of compliance testing for food contact polymers the use of **migration** modeling is under discussion and being evaluated by an EU Commission funded project (Evaluation of **Migration** Models No. SMT4-CT98-7513). The work reported in this paper was exclusively funded by industry to provide data for the independent evaluation of a diffusion based model using eight different samples of polypropylene (PP) covering the range of polymers specification and five commonly used plastics **additive**s. One hundred and fifty exptl. **migration** data have been obtained in triplicate and used to evaluate a Fickian-based **migration** model in the prediction of specific **migration** of five **additives** into olive oil. All tests were conducted using olive oil, representing the most severe case for fatty foods, with test conditions of 2 h at 121°, 2 h at 70° and 10 days at 40°, representing short term exposures at high temps. and room temp. storage. Predicted **migration** values were calcd. using the Pringer **"Migratest** Lite" model by entering the measured initial concn. of **additive** in the polymers(Cp,0) in to the equations together with known variables such as **additive** mol. wt., temp. and exposure time. Where necessary the data generated in this study have been used to update the model. The results indicate the Piringer **migration** model, using the "exact" calcns. of the **Migratest** Lite

program, predicted **migration** values into olive oil close to, or in excess of, the exptl. results for >97% of the **migration** values generated in this study. For all measurements, the predicted **migration** from the **Migratest** Lite program was greater than 70% of the obsd. value. This study has identified the possibility that random co-polymers of propylene and ethylene give higher **migration** than other grades of polypropylenes and could be treated as a sep. case. However, further work on more samples of random co-polymers is required to confirm this finding.

Document Type
Journal
Language
English
Accession Number
2001:317289 CAPLUS
Document Number
135:76005
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Display from CAplus database
ANSWER 36 ©2002 ACS
Title
Comparison of techniques to measure **additive** diffusivity in **polymer** films
Author
McKibbin, John P.; Sankhe, Shilpa Y.; Bishop, Keisha A.; Hirt, Douglas E.
Organization
Department of Chemical Engineering and Center for Advanced Engineering Fibers and Films, Clemson University, Clemson, SC, 29634-0909, USA
Publication Source
Annu. Tech. Conf. - Soc. Plast. Eng. (2000), 58th(Vol. 3), 3497-3501
Identifier-CODEN
ACPED4
ISSN
0272-5223
Publisher
Society of Plastics Engineers
Abstract
The surfaces of a **polymer** film can be modified by allowing **additives** within the film to diffuse to the surfaces and accumulate there. To model the diffusion/accumulation process, it is necessary to accurately measure the diffusion coeff. of the **additive** in the **polyme**r. We have attempted to characterize the diffusivity of erucamide in LLDPE through several means: mass sorption ("diffusion in") and surface washing and ATR-FTIR ("diffusion out"). Expts. demonstrate that surface washing can provide inconsistent results. Mass sorption and ATR-FTIR provide comparable results, although emphasis is placed on the ATR-FTIR technique because the **migration** process more closely mimics the behavior of com. films.
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Accession Number
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Document Number
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ANSWER 57 ©2002 ACS
Title
Loss of high molecular weight, sterically hindered amines from polypropylene
Author
Mar'in, A. P.; Borzatta, V.; Bonora, M.; Greci, L.
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Dipartimento di Scienze dei Materiali e della Terra, Universita degli Studi di Ancona, Ancona, I-60131, Italy
Publication Source
J. Appl. Polym. Sci. (2000), 75(7), 897-903
Identifier-CODEN
JAPNAB
ISSN
0021-8995
Publisher
John Wiley & Sons, Inc.
Abstract
The loss from polypropylene (PP) of sterically hindered amines with mol. wt. ranging from 1364 to 2758 in heptane, chloroform, and methanol at room temp. was studied. The **additives** leak from **polymer** in heptane and in chloroform and some residual concn. remains in the **polyme**r; the stabilizers show slight **migration** in methanol. The rate of loss increases with **additive** concn. in the **polyme**r. The effect of solvent during washing out could be explained by its different soly. in PP resulting in changes in **polymer** chain mobility and **additive migration** from the **polyme**r.
Document Type
Journal
Language
English
Accession Number
2000:42106 CAPLUS
Document Number
132:181354
Cited Reference or Reference
(1) Al-Malaika, S; Toxicological implications associated with loss of antioxidants from plastics for use in food-packaging applications; Free Radicals and Food Additives 1991, P151
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Display from CAplus database
ANSWER 45 ©2002 ACS
Title

The estimation of partition coefficients, solubility coefficients, and permeability coefficients for organic molecules in polymers using group contribution methods
Author
Baner, A. L.
Organization
Friskies R and D Center Inc., St. Joseph, MO, 64503, USA
Publication Source
ACS Symp. Ser. (2000), 753(Food Packaging), 37-55
Identifier-CODEN
ACSMC8
ISSN
0097-6156
Publisher
American Chemical Society
Abstract
Partition, soly. and permeability coeffs. of org. substances are necessary for modeling mass transfer phenomena (aroma permeation and scalping, **polymer additive migratio**n) in polymeric food packaging systems. The uncountable no. of different **polyme**r/org. mol./food system combinations of interest coupled with the laborious and difficult exptl. work needed for measurement makes it desirable to explore the use of semiempirical thermodynamically-based group contribution methods to est. these parameters. The accuracy of partition, soly. and permeability coeffs. estns. using the UNIFAC, GCFLORY, ELBRO-FV, Regular Soln. and Retention Indexes methods are compared with exptl. data for aroma compds. and **polymer additives** in polyolefin, PET, nylon-6 and PVC polymers.
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Title
Polymer additive migration to foods-a direct comparison of experimental data and values calculated from
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Abstract
To reduce the amt. of compliance testing for food contact polymers the use of **migration** modeling has been proposed. This study was conducted to provide valid data for the independent evaluation of two such diffusion-based models using a range of different high d. polyethylene (HDPE) polymers and plastics **additive**s. Seventy-two exptl. **migration** data were obtained in triplicate and used to evaluate two Fickian-based **migration** models in the prediction of specific **migration** of four HDPE **additives** into olive oil. All tests were conducted using olive oil, representing the most severe case for fatty foods with test conditions of 2 h at 70°C, 6 h at 70°C, 10 days at 40°C representing short term exposures at high temps. and room temp. storage. Predicted **migration** values were calcd. by inserting the measured initial concn. of **additive** in the polymers (Cp,0) into the equations together with known variables such as **additive** mol. wt., temp. and exposure time. The results indicate that both models predict **migration** values into olive oil close to, or in excess of, the exptl. results. The Piringer **migration** model, using the "exact" calcns. of the **Migratest** Lite program, gave an overestimation for 83% of the **migration** values generated in this study. The highest overestimation was 3.7 times the measured value. For all measurements, the predicted **migration** from the **Migratest** Lite program was greater than 50% of the obsd. value. The FDA model was found more accurately to predict **migration** in most situations but underestimated **migration** more frequently. Differences in the **polymer** specification had little effect on specific **migration** of the **additives** investigated.
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Appendix C: Comparative property data Table for TCEP, TCPP, TDCP and V6

Reliabilities recorded in the table ('R') use the standard Klimisch code system.

IUCLID ref	Endpoint	TCEP	R [a]	TCPP	R	TDCP	R	V6	R	Comment on the data, QSAR or read-across
Physicochemical properties										
	Molecular weight	285.49		327.57		430.91		583.00		
2.1	Melting/freezing	<-70	1	<-20	1	<-20	1	<-50.5 (freezing point)	1	Not possible or necessary to obtain an exact value
2.2	Boiling	320 (decomp)	1	~288 (decomp)	1	~326 (decomp)	1	252 (decomp)	2	
2.3	Density at 20°C	1.4193 at 25°C	1	1.288	1	1.513	1	1.473	1	
2.4	Vapour pressure (Pa, 25ºC)	0.00114	1	1.4×10^{-3}	1	5.6×10^{-6}	1	*2.75×10^{-6}*		Value predicted for V6: EPIWIN[b] Version 3.05, modified Grain method
2.6.2	Surface tension	-	ND	-	ND	-	ND	-	ND	-
2.6.1	Water solubility (mg/l, 20ºC)	7820	1	1080		18.1	1	232	1	Data make a self-consistent set
2.5	Octanol-water partition coefficient	1.78	1	2.68		3.69	1	2.83	1	
2.7	Flashpoint (closed cup)	200°C	1	No flash up to 245°C, then decomposes	1	-	ND	191°C [c]	1	Read across could be considered for TDCP
2.9	Flammability, Pyrophoric properties	-	ND	-	ND	-	ND	-	ND	Not possible or necessary
2.10	Explosivity	-	ND	-	ND	-	ND	-	ND	Not possible or necessary
2.8	Autoignition temperatureºC	480	1	>400	1	513 [d]	4	>400 [c]	1	

IUCLID ref	Endpoint	TCEP	R [a]	TCPP	R	TDCP	R	V6	R	Comment on the data, QSAR or read-across
2.11	Oxidising properties	-	ND	-	ND	-	ND	-	ND	Not possible or necessary
Environmental fate and behaviour										
3.5	Ready biodegradability	No	1	No	2	No	2	No (not GLP)	2	Weight of evidence is that none of these is readily biodegradable
3.5	Inherent biodegradability	No (based on two tests, one of short duration)	1	Evidence of partial degradation	2	No	2	Evidence of partial degradation (not GLP)	2	A consistent picture of lack of ready degradability. The mono-chloro chain substances show some degradation after acclimation; it cannot be assumed that TDCP would behave similarly.
	Other biodegradation results	Not anaerobically biodegradable Not degraded by soil micro-organisms	1			Not degraded by soil micro-organisms	1			
3.7	Bioaccumulation in fish	0.6 - 5.1 (From 3 tests, with *Cyprinus carpio*, *Carassius auratus* and *Oryzias latipes*)	1	–0.8 – 4.6 *Cyprinus carpio*	2	0.3 – 89 (From 2 tests, with *Cyprinus carpio* and *Oryzias latipes*)	2	*50.8*		Value predicted for V6: Veith *et al*, 1979. Read-across not recommended due to possible importance of metabolism; no available evidence suggests that high BCF values are likely.
3.1.2	Hydrolysis pH 7	t1/2 >1 year	1	t1/2 >1 year	1	t1/2 >1 year	1	t1/2 >1 year	1	

IUCLID ref	Endpoint	TCEP	R [a]	TCPP	R	TDCP	R	V6	R	Comment on the data, QSAR or read-across
3.3	Log Koc Log Koc (estimated by HPLC method) (Estimated using TGD QSAR for TCEP)	 *2.04* (Koc estimated from log Kow)	 1	2.24 (Koc = 174, calculated from TDCP value) 2.76	1 1	3.25 (OECD 106) (Koc = 1780) 4.09	1 1	2.39 (Koc = 245, calculated from TDCP value) 4.04	1 1	Full study more reliable than HPLC estimation.
Ecotoxicity (most sensitive values only reported, test species and test guidelines (where known) are reported in italics)										
4.1	Acute toxicity to fish (mg/l)	LC50 = 90 *Carassius auratus*	1	LC50 = 51 *P. promelas*	1	LC50 = 1.1 O. mykiss OECD 203	1	LC50 = 52 *O. mykiss* *OECD 203*	1	
	QSAR[b] (Esters) acute toxicity to fish (96 h LC_{50})	*36*	*2*	*21*	*2*	*8.1*	*2*	*32*	*2*	ECOSAR Program (v0.99h). The QSAR estimates are of the same order of magnitude as the measured data, but tend to over-predict toxicity slightly (with the exception of TDCP).

IUCLID ref	Endpoint	TCEP	R [a]	TCPP	R	TDCP	R	V6	R	Comment on the data, QSAR or read-across
	QSAR[b] (Phosphate esters) acute toxicity to fish (96 h LC_{50})	*19*	*2*	*11*	*2*	*4.5*	*2*	*17*	*2*	ECOSAR Program (v0.99h). The QSAR estimates are of the same order of magnitude as the measured data, but tend to over-predict toxicity slightly (with the exception of TDCP).
4.2	Acute toxicity to invertebrates (48 h EC_{50} in mg/l)	EC50 = 235 (24 h) *D. magna*	1	EC50 = 131 *D. magna*	1	EC50 = 3.8 *D. magna* *OECD 202*	1	EC50 = 42 *D. magna* *OECD 202*	1	
	QSAR[b] (Esters) acute toxicity to invertebrates (48 h LC_{50})	*230*	*2*	*63*	*2*	*9.9*	*2*	*81*	*2*	ECOSAR Program (v0.99h). The QSAR estimates are of the same order of magnitude as the measured data, but tend to under-predict toxicity slightly (with the exception of TCPP).
4.3	Acute toxicity to algae (72 h E_rC_{50} in mg/l)	ErC50 = 3.6 *Scenedesmus subspicata*	1	ErC50 = 82 *Pseudokirchneriella subcapitata* OECD 201	1	ErC50 = 2.8 *Pseudokirchneriella subcapitata* *OECD 201*	1	ErC50 = 35 *Pseudokirchneriella subcapitata* *OECD 201*	1	TCEP result appears out of line with the other results

IUCLID ref	Endpoint	TCEP	R [a]	TCPP	R	TDCP	R	V6	R	Comment on the data, QSAR or read-across
	QSAR[b] (Esters) toxicity to algae (96 h EC_{50})	*2.9*	*2*	*1.8*	*2*	*0.69*	*2*	*2.6*	*2*	ECOSAR Program (v0.99h). The selected QSAR appears to over-predict toxicity in general
4.5.1	Chronic toxicity to fish (mg/l)	-	ND	-	ND	-	ND	-	ND	
	QSAR[b] (Esters) chronic toxicity to fish	*16*	*2*	*5.2*	*2*	*1.0*	*2*	*7.0*	*2*	ECOSAR Program (v0.99h)
4.5.2	Chronic toxicity to invertebrates (mg/l, 21-day repro test)	NOEC = 13 *D. magna*	1	NOEC = 32 *D. magna* *OECD 202*	1	NOEC = 0.5 *D. magna* *OECD 211*	1	NOEC ≥3.68 *D. magna* *OECD 211*	1	
	QSAR (Neutral organics) chronic toxicity to invertebrates			NOEC (reproduction) = 4.3	2	NOEC (reproduction) = 1.1	2	NOEC (reproduction) = 6.0	2	ECOSAR Program (v0.99h)
4.3	Chronic toxicity to algae (72 h growth rate results in mg/l)	48h ErC10 = 0.65 *Scenedesmus subspicatus*	1	ErC10 (72hr) = 42 *Pseudokirchneriella subcapitata* OECD 201	1	ErC10 (72hr) = 2.3 *Pseudokirchneriella subcapitata* OECD 201	1	NOEC (96hr) = 10 *Pseudokirchneriella subcapitata* *OECD 201*	1	
	QSAR[b] (Esters) chronic toxicity to algae (96 h NOEC)	*2.2*	*2*	*1.4*	*2*	*0.55*	*2*	*2.1*	*2*	ECOSAR Program (v0.99h)
	Toxicity to WWTP micro-organisms (mg/l)	IC50 = 3200 Activated sludge OECD 209	1	IC50 = 784 Activated sludge ISO 8192	1	IC50 = >10000 Activated sludge	2	IC50 = >1000 Activated sludge OECD 209	1	

IUCLID ref	Endpoint	TCEP	R [a]	TCPP	R	TDCP	R	V6	R	Comment on the data, QSAR or read-across
						OECD 209				
4.6.1	Toxicity to sediment dwelling organisms (mg/kg dw)[e,f]					28 d NOEC = 10.6[g] (10)[2.2] 28 d NOEC = 8.8[h] (8.3)[1.8] 28 d NOEC = 3.9[i] (3.7)[0.8] *Chironomus riparius OECD 218*	1			
	Toxicity to higher plants (mg/kg dw)	EC50 = 64 NOEC = 10 *Avena sativa Modified OECD 208*	1	NOEC = 17 *Lactuca sativa OECD 208*	1	NOEC = 19.3 *Sinapis alba OECD 208*	1	NOEC = 17 (Read-across from TCPP)		
	Toxicity to earthworms (mg/kg dw)[j]	14 d NOEC = 580 *Eisenia andrei*	1	14 d LC50 = 97 (33) OECD 207 56 d NOEC = 53 (18) *Eisenia foetida OECD draft guideline (January 2000): Earthworm Reproduction Test*	1	14 d LC50 = 130 (44) OECD 207 57 d NOEC = 9.6 (3.3) *Eisenia foetida OECD draft guideline (January*	1	14 d LC50 >1000 (>340) 14 d NOEC >1000 (>340) (not GLP) *Eisenia foetida OECD207*	1	

IUCLID ref	Endpoint	TCEP	R [a]	TCPP	R	TDCP	R	V6	R	Comment on the data, QSAR or read-across
						2000): Earthworm Reproduction Test				
	Toxicity to other soil invertebrates (mg/kg dw)	28d LC50 = 66.5 (mortality) 28d LC10 = 19.3 (mortality) 28d EC10 = 44.6 (repro) (*Folsomia candida* springtail)	1	-	ND	-	ND	-	ND	
	Toxicity to soil micro-organisms	Inhibition 15-42% at 5-50 mg/kg dw in various soils.	1	28 d NOEC = ≥128 mg/kg ww Nitrifying micro-organisms in sandy loam soil (Read-across from TDCP)		28 d NOEC = ≥128 mg/kg ww Nitrifying micro-organisms (species not stated) in sandy loam soil OECD 216	1	28 d NOEC = ≥128 mg/kg ww Nitrifying micro-organisms in sandy loam soil (Read-across from TDCP)		
	Toxicity to birds (g/kg)	Neurotoxicity not observed at 14.2 g/kg *Gallus domesticus*	1	-	ND	-	ND	-	ND	

Notes:

ND – not determined (no data available)

a The TCEP ESR RAR does not state data reliabilities. It has been assumed here that values used in the risk assessment must be considered to be of high reliability. This is useful to provide a point of reference for comparison with the reliability of available data on the other three substances.

b SRC Syracuse Research Corporation programs for estimating properties

c subject to clarification of test substance composition

d Industry considers result to be invalid but reason is unknown

e Values in (parentheses) have been corrected to standard organic matter content of 5.0%

f Values in [parentheses] have been corrected to standard organic matter content of 5.0% and expressed as wet weight

g Based on initial (day 0) measured exposure concentrations in sediment

h Based on geometric mean of measured exposure concentrations in sediment on days 0 and 3

i Based on geometric mean of measured exposure concentrations in sediment on days 0 and 28

j Values in parentheses have been corrected to standard organic matter content of 3.4%

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The evaluation considers the emissions and the resulting exposure to the environment and the human populations in all life cycle steps. Following the exposure assessment, the environmental risk characterisation for each protection goal in the aquatic, terrestrial and atmospheric compartment has been determined. For human health the scenarios for occupational exposure, consumer exposure and humans exposed via the environment have been examined and the possible risks have been identified.