European Union Risk Assessment Report

TRIS(2-CHLORO-1-METHYLETHYL) PHOSPHATE (TCPP)

CAS No: 13674-84-5

EINECS No: 237-158-7

RISK ASSESSMENT

LEGAL NOTICE

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RISK ASSESSMENT

May 2008

Ireland (lead) and United Kingdom

Rapporteur for the risk assessment of TCPP 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**.

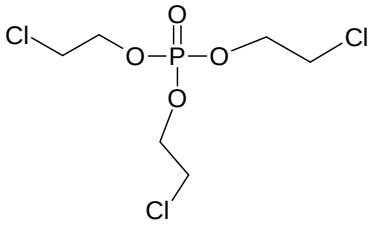
Table i Priority scores of chlorinated alkyl phosphate esters

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

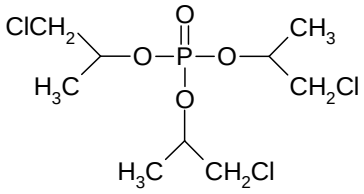
Note: A priority score of 100 is the highest priority.

The substance structures are shown below.

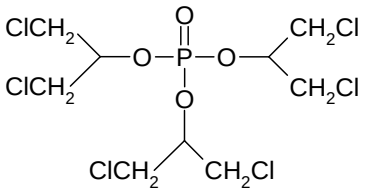
Tris(2-chloroethyl) phosphate (TCEP)



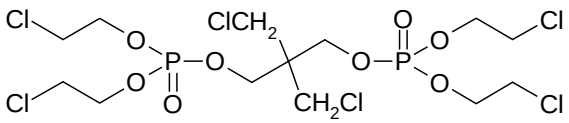
Tris(2-chloro-1-methylethyl) phosphate (TCPP)[4]



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 (KEMI 1996). 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 are commercially available as such, or produced as isolated products, by EU manufacturers. 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. At present, none of these documents are published, and so references are informal only.

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

Much of the data upon which the 1996 SIAR for TCPP was based are now considered invalid or simply out of date, having been superseded by new measured data. It is effectively superseded by this assessment and is not directly referred to herein.

OVERALL RESULTS OF THE RISK ASSESSMENT[5]

CAS Number: 13674-84-5
EINECS Number: 237-158-7
IUPAC Name: Tris(2-chloro-1-methylethyl) 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 to all compartments for all local life cycle stages, and at the regional scale in all compartments.

With regard to secondary poisoning, 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, due to the low ratios and lack of any significant bioaccumulation potential of TCPP, it is reasonable to conclude that there are no risks.

TCPP does not meet all of the PBT criteria (it meets the screening criteria for P or vP).

Human Health

Human health (toxicity)

Workers

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 TCPP (worker scenario 1) in relation to effects on fertility and developmental toxicity.

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, repeated dose toxicity, mutagenicity and carcinogenicity.

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 (ii) applies to typical dermal exposure and inhalation exposures, both reasonable worst case and typical, during the manufacture of TCPP (worker scenario 1) in relation to effects on fertility and developmental toxicity.

Conclusion (ii) applies to all other worker exposure scenarios (worker scenarios 2-10) for both reasonable worst case and typical exposures in relation to effects on fertility and developmental toxicity.

Consumers

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 in relation to all toxicological endpoints.

Humans exposed via the 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 to both regional and local exposures in relation to all toxicological endpoints.

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 in relation to all toxicological endpoints.

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 presents confidential details of the release scenarios for production and uses of TCPP used in the risk assessment. It is referred to in the text as the 'Confidential Annex'.

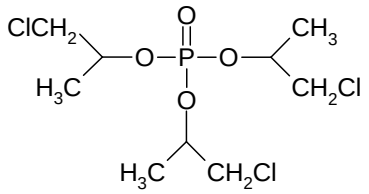
Confidential analytical data annex: this presents confidential details of the purity and impurities of commercially available TCPP together with various spectra. It is referred to in the text as the 'confidential annex of compositional data'.

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

1 GENERAL SUBSTANCE INFORMATION

1.1 IDENTIFICATION OF THE SUBSTANCE

CAS Number: 13674-84-5
EINECS Number: 237-158-7
IUPAC Name: Tris(2-chloro-1-methylethyl) phosphate
Molecular formula: $C_9H_{18}Cl_3O_4P$
Structural formula:



Molecular weight: 327.57
Synonyms: 2-Propanol, 1-chloro, phosphate (3:1)
Tris(monochloroisopropyl) phosphate (TMCP)
Tris(2-chloroisopropyl) phosphate (TCIP)
Phosphoric acid, tris(2-chloro-1-methylethyl) ester
Tris(beta-chloroisopropyl) phosphate
1-Chloro-2-propanol phosphate (3:1)
TCPP: this common acronym is used throughout this report

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

It can be seen from the structural formula that TCPP has chiral centres. The producers have confirmed that TCPP is a mixture of stereoisomers.

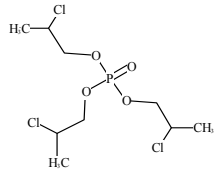
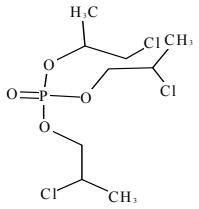
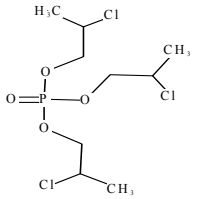
1.2 PURITY/IMPURITIES, ADDITIVES

Isomers

The flame retardant product supplied in the EU, marketed as TCPP (or other synonyms as given above), is actually a reaction mixture containing four isomers. The individual isomers in this reaction mixture are not separated or marketed. The individual components are never produced as such. These data are true for TCPP produced by all EU manufacturers.

TCPP as shown in the accompanying diagrams is the tris(1-chloro-2-propyl) form. The CAS number 13674-84-5 is used for this structure and also for the mixture of isomers as commercially produced. The 1-chloro-2-propyl- can be replaced up to three times by 2-chloro-1-propyl (i.e. an n- hydrocarbon chain). Therefore three isomers of the main component are possible, although tris (2-chloro-1-propyl)phosphate is only present in trace levels.

Table 1.1 Compositional description for TCPP across all commercial products

Name	Structural diagram	EINECS number	CAS number	% (w/w)
Tris(2-chloro-1-methylethyl) phosphate	Shown above	237-158-7	13674-84-5	50 – 85
Bis(1-chloro-2-propyl)-2-chloropropyl phosphate		-	76025-08-6	15 – 40
Bis(2-chloropropyl)-1-chloro-2-propyl phosphate		-	76649-15-5	<15
Tris(2-chloropropyl) phosphate		228-150-4	6145-73-9	<1

The assumption is made that all isomers have identical properties in respect of risk assessment. The assumption is justified in part by the fact that they exhibit very similar chromatographic properties, even under conditions optimised to separate them. Predicted physicochemical properties differ to only a small extent. Modelling procedures required for predicted environmental concentration (PEC) values for the separate isomers would not be affected by the small differences that are expected to apply. Testing has been carried out using the commercial product, i.e. a mixture of isomers, in a composite sample.

There are differences in the isomer content from each supplier, but these are not important given that the properties of the isomers are expected to be very similar.

Purity

A typical purity (total of the four isomers) is >97.9%. All testing described in this report is for the commercial product.

Impurities

The impurity profile of the commercial product TCPP is specific to individual manufacturers. Details are given in the confidential annex of compositional data. It is not likely that the impurities will have had particular influence on any of the results obtained.

Additives

No additives are used.

1.3 PHYSICO-CHEMICAL PROPERTIES

The physico-chemical property values of TCPP that have been reviewed are summarised in **Table 1.2**. The values selected for use in the risk assessment are as follows:

Melting / freezing

The preferred value is <-20°C, which was obtained in a modern GLP study (Cuthbert and Mullee 2002a) in accordance with Directive 92/69/EC.

Boiling

The preferred value is 288°C, although decomposition occurred, which was obtained in a modern GLP study (Cuthbert and Mullee, 2002a) in accordance with Directive 92/69/EC.

Density at 20°C

The preferred value of the relative density is 1.288, which was obtained by the pycnometer method in a modern GLP study (Cuthbert and Mullee 2002a) in accordance with Directive 92/69/EC.

Vapour pressure

The preferred value is 1.4 x 10^{-3} Pa at 25°C, which was obtained by the vapour pressure balance method in a modern GLP study (Tremain 2002) in accordance with Directive 92/69/EC.

Surface tension

Based upon the chemical structure and the known physico-chemical properties of the substance of concern, TCPP 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 1080 mg/l at 20°C, which was obtained by the flask method in a modern GLP study (Cuthbert and Mullee 2002b) in accordance with Directive 92/69/EC.

Octanol-water partition coefficient

The preferred value is log K_{ow} = 2.68±0.36, which was obtained by the HPLC estimation method[6] in a modern GLP study (Cuthbert and Mullee 2002b) in accordance with Directive 92/69/EC. The ± value is the 95% confidence limit.

[6] 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 TCPP 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 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.

Flash point (closed cup)

The most reliable value shows no flash up to 245°C, derived in a GLP compliant study (Tremain and Bartlett 1994), although the composition of the sample used is not known.

Flammability (in contact with water)

Based on the known chemical and physical properties of the substance TCPP 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 TCPP 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 TCPP 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.

Autoignition temperature

A single reliable study giving an autoignition temperature of >400°C, derived in a GLP compliant study (Tremain and Bartlett 1994) is available although the composition of the sample used is not known.

Oxidising properties

By reference to the structural formula, it can be seen that TCPP 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 TCPP.

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.

$$H = \frac{\text{Molecular weight} * \text{Vapour pressure (Pa)}}{\text{Water solubility (mg/l)}}$$

A value of 3.96×10^{-4} $Pa.m^3/mol$ is used in the risk assessment, based on EUSES adjustments of the properties for temperature dependence.

The results in **Table 1.2** below are taken directly from the industry submission unless stated otherwise.

Table 1.2 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	-42°C pour point	(4) not assignable	Coomber, 1993. Result only
	<-30°C pour point	(4) not assignable	Result only; of unknown source
	<-20°C**	(1) valid without restriction	Cuthbert and Mullee, 2002a
Boiling point	341.5°C	(4) not assignable	Coomber, 1993. Result only
	Ca. 288°C (decomp.)**	(1) valid without restriction	Cuthbert and Mullee, 2002a
Relative density	1.2932 Specific gravity 20/20	(4) not assignable	Coomber, 1993. Result only
	1.29	(4) not assignable	Result only; of unknown source; IPCS209 X
	1.288 at 20°C**	(1) valid without restriction	Cuthbert and Mullee, 2002a
Vapour pressure	<689 Pa	(4) not assignable	Result only; of unknown source.
	Ca. 3.3 Pa at 20°C	(4) not assignable	Krawetz, 2000. Result certificate only
	<100 Pa	(4) not assignable	Result only; of unknown source
	3590 Pa	(4) not assignable	Rhodia MSDS
	100 Pa	(4) not assignable	Akzo MSDS
	3.3 Pa	(4) not assignable	
	1.4×10^{-3} Pa at 25°C**	(1) valid without restriction	Tremain, 2002. The result is consistent with the chemical structure of the main component and its isomers, and the other properties, in particular the boiling point.
Surface tension			No study available, but not expected to exhibit surface activity
Water solubility	1600 mg/l	(4) not assignable	Robson, 1994. Summary of methods and results only; no information on analytical method.
	900 mg/l	(4) not assignable	Bayer MSDS
	1080 mg/l at 20°C **	(1) valid without restriction	Cuthbert and Mullee, 2002b
Partition coefficient n-octanol/water (log value)	3.33	(4) not assignable	Robson, 1994. Summary of methods and results only; no information on analytical method or stock concentration.
	2.59	(4) not assignable	CITI, 1992. Result only; MITI experimental result
	2.68±0.36**	(1) valid without restriction	Cuthbert and Mullee, 2002b

Property	Value	Reliability[1]	Comments
	2.89	(2) valid with restrictions	Accepted calculation method (SRC KOWWIN v. 1.67)
Granulometry			
Conversion factors			
Flash point	**No flash up to 245°C, then decomposes**	(2) valid with restrictions	Tremain and Bartlett, 1994. Information about the composition of the sample used is not available
	199°C	(4) not assignable	Coomber, 1993. Result only
	185°C	(4) not assignable	Result only
Autoflammability	>400°C	(2) valid with restrictions	Tremain and Bartlett, 1994. Information about the composition of the sample used is not available
Flammability	Non-flammable	(4) not assignable	Not expected to be flammable. Derogation accepted by TC NES
Explosive properties	Not explosive	(4) not assignable	Not expected to be explosive. Derogation accepted by TC NES
Oxidizing properties	No oxidising properties	(4) not assignable	Not expected to be oxidising. Derogation accepted by TC NES
Viscosity (kinematic viscosity)	68.5 cP at 20°C	(4) not assignable	Coomber, 1993. Result only.
Refractive index	1.4642 at 20°C	(4) not assignable	Coomber, 1993. Result only.
Henry's law constant	3.96 x 10^{-4} 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 97.9% (total of the four isomers), derived from recent representative commercial products from the main producers.

[1] Klimisch code

1.4 CLASSIFICATION

1.4.1 Current classification

A classification of not dangerous for the environment (not classified) was agreed at EU level in 2005[7].

1.4.1.1 Basis of classification for the environment

Data presented in this report are consistent with no classification for the environment being necessary. The fish, *Daphnia* and algae acute E(L)C_{50} values all fall in the range 10 to 100 mg/l, and there is no evidence of ready degradability in standard tests. However, R52-53 is not applicable for TCPP for the reasons outlined below:

[7] 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

- The acute effect concentrations range from 51 to 131 mg/l (fish and *Daphnia* respectively). The difference in acute susceptibility across the taxa is therefore quite small (approximately 3-fold).
- Reliable chronic NOECs are available for invertebrates and algae and both are well above 1 mg/l (32 and 23 mg/l respectively). The acute-to-chronic ratios are 4 and 3.6 respectively.
- The tests have been conducted well below the water solubility limit (1080 mg/l), and the low measured BCF values do not suggest that the substance will accumulate over long periods. The acute toxicity therefore probably reflects the effect of uptake at steady state (i.e. not just partial uptake).
- There is reasonable agreement between the measured acute fish LC_{50} (51 mg/l) and QSAR predictions (11-21 mg/l, using SRC ECOSAR with measured physicochemical data entered). The substance therefore appears to be behaving in a predictable way.
- There is no indication of neurotoxicity in this chemical class from mammalian and avian studies.
- There is therefore no reason to suppose that there will be a significant difference in chronic effects in fish compared to the other taxa. Applying the *Daphnia* acute-to-chronic ratio to the acute fish result would give a NOEC of approximately 4.5 mg/l. This is very similar to the QSAR estimate of 5.2 mg/l (using SRC ECOSAR with measured physicochemical data entered).
- The acute-to-chronic ratio would be above 50 if the fish NOEC were below 1 mg/l, which is clearly out of line with the observations for *Daphnia* and algae.

Given these considerations it is unlikely that TCPP would be chronically toxic to fish at $\leq$1 mg/l and testing to confirm this assertion could not be justified on animal welfare grounds. TCPP should not therefore be classified.

1.4.2 Proposed classification

1.4.2.1 Basis of proposed classification for human health

Regarding human health, the data presented are consistent with the classification R22 (harmful if swallowed). This is based on the fact that the majority of LD_{50} values determined from acute oral toxicity studies were <2000 mg/kg.

There are no carcinogenicity data for TCPP. In order to address the data gap for this endpoint, a qualitative read-across to the structurally similar substances, TDCP and TCEP was performed, details of which are presented in Appendix D to this report. TDCP and TCEP are considered to be non-genotoxic carcinogens and have agreed classifications of Carc Cat 3 R40[8].

It is considered that there is sufficient information from the structures, physical chemical properties, toxicokinetics and mutagenic profiles of TCEP, TDCP and TCPP to support a qualitative read-across to address the hazard and risk assessment for the carcinogenicity

[8] 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.

endpoint for TCPP. However, it is accepted that there are some differences in the metabolism, the target organs and the severity of the effects observed with the three substances. Also, there are no insights into an underlying mode of action for TCEP and TDCP which would make a prediction on a relatively potency of TCPP possible. Therefore, a quantative read-across to carcinogenicity data of either TCEP or TDCP was not performed.

The above approach can be considered to be precautionary, in order to complete a risk characterisation for this endpoint and is preferred to a situation in which a data gap would trigger the need for a cancer bioassay. However, as the mechanism of tumour formation in either TDCP or TCEP is not understood, and given that the effects seen in the repeated dose toxicity study with TCPP were slight, it is considered that there is not sufficient evidence to classify TCPP for carcinogenicity and therefore no classification for this endpoint is proposed.

In the two generation reproductive toxicity study with TCPP, an increase in oestrus cycle length and a decrease in uterus weight were observed in all dosed females in F0 generation and in high dose females in F1. The mean number of oestrus cycles was also increased in high dose animals of both generations. Effects were also noted on ovarian weights in all high dose females and pituitary weights in high dose females in F0 and all dosed females in F1. It is noted that all organ weight changes occurred in the absence of any histopathological changes, and it is accepted that uterine weight can fluctuate during the oestrus cycle. Therefore, the effects observed may be due to normal variation in cycling females. Based on the above, this is considered to be a borderline case between classification as Repro Cat 3, R62 and no classification for effects on fertility.

In the same study, an increased number of runts was observed in all dose groups and a decrease in the mean number of pups delivered was observed in the mid dose group of F1 and the high dose groups of both generations. A decrease in pup weight was also noted during the lactation period. Pup mortality (PN1-4) was also increased in the low and high dose groups of F0 and in the high dose group of F1 (although the latter was mainly due to the loss of one litter of a single dam on PN4). Based on the above, it is possible that TCPP has an effect on the developing pups. Therefore, this is considered to be a borderline case between classification as Repro Cat 3, R63 and no classification for developmental toxicity.

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

2 GENERAL INFORMATION ON EXPOSURE

Due to commercial confidentiality it has not been possible to provide information on all life cycle stages in the main report. Whilst there are several producers, and many of the life cycle stages are well known in the industry, information concerning some uses is specific to one or two companies. Further information on the life cycle is given in the confidential use pattern and exposure 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 substances.

The four producers (see below, along with Clariant) have participated as an industry consortium on the risk assessment of TCPP. This consortium assisted in the early stages of the study by sending out a questionnaire to users of TCPP. The results were collated confidentially by the Rapporteur. More recently, the consortium has assisted with further consultation with the confidential downstream users. Relevant industry organisations (ISOPA, the European Di-isocyanate and Polyol Producers' Association; EUROPUR, the European Association of Flexible Polyurethane Foam Blocks Manufacturers; and BING, the Federation of European Rigid Polyurethane Foam Associations) have acted as a focal point for input from downstream users of TCPP.

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 >95% 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 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.

2.1 PRODUCTION

2.1.1 Production processes

All commercial TCPP is produced by the reaction of phosphorus oxychloride with propylene oxide followed by purification (WHO 1998). Both batch and continuous processes can be used in the manufacture of TCPP (UNEP 1999).

Data on the TCPP production process has been provided by three of the four producers, which indicate that production is carried out along the lines suggested in UNEP (1999). The reaction is carried out in a closed reactor. The crude product is washed and dehydrated in a closed vessel to remove acidic impurities and residual catalyst. All transfers are done using closed lines. The product is then filtered, transferred, and packaged using sealed pumps

through closed lines. Storage is in closed vessels under nitrogen to exclude moisture and oxygen.

2.1.2 Production capacity

2.1.2.1 Production

There are four producers of TCPP in the EU:

- Supresta, whose TCPP business was owned earlier in the assessment process by Akzo Nobel
- Lanxess, whose TCPP business was owned earlier in the assessment process by Bayer
- BASF, which sells through Elastogran
- Albemarle, whose TCPP business was owned earlier in the assessment process by Rhodia, and previously Albright and Wilson.

Total EU production of TCPP in the year 2000 was 36,000 tonnes, with production taking place at three sites in Germany and one in the UK. Between 1998 and 2003, production has increased significantly 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 five 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.

Discussions with the Phosphate Ester Flame Retardant Consortium (PEFRC) indicate that there is unlikely to be any future increase due to substitution for TCEP, replacement having been completed for all the applications for which replacement is possible.

2.1.2.2 Imports and Exports

8,304 tonnes of TCPP were imported into the EU in 2001. Data provided by CEFIC (pers. comm. 19th February 2002, CEFIC) indicate that most of this was imported by companies other than the four main producers and sourced in Russia. Consultation with members of the Industry Consortium originally indicated Russia to be the only source of non-Consortium imports (pers. comm. 27th February 2002, Akzo Nobel and pers. comm. 28th February 2002, PEFRC), though it has since been indicated that the main non-consortium TCPP imports have altered from Russia to Poland (EFRAx 2006a and b).

A total of 6,211 tonnes of TCPP was exported from the EU in the year 2000. It is assumed that no handling (e.g. repackaging) takes place and that no losses of TCPP arise through import or export.

Table 2.1 EU production and consumption of TCPP in the year 2000

Life Cycle Stage	Tonnes
Production	36,038
Imports	8,304
Exports	6,211

A further quantity of 1,201 tonnes of TCPP is believed to be imported into the EU in finished goods and this is accounted for in the risk assessment:

- Up to 680 tonnes per annum is imported into the UK in furniture sourced from outside the EU (see Section 2.2.2.2.6)
- Around 500 tonnes of TCPP is imported in canned (one component) foams (see Section 2.2.2.5.6)
- It is possible that finished goods containing TCPP 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.

2.2 USES

2.2.1 Introduction

TCPP 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.

Over 40,000 tonnes of TCPP were consumed in the EU in the year 2000. Most TCPP (over 98%) is used as a flame retardant in the production of polyurethane (PUR) for use in construction and furniture.

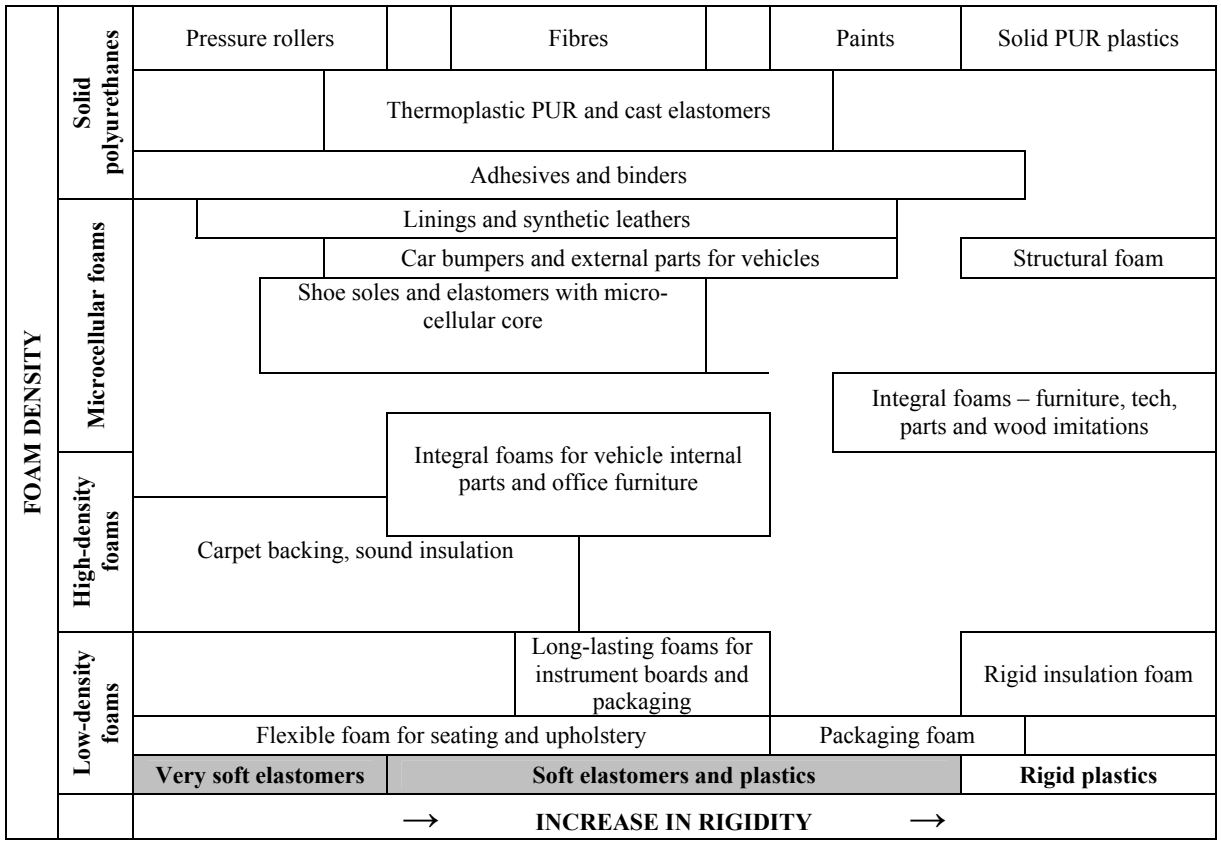
PUR is produced from the reaction of di-isocyanates with polyols. TCPP can be added to polyols in the production of PUR systems (formulations, refer to section 2.2.2.1; around 50-60% of TCPP is used in this way), or added directly at the point of foaming.

Most TCPP is used in rigid PUR foam (over 80%) mainly for construction applications. The remaining PUR applications are accounted for by flexible foam (over 17%), used in upholstery and bedding for the UK and Irish markets. TCPP tends not to be used in flexible PUR for automotive applications owing to its volatility and fogging potential.

Use of TCPP 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 across all producers confirms that TCPP is not widely used outside the PUR industry.

Figure 2.1 below, which is a simplified diagram taken from Koschade (2002), shows the variation of end uses associated with PUR over a range of density and rigidity.

Figure 2.1 Examples of the application of polyurethanes by density and rigidity



The life cycle stages considered in this assessment are reported in **Table 2.2** and shown in **Figure 2.2**. Further information including information on the confidential life cycle stages is given in the Confidential Annex. The tonnages used in the risk assessment are principally derived from survey data relating to the consumption in the year 2000.

As all members of the industry consortium have provided a detailed breakdown of tonnage it is believed that the life cycle is well defined. However, no data was provided by CEFIC concerning the downstream uses of the TCPP imported from Russia (the main non consortium TCPP imports have since altered from Russia to Poland) (see section 2.1.2.2). In addition, some TCPP is sold by members of the industry consortium to traders and distributors. Together these account for over 10% of the TCPP tonnage. In the absence of information concerning the downstream uses of this TCPP it is assumed that this is consumed in Uses A to E in the same proportions as for the TCPP arising from uses specified by the Industry Consortium.

Table 2.2 Use pattern for TCPP

Ref. Env[1]	Ref. HH[2]	Industry Category	Use category	Description	Percentage of total use	Tonnage
A	5	11	22	PUR systems (formulation)	[51.1%][3]	20450
B	2,3	11	22	PUR foam for use in furniture	17.0%	6800
C	7,8	11	22	Rigid PUR foam for use in construction	66.5%	26,650
D	6	11	22	Spray foams	9.6%	3850
E	9,10	11	22	One component foams	4.7%	1900
F	-	Confidential	22	Confidential	<2.5%	
G	-	Confidential	22	Confidential		
H	-	Confidential	22	Confidential		
I	-	Confidential	22	Confidential		
J	-	Confidential	22	Confidential		
K	-	Confidential	22	Confidential		
L	-	Confidential	22	Confidential		
M	-	Confidential	47	Confidential		
N	-	Confidential	22	Confidential		
P	-	Confidential	22	Confidential		
O	4	11	22	Rebonding of flexible foam	This is a form of recycling	
Q	-	11	22	Adhesive pressing of waste rigid foam	This is a form of recycling	
R	-	11	22	Recycling as loose crumb	This is a form of recycling	
Total					100%[3]	

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 – Since systems go on to be used in certain other life cycle stages, the tonnage is not included in the summation.

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 (year 2000) and Denmark account for 1,312 tonnes of TCPP (around 3.5% of EU consumption in the year 2000). Data for Sweden in 1999 are for TDCP combined with TCPP and are therefore of limited use.

It is notable that the industry's view is that not all uses here are current or recommended uses: in particular foaming agent, concrete, intermediate plastic manufacture, metal products, wood applications and cement are considered not to apply (EFRA, 2006).

Table 2.3 Product register and SPIN data

Country	Tonnage	No. of Products[a]	Concen-tration[b]	Description	
Denmark	499	15	5 –10% (4) 10-20% (9) 20-100% (2)	Fillers	Building and civil engineering Manufacture of rubber and plastic products Manufacture of chemicals and chemical products Manufacture of machinery and equipment Manufacture of transport equipment Private household
	277	22	1 –10% (9) 10-20% (10) 20-100% (3)	Insulating materials	
	190	3	5-50%	Foaming agents	
	185	13	5-10% (8) 10-50% (5)	Adhesives, binding agents	
	23	7	5-20% (7)	Construction materials	
Denmark 2001 (SPIN)	704.2	55		287.7 t (16 preparations)	Manufacture of rubber and plastic products
				42.4 t (7 preparations)	Manufacture of machinery and equipment
				53.1 t (25 preparations)	Construction
				6.6 t (4 preparations)	Private households with employed persons
Denmark 2000 (SPIN)	553.1	50		287.7 t (14 preparations)	Manufacture of rubber and plastic products
				42.4 t (7 preparations)	Manufacture of machinery and equipment
				59.7 t (23 preparations)	Construction
				10.2 t (4 preparations)	Private households with employed persons
Finland 2001 (SPIN)	812.9	13		775.0 t (6 preparations)	Manufacture of rubber and plastic products
					Manufacture of fabricated metal products, except machinery and equipment
				17.3 t (4 preparations)	Construction
Finland 2000 (SPIN)	Not stated	11		4 preparations	Manufacture of rubber and plastic products
				1 preparation	Manufacture of electrical machinery and apparatus
				4 preparations	Construction
Sweden[c] 1999	350	45 (9)	-	Plastics, concrete, textiles and insulation materials	
Sweden 2000	-	3 (0)	-	Use: raw material (fire prevention additive in plastics). Trade code: Industry for plastic products; industry for other chemical products.	

Country	Tonnage	No. of Products[a]	Concen-tration[b]	Description	
	67	20 (0)	-	Use: intermediates (plastics manufacture). Trade code: Wholesale of chemical products; industry for plastic products; export.	
	42	10 (0)	-	Use: binders (paints, adhesives); adhesives; hardeners (for adhesives). Trade code: Industry for other non-metallic mineral products; industry for fabricated metal products (except machinery and equipment); industry for wood and products of wood, cork, cane, etc. except furniture; industry for electrical machinery and apparatus.	
	13	12 (4)	-	Use: insulating materials; jointing materials: Trade code: construction industry; export.	
	8	12 (8)	-	Use: caulking compounds; sealing compounds. Trade code: construction industry; wholesale and retail trade, repair shops for motor vehicles, motorcycles and personal and household goods; export.	
	2 to 8	2 (1)	-	Use: other. Trade code: paint stores; industry for wood and products of wood, cork, cane, etc. except furniture export.	
Sweden 2000 (SPIN)	195.0	60[e]		26.0 t	Manufacture of chemicals and chemical products
				84.0 t	Manufacture of rubber and plastic products
				7.0 t	Construction
				29.0 t	Wholesale trade and commission trade, except of motor vehicles and motorcycles
				6.0 t	Retail trade, except of motor vehicles and motorcycles; repair of personal and household goods
Sweden 1999 (SPIN)	185.0	60[e]		25.0 t (4 preparations)	Manufacture of chemicals and chemical products
				91.0 t (23 preparations)	Manufacture of rubber and plastic products
				8.0 t (18 preparations)	Construction
				29.0 t (7 preparations)	Wholesale trade and commission trade, except of motor vehicles and motorcycles
				4.0 t (4 preparations)	Retail trade, except of motor vehicles and motorcycles; repair of personal and household goods
Norway 2001 (SPIN)	50.5	21[e]		23.6 t (5 preparations)	Manufacture of chemicals and chemical products
				5.4 t (5 preparations)	Manufacture of rubber and plastic products
				14.4 t (11 preparations)	Construction
Norway 2000 (SPIN)	43.6	14		12.8 t (4 preparations)	Manufacture of chemicals and chemical products
				10.4 t (5 preparations)	Manufacture of rubber and plastic products
				15.9 t (8 preparations)	Construction

Country	Tonnage	No. of Products[a]	Concen-tration[b]	Description
Switzerland	-	25 (10)	1-10% (4) 10-50% (21)	Use in glue, surfacer, cement, sealing mass
	-	26 (0)	1-10% (2) 10- 50% (23) 50-100% (1)	Use in polymers
	-	4 (0)	1-10% (2)	Use in paints, dyes, varnish
	-	8 (0)	1-10% (1) 10- 50% (3) 50-100% (3)	Not defined
Total[d]	1312			

a: Total number of products (number of consumer product).

b: Danish and Swiss data – number in brackets is number of products at this concentration

c: Combined data with TDCP

d: Uses data for Sweden for the year 2000

e: Confirmed in SPIN database that some preparations are for consumer use, but number not presented

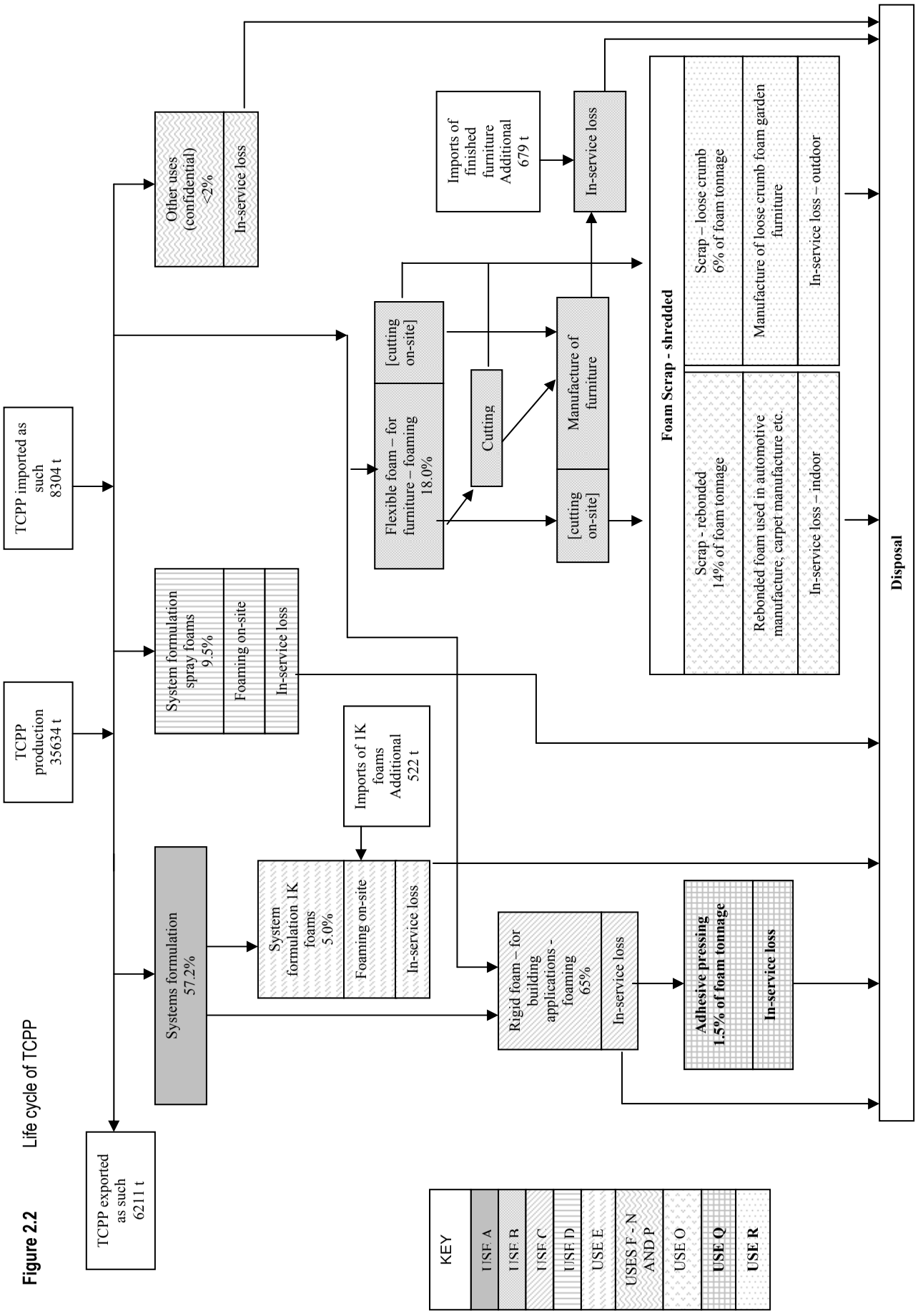
The product register data indicates that most products are available for professional use only, with limited use of products by consumers, for example, in one-component foams (see Section 2.2.2.5).

On the basis of the general description of uses reported in the product registers and the detailed descriptions of use pattern given by producers it is believed that the product register data do not provide new information concerning uses of 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, two of which were made with TCPP-containing foam. 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.

Figure 2.2 Life cycle of TCPP



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 interesting; the schematic indicates mechanical/feedstock recycling but elsewhere in the report the only route of 'recycling' investigated for releases is for heat recovery (i.e. incineration).. Mechanical/feedstock recycling is not believed by the Rapporteur to be a valid route and is not assessed in this RAR.

2.2.2 Scenarios

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

2.2.2.1 Formulation of systems: Use A

2.2.2.1.1 Overview

PUR is produced from the reaction of di-isocyanates with polyols. While some PUR producers buy polyols, di-isocyanates and other raw materials direct from manufacturers, others purchase pre-mixed, ready-to-use systems. PUR systems consist of (BASF, undated 1):

- Component A, the polyol component: a mixture of polyols, catalysts and other additives such as flame retardants
- Component B, the di-isocyanate component: containing the di-isocyanate or a di-isocyanate containing pre-polymer.

TCPP is added to polyols in the formulation of PUR systems. 16,600 tonnes of TCPP was used in the production of PUR systems in the year 2000. Additionally, 3850 tonnes of spray foam were formulated, also at systems houses; the two formulations are taken to be so similar that they are assessed together in the risk assessment. The total tonnage is therefore 20,450 tonnes. There are two types of systems house (pers. comm.[9]):

- raw material suppliers (i.e. polyol and di-isocyanate producers) who also formulate systems
- other smaller systems houses that purchase polyols and other raw materials for the formulation of systems.

Both types of companies formulate systems containing TCPP. An estimated 75% to 80% of PUR systems are manufactured and supplied by the major raw material manufacturers Elastogran, Bayer, Dow Chemical and Huntsman Polyurethanes (IAL 2000). The first two of these are also producers of TCPP and members of the Industry Consortium and are reported to have 40% to 50% of the polyol market for rigid applications (EC 2000b). The main European polyol producers have plants in the UK, France, Spain, Italy, Germany, Netherlands and Belgium (IAL 2000).

[9] In all cases of an unattributed pers. comm. it is not possible to reveal the source of the data. The information was provided by industry during the consultation process.

The suppliers of raw materials (i.e. polyols, di-isocyanates) are members of ISOPA, the European Diisocyanate and Polyol Producers' Association. ISOPA is the European trade association for the producers of di-isocyanates. It was formed in 1987 by seven chemical companies that have European interests in the production of raw materials for PUR and is an affiliate of European Chemical Industry Council (CEFIC) (ISOPA 2002a). ISOPA has provided information regarding systems for the development of this risk assessment.

Small to medium-sized system houses tend to manufacture small volumes of systems to supply local manufacturers and smaller PUR processors. They often supply niche markets where the major manufacturers are unwilling to manufacture in small enough volumes. Some system houses manufacture only a number of standard systems for various applications, whilst others also offer custom manufacture. There are at least 50 small to medium sized systems houses in the EU (IAL 2000).

System houses tend to purchase TCPP direct, but some of the smaller houses may purchase TCPP-containing polyols from the raw materials suppliers. Based on discussions with industry (pers. comm. 31st July 2002, producers and downstream users), it was estimated that less than 1% of the TCPP used by systems houses would be used as pre-formulated polyol.

2.2.2.1.2 The market for systems

TCPP-containing systems are used almost exclusively in the manufacture of rigid foams (pers. comm. 16th October 2001).

The end use of TCPP-containing systems is reported in **Table 2.4**. Producers have not specified an end-use for around 25% to 30% of TCPP in polyols. General information implies that most of these are used in the manufacture of rigid foams for use in construction (see section on rigid foam). There is some limited use in other applications such as rigid insulation. TCPP is not used in appliances such as refrigerators.

Table 2.4 End use of systems containing TCPP

End use	Percentage
General building applications	50% to 60%
Spray foams	15% to 20%
Unspecified	25% to 30%
Other (including furniture)	0% to 5%

2.2.2.1.3 Imports and exports of systems

There is a possibility that TCPP-containing polyols could be imported into the EU. EC (2000) reports that polyether polyols are imported into and from EU Member States in large quantities. They are easily and safely transported and transport cost is modest, deliveries over a distance of 2000 kilometres or more not being exceptional.

Based on knowledge of the industry, it has been suggested that on balance there is likely to be a net export of TCPP containing polyols from the EU, accounting for around 5% to 10% of EU consumption (pers. comm. 31st July 2002, producers and downstream users). To be conservative, no attempt has been made to account for these exports of TCPP from the EU in the assessment.

2.2.2.2 Flexible foam for furniture: Use B

2.2.2.2.1 Overview

6800 tonnes of TCPP was used in the production of flexible foam in Europe in the year 2000 (18% of total TCPP use). It is known that the vast majority of TCPP is added direct by foamers, although some systems are sold into this sector. Slabstock foam is almost exclusively produced with direct addition of TCPP; systems use in flexible foam is confined to flexible foam moulding (ISOPA 2003).

TCPP is used in slabstock (block) foam for upholstery and mattresses for the UK market. The use of TCPP is in direct response to flammability regulations covering these goods. TCPP has limited use in the rest of Europe. TCPP tends not to be used in the automotive industry owing to its potential for fogging – the condensation of volatile products on the inside of a car windscreen which occurs as a result of subjecting the TCPP-foam to high temperatures.

While settees, armchairs and other furnishings incorporate a wide range of foams as filling materials (Europur, 2002), the use of PUR foam in UK bedding is more limited than in the rest of Europe as these items are traditionally made of springs. In such sprung bedding foam is only used between the pieces of (often diamond stitched) fabric used as mattress covers (pers. comm., not attributable). Owing to the nature of the foam market, TCPP could well be present in UK-produced foams for other applications.

2.2.2.2.2 Flexible foam production

Flexible foams are produced by pouring the blend of the two raw materials (polyol containing additives including flame retardants such as TCPP, and di-isocyanate) onto a rolling conveyor belt (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[10]. Slabstock foam is cut in accordance with the specifications demanded by customers, the main application being for furniture (EC 1997).

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 2 Appendix A. The majority of the description of foam production presented in this section is taken from the risk assessment for pentabromodiphenyl ether (EC 2000a).

[10] Only slabstock foams are discussed here. Details of the moulding process can be found in the risk assessments for TDCP and V6 (HSA/EA, 2008a and b).

2.2.2.2.3 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, 2000a).

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.[11]). 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).

The major centre for foam cutting in the UK is Lancashire. There are 140 cutters in this county, of which only 40 to 60 are of any appreciable size (i.e. employing 3 or 4 people or more). Of these, six or seven employ over 150 people and a further two employ over 50 people. The remainder can be divided into two large groups, made up of companies employing around 20 people and companies employing four or five people. The remaining 80 to 100 companies are very small companies with only a few employees, which may sell to just one specialist sector of the market (e.g. stage scenery) (pers. comm., not attributable).

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)
- 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.

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

2.2.2.2.4 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. In this regard end product manufacturers may carry out cutting of polyurethane foam (EC 2000a). In contrast, some cushions arrive at the furniture manufacturer pre-covered with polyester fibre (pers. comm., not attributable).

There are an estimated 8,500 furniture manufacturing businesses in the UK (DTI 2002).

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 rolls. The flame treatment generates a chemically active surface which 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).

2.2.2.2.5 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).

Some UK foamers manufacture re-bonded foam at the same site as foaming takes place, in separate buildings (pers. comm., not attributable); indeed rebonding sites have traditionally been set up to remove trim foam from specific foaming sites (pers. comm. 29th April 2004). Alternatively foam is shipped outside the UK for re-bonding.

In some cases TCPP is added in the rebonding process to reduce the viscosity of the pre-polymer and to provide flame retarding properties (pers. comm., not attributable). This has not been accounted for in the risk assessment since it is considered to be insignificant; releases from any such use of TCPP will be accounted for within the general use in foam, together with rebonding.

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). 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 re-bonded 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).

A large proportion of the scrap foam generated in the UK (as much as 80%) will contain TCPP. Some scrap foam generated in the EU will also contain TCPP.

In the risk assessment of pentabromodiphenyl ether (EC 2000a), 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 (EFRA 2003). 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[12].

While all such furniture previously was to be returned to the UK to meet the demand generated by UK regulations, now 50% 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 TCPP 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.

[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.

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 3 of Appendix A.

2.2.2.2.6 Imports and exports of foams

As indicated in Appendix A, the movement of foam across large distances is limited by costs considerations. That said, consultation indicates the following UK imports and exports of foam and finished goods (pers. comm., not attributable):

- some foam manufactured in the UK is exported then imported as finished articles;
- there is import and export of finished articles to and from the UK; and
- there is export of scrap foam from the UK to both the US and mainland Europe, some goods incorporating re-bonded foam are imported.

Data on the UK furniture market are given in **Table 2.5**. In terms of value, imports of furniture into the UK from mainland Europe represent around 15% of the UK market.

Table 2.5 Imports of furniture to the UK

	£ million 2000	£ million 2001
EU total	**176.1**	**176**
Italy	134.5	134
Belgium	33.8	33.3
Norway	7.8	8.7
Non-EU total	**42.8**	**79.5**
Poland	9.7	15.6
China	4.3	12.3
Thailand	7.5	7.8
Others*	21.3	43.8
Total imports	**218.9**	**255.5**
Total UK market	**1360**	**1440**
UK manufacture	**1141.1**	**1184.5**
EU imports as % of UK production	**15.4%**	**14.9%**
Non-EU imports as % of UK production	**3.8%**	**6.7%**
* origin unknown, assumed to be non-EU		
Source: BRMA 2002		

In terms of value, between 4% and 7% of the UK upholstered furniture market is from outside the EU. If it is assumed that imports are of furniture at the lower end of the price range, these imports could represent more than 4% to 7% of the total TCPP tonnage used. To be conservative it is assumed that imports of furniture to the UK from outside the EU could

account for an additional 10% of TCPP usage (i.e. up to approximately 680 tonnes per annum).

Data regarding exports of furniture containing TCPP are not available. With respect to the EU as a whole, there was a net export of upholstered furniture from the EU in 1997, valued at 322 million Euros (UEA 2002). On this basis it is considered that across the EU as a whole there is likely to be a net export from the EU of TCPP in furniture products.

The risk assessment is already conservative in both total tonnage consumed in EU (see section 2.1.2.1) and local and regional scenario for this use specifically. Therefore, this additional relatively small tonnage is treated as an additional source of release in the continental background.

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).

2.2.2.3 Rigid PUR foams for use in construction: Use C

2.2.2.3.1 Overview

26,650 tonnes of TCPP were used by rigid foamers in the production of construction products in the year 2000 (66.5% of total consumption), with a further 14.3 % used in the production of spray and one component foams (considered separately in Section 2.2.2.4 and 2.2.2.5).

70% of this TCPP was added via systems and the rest direct by rigid foamers. These figures agree with general data for the rigid foam industry as a whole. For example, the German government indicates that if the market for PUR rigid foam is viewed as a whole, then more than 70% of the base products are delivered by 'system suppliers' (Leisewitz A, Hermann K and Schramm E 2001). In general, it is the larger foamers who purchase TCPP direct from producers and the smaller foamers who purchase TCPP-containing systems (pers. comm. 28th February 2002, PEFRC).

Consultation with the producers of TCPP indicates that these foams will all be used in the construction industry in the production of PUR rigid panels and laminates for insulation purposes (pers. comm., not attributable). There are many other applications of rigid polyurethane, but industry has indicated that the only rigid foam application for TCPP is in construction panels. Other applications use either no FR or other types of FR.

On this basis, and in the absence of additional information, these additional tonnages are assumed to be associated with general building applications. Thus, the total tonnage of TCPP used in general building applications is taken to be 26,650 tonnes.

2.2.2.3.2 Key products

Rigid foams are mainly produced as blocks and panels and used for insulation purposes (EC 1997). 90% of all external roof and wall panels used on modern commercial and industrial buildings use rigid PUR (EPIC 2002). For PUR insulation foams in general, 90% of the usage of additive flame retardants is currently accounted for by TCPP (Leisewitz *et al*. 2001).

Some of the key products associated with PUR insulating foam are the following:

- Flexible-faced laminate
- Sandwich panels
- Discontinuous panels
- Block foams
- Injected foams

For further information on these products, and their production and use, refer to section 4 of Appendix A.

2.2.2.3.3 Legislation relating to fire safety

Furniture

United Kingdom

Statutory standards exist in the UK for the flame retardancy of furniture and similar goods. This legislation is 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 regulations affect the following consumer products (DTI undated[13]):

- all indoor and outdoor upholstered furniture, foam and loose fillings, permanent and other covering fabrics;
- mattress foam fillings; and
- all second hand upholstered furniture for retail sale

These are expected to meet the fire resistant ignitability tests according to various British Standards including BS 5852 part 1 (1979), BS 5852 part 2 (1982) or BS 7177 which in turn makes reference to BS 6807 (which requires cigarette and match ignition resistance). The regulations do not stipulate the means by which the fire resistance tests are to be met; they are therefore performance centred and manufacturers can elect to meet them in whatever ways are appropriate. In the main the requirements appear to be met by the use of chemical flame retardant systems included in combustion modified foam and in backcoating for covering fabrics (DTI undated). In the main TCPP is used along with melamine (Pers. comm., 16th October 2001).

[13] All details of the 1988 and 1989 regulations are taken from this publication.

It is reported that in the UK, the introduction of the 1988 regulations have resulted in a move away from 'standard' foam to 'combustion modified' foam. As a result, more products are made with combustion modified foam, even though the flame retardancy properties are not required. Thus TCPP could be found in a wide range of products, including, for example, the padding in padded greeting cards. There will be some packaging that may not contain TCPP, nor will the foam used in the top of pill boxes and in surgical swabs, however these are reported to be the exception (pers. comm., not attributable). It has separately been reported that 80% of the foam produced in the UK contains TCPP and 20% does not (pers. comm).

Ireland

The equivalent legislation in Ireland is the Industrial Research and Standards (Fire Safety) (Domestic Furniture) Order 1995 (S.I. 316 of 1995). This Order makes it unlawful to manufacture or assemble furniture (including refurbishment of old furniture) using filling material that does not meet Irish Standards I.S. 419:1988, which relates to ignition resistance in standard tests, or to sell such filling material.

European Union

There is currently no European Directive concerning the flame retardancy of furniture and similar goods. As a result, TCPP and other flame retardants tend not to be used in furniture and there is a much lower use of TCPP in Europe than in the UK (Pers. comm., 16th October 2001). For example, flame-retarded upholstered furniture and mattresses are produced in Germany, but only for a limited part of the institutional/commercial buildings/facilities sector (e.g. for ships, hospitals, hotels). Quantitatively, this represents a maximum of 1% of the mattresses and 2% of the upholstered furniture produced, of which 1% is flame-retarded for institutional/commercial buildings/facilities and 1% for export (UK, USA, etc.).

Industry reports that there are no firm proposals for a European Furniture Directive and that such a directive is unlikely to be introduced in the foreseeable future. If a Directive were to be introduced it would represent a major change, but it would take some time for changes to be implemented; say a minimum of five years. The impacts of such a Directive are difficult to predict as these would depend on the stringency of the requirements. For example, if there were to be a new fabric requirement only, this would not affect the foam industry at all (pers. comm. 31st July 2002, producers and downstream users).

US requirements

California 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 hotel chains). TCPP cannot meet the heat-ageing requirements of this standard owing to its volatility. TCPP is thus not used in products meeting the requirement of California 117. These observations support the view that losses must be related to volatility.

Construction products

The European Union Directive for construction products (89/106/EEC) was adopted with the objective of creating a single market for construction products in the European Economic Area (EEA). To place a construction product on the market in any Member State, the product should carry a CE mark, which guarantees conformity with a range of technical specifications (Koschade 2002):

- mechanical resistance and stability
- safety in case of fire
- hygiene, health and the environment
- safety in use
- protection against noise
- energy economy and heat retention

On the basis of the essential requirements of the Directive the EC issues mandates to CEN (Comité Européen de Normalisation – European standards Organisation) and EOTA (European Organisation for Technical Approvals). Whilst the Directive relates to test methods, product performance and conformity assessment, it does not harmonise regulations: the Member States are free to set their own requirements for the performance of products (Koschade 2002). For example, in Germany, all foam materials for construction must have a minimum performance of B2 according to DIN 4102 part 1. Other countries, such as France, Spain, UK or Benelux, do not require such minimum performance levels, as long as the building element meets the fire requirement specified for the building regulation (ISOPA 1999).

The mandate for creating the harmonised standard for reaction to fire was given in CEN/TC 127: Fire Safety in Buildings in co-operation with ISO/TC 192. The task of harmonising national fire safety standards is very complex; for a number of practical reasons, these standards have developed along very different lines in the various countries. Risk assessment, evaluation of the level of safety and testing methods of the European nations sometimes deviate widely (Koschade 2002).

In 1994, a Commission decision was made to implement a European classification system for reaction to fire with a supporting set of text methods, the so-called Euroclassification. One of the test methods was to be newly developed, namely the single item burning test (SBI, prEN 13823). In addition there is the small flame test (prEN ISO 11925-2), the non-combustibility test (prEN ISO 1182) and the determination of the calorific value (prEN ISO 1716). The Euroclassification system will be in place in the course of 2002 (prEN 13501-1). This new system will allow the reaction-to-fire performance of products to be labelled according to Euroclasses A to F. Combustible building materials, which include all organic building materials including rigid PUR foam, are all presumably assigned to classes B, C, D and E (Koschade 2002).

Besides the reaction-to-fire behaviour of building materials, CEN/TC 127 and CEN/TC 128 also refer to the fire resistance of buildings. To determine fire resistance, the materials are set alight so that a full fire in a space is simulated. The measured fire resistance time is classified within a time span of 15 to 360 minutes into ten classes (Koschade 2002). The relevant standard for fire resistance is EN 13501-1 - Fire classification of construction products and building elements - Part 1: Classification using test data from reaction to fire tests (CEN online, undated).

2.2.2.3.4 Imports and exports of PUR for construction

Industry reports that excluding Switzerland and near Eastern European countries, there is only limited trade in rigid foam products as it is too expensive to transport products over long

distances. There is also no need for such transport as there are many regional producers. The trade association for the rigid foam industry BING (the Federation of European Rigid Polyurethane Foam Association) does not have data on this trade, but indicated that it is not significant (pers. comm. 31st July 2002, producers and downstream users). ISOPA (2002b) indicate that export of rigid foam from the EU is low (<5%). To be conservative and in the absence of firm information, the tonnages of rigid foam in service and for disposal in the EU assume no exports.

2.2.2.3.5 Recycling and end of life

Production waste

Waste from the production of rigid foam is used for adhesive pressing in the production of moulded boards for use in kitchen furniture and flooring (ISOPA 2001b). For example, there is a company in the Netherlands making fixed board, comparable with chipboard. The total capacity for adhesive pressing is 10,000 tonnes per annum rigid foam (pers. comm. 31st July 2002, producers and downstream users).

Particles can also be used as oil binders or in combination with cement as insulating mortar (ISOPA 2001c). With respect to the first of these, rigid foam scrap is used by fire brigades for oil spill clean up. After use, this is incinerated (pers. comm. 31st July 2002, producers and downstream users).

50% of scrap is used in adhesive pressing and 50% as oil binders (pers. comm. 31st July 2002, producers and downstream users). These along with other options for recycling PUR are further described in section 3 of Appendix A.

The amount of production scrap foam generated is less than for flexible foam as some panels are produced discontinuously and not as slabstock. Waste is reported to be of the order of 2% to 3%.

Therefore 1.5% of rigid foam tonnage is included in the risk assessment for processing, in-service loss and in disposal associated with adhesive pressing.

Adhesive pressing Use Q

PUR is granulated and blended with 5% to 10% polymeric methylenediphenyl di-isocyanate (MDI) and formed into boards/mouldings at temperatures up to 200ºC and under pressure (20 to 200 bar). Products are finished by sawing and sanding or by applying additional facings. Based on the information given above, 1.5% of the rigid foam tonnage is recycled by adhesive pressing. This is a tonnage of 400 tonnes per year.

Cutting

On construction sites, small modifications are sometimes made to the physical form of panels, e.g. panels are cut to the required size. In the Netherlands, the resultant saw dust is compressed to brickettes, which are incinerated or used as raw materials for other products (pers. comm., not attributable). The extent to which this practice occurs in other Member States is not known and therefore it is not possible to calculate the wastage level. Since this release will be diffuse and quantities will be very low, this route of release is not considered

in the risk assessment. In any case, the loss to soil of 2% from weathering and wear in service or at disposal will account for this.

End of life

The recommendation is for incineration with energy recovery. Thus there is a trend away from landfill towards energy recovery (pers. comm. 31st July 2002, producers and downstream users). In the risk assessment, a proportion of 50% is taken to be landfilled; the remaining 50% is taken to be incinerated with energy recovery. When insulation foams are removed from buildings at the end of life the usual practice is to bury these foams in landfill. The implementation of the Landfill Directive may affect the fate of TCPP-containing items at the end of their service life.

2.2.2.3.6 Trends in the industry

The move towards polyisocyanurate (PIR) foams

There is a trend in the industry for a move from PUR foams to so-called PUR-modified PIR foams, or isocyanurate-modified PUR foam in some applications. PIR foams have different requirements in terms of flame retardant types and quantities than PUR foams (Leisewitz, Hermann and Schramm 2001), generally requiring lower levels of flame retardant to be added than for PUR (ISOPA, 2003).

The rigid foam industry indicates that PIR foams are very important as PIR manufacturers may be buying polyols without realising they contain TCPP (ISOPA 2003). In this risk assessment since there is a full tonnage balance, hence any TCPP that might in reality be used in PIR is simply being risk assessed in a different substrate (PUR). The ESD approach would be the same for both so this is not a major source of concern.

Construction Products Directive

It is also reported that the Construction Products Directive may bring about changes in the classification of PUR insulation foams and have an effect on their flame-retardant composition (Leisewitz, Hermann and Schramm, 2001).

Industry reports that flammability of construction products is an area that is well controlled at present through national standards. Thus changes brought about by the Construction Products Directive will not introduce new requirements but change existing ones. Effects will thus be subtle. That said, while changes in flammability regulations may increase use of flame retardants such as TCPP, the new regulations will not come into play until 2007 or 2008. Such changes may also result in increased use of PIR however (pers. comm. 31st July 2002, producers and downstream users), which may lead to a subsequent reduction in use of TCPP.

Replacement of TCEP

Finally, TCPP is a drop-in replacement for TCEP. There is a move away from use of TCEP by industry. In Western Europe, by far the largest field of application of TCEP (80-90% of the quantity produced) is that concerned with reducing the brittleness and with the simultaneous flame-resistant finishing of polyurethane in the production of celled, rigid or semi-rigid foam (GDCh, 1987, from BAUA 2006). One of the main industrial branches to use TCEP is (roof) insulation for the building industry.

Discussions with the flame retardant industry have indicated that where TCEP can be replaced with TCPP, then this will already have taken place, i.e. a further increase in the use of TCPP is unlikely (pers. comm. 18th April 2002, PEFRC). Indeed the rigid foam industry has indicated that TCEP is not used in rigid foams, TCPP being the main flame retardant (pers. comm. 31st July 2002, producers and downstream users).

Kyoto protocol

As a result of the Kyoto protocol, the use of foam insulation in buildings is increasing, as insulation is an effective way of reducing CO_2 emissions (ISOPA 2003, pers. comm. 31st July 2002, producers and downstream users This use is reinforced by more stringent insulation requirements in several member states and by the new EU Directive on the energy performance of buildings published in January 2003 (ISOPA 2003).

2.2.2.4 Spray foams: Use D

2.2.2.4.1 Overview

Spray foams are surface-adapted technical insulation materials for roofs, interior spaces and technical applications (sometimes known as moulding foams). These are used in building construction and maintenance and repair. They are not available for use by the general public.

Companies using spray foams are in general small companies (up to ten employees), who purchase formulated systems ready for use (pers. comm., not attributable).

Spray foams are formulated by systems houses and are usually applied *in situ* to walls, roofs, tanks and pipes. Most applications are external but some are inside buildings. Spray foams are very versatile and can be applied over uneven surfaces and used, for example, to repair and insulate damaged roofs (Jeffs 2000).

It is assumed that 3,850 tonnes of TCPP was consumed in spray foams in the year 2000[14], all of which was added by systems houses.

For further information about application of spray foams, refer to section 5 of Appendix A.

2.2.2.5 One-component foams: Use E

2.2.2.5.1 Overview

One-component foams (also known as 1K foams or OCF) are dispensed from aerosol cans containing polyols, MDI and propellants. These are used as fillers for joints and cavities around, for example, doors and window frames.

[14] Some data provided by TCPP producers related to production of one component, two component and spray foams. General discussions with industry indicate that while spray foams and one component foams are clearly identifiable products with specific applications, the term "two component foams" is generic and is believed to apply to spray foams.

Data provided by the producers of one-component foams indicates 1,900 tonnes of TCPP used in the production of one-component foams in the EU (Rhee 2002). Further details are presented in the Confidential Annex. While most manufacturers of one-component foams use TCPP direct, some purchase and use pre-formulated polyols.

2.2.2.5.2 Production of one-component foams

Large producers of one-component foams receive TCPP in bulk and store it in large tanks. All of the large producers have special chemical unloading docks with provisions to collect spillage. Smaller producers use TCPP in one-tonne containers or drums (Rhee 2002).

TCPP is without exception used in one-component PUR foam at room temperature (i.e. 20ºC to 25ºC). TCPP is pumped from the closed storage-tanks into a closed weighing tank where the product is mixed with polyols. From the weighing tank there is a direct connection with the filling heads of the aerosol machines. In general, ten seconds after filling the aerosol can with the polyol component containing TCPP, the can is closed air-tight by the valve (Rhee 2002).

The polyol and the di-isocyanate are brought together in such a way that the pre-polymerised polyurethane remains liquid and has some capacity to react with humidity. A propellant (pentane/butane) is added so that the pre-polymer is able to emerge through a small plastic pipe (pers. comm., not attributable). In the storage, filling and mixing areas there are no water-supply points and no sewer outlets. Water is the "biggest enemy" in the process as it can cause cans to explode (Rhee 2002).

2.2.2.5.3 Use

Some products are used by construction workers at building sites, while others are available to the general public for the DIY filling of cavities (Pers. comm., 16th October 2001). A producer of one-component foams indicates that the aerosol cans it produces are used almost exclusively on the inner shell or inside joints of buildings. In 60-80% of applications, the foam is covered with plaster (pers. comm., not attributable). Furthermore, it has been indicated that the remaining 20-40% is also covered by, for example, wooden doorframes. These foams are not UV resistant and so they must be covered (ISOPA and the rigid polyurethane foam industry, 2006).

During application the foam is extruded from the can. After one hour the foam is fully cured. During curing the temperature remains ambient. After curing the TCPP is embedded in the polycondensate structure of the PUR and has no tendency to migrate (Rhee 2002).

2.2.2.5.4 Recycling of aerosol cans

In Germany, there is a collection system for the recycling of used aerosol cans. Cans are collected at specified locations and sent to PU-Dosen-Recycling GmbH (PDR) in Thurnau which is a dedicated PUR can recycling factory. The cans are split into the following main streams (pers. comm., not attributable):

- paper
- polyethylene (caps)

- aluminium
- tinplated steel
- propellant gas
- polyurethane pre-polymer.

TCPP is recovered completely in the polyurethane pre-polymer, which is completely re-used in polyurethane aerosol cans (pers. comm., not attributable).

The extent to which recycling takes place in other Member States, such as the UK, is not known.

2.2.2.5.5 End of life

In Germany, at the end of the lifetime of a building, one-component foam is collected in building-waste in the 'light fraction'. It is reported that the collection rate for such foam is almost 100%. Foam adhering to windows, concrete or brick, is separated from these materials in the crushing operation and is separated from the heavy fraction by cyclones or wind-sifting. The light fraction is incinerated (pers. comm., not attributable). The situation in other Member States is not known.

2.2.2.5.6 Imports and exports of one-component foams

Data provided by the producers of one-component foams indicates that a further 1,915 tonnes of TCPP are used in the production of one-component foams in the rest of geographic Europe (Rhee 2002). Further details are given in the Confidential Annex.

It is believed that there is a net import of one-component foams containing TCPP into the EU. In total, 2,400 tonnes of TCPP are believed to be associated with one-component foams consumed in the EU. Thus, imports are believed to account for around 500 tonnes of TCPP, equivalent to around 25% of EU production (Rhee 2002).

2.3 OTHER USES

The following use codes are covered in the confidential sections of the report: F, G, H, I, J, K, L, M, N, and P.

2.4 TRENDS

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

- increasing use of sandwich panels (Koschade, 2002)
- a trend away from exporting scrap furniture foam to the US
- a trend towards increased recycling and recovery of PUR foams in general
- a trend away from disposal of waste to landfill.

2.5 LEGISLATIVE CONTROLS

There appear to be no EU emissions or exposure controls related to the substance itself.

The use of the flame retardant TCPP in furniture applications is driven by fire safety standards.

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). These regulations are important in driving the market for flame retardants, and TCPP in particular.

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

While the Construction Products Directive makes some provision for fire safety of buildings, it does not harmonise regulation. Requirements vary across Europe. A new CEN (European) standard is currently being developed.

For the parts of the life cycle associated with polyurethane foaming, emissions of TCPP 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. TCPP is not specifically covered by this legislation beyond this general aspect.

3 ENVIRONMENT

3.1 ENVIRONMENTAL EXPOSURE

In the assessment of some life cycle stages, it has been necessary to use appropriate defaults to characterise a reasonable worst-case emission pattern. Site-specific data have been used where available, to refine the exposure assessment. Since the market cannot be considered static (e.g. the market supply of TCPP may be affected by the regulation of other flame retardants), it is appropriate to apply a model in which defaults are not overruled without evidence that is widely applicable. This is particularly important for the most significant applications.

Consultation with key downstream users was used to supplement the information provided by producing companies. All producing companies co-operated with the assessors and provided detailed information about the life cycle for TCPP. Two companies 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.

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 TCPP has not been evaluated in this risk assessment for industrial sites, 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 3.1.2.3.4.

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 flame retardants (FRs) from foams.

EUROPUR has sponsored a study to investigate volatile losses of 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 experimental data is that initial rapid losses occur 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 available data. This finding is described in more detail in Appendix B.

The B-tables and ESD methods are not used in most cases to derive site sizes; 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.

3.1.1 Properties of TCPP in the context of the ESD (OECD, 2004)

The main desired activity of TCPP is as a flame retardant, though it also has plasticising properties. As TCPP 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 falls within the bracket identified as 'high' within the ESD (OECD, 2004).

The ESD envisages flame retardants as being either organic solids or inorganic solids. As stated above, TCPP is a liquid, with a 'high' vapour pressure (in this context). For this reason it would be inappropriate to simply apply the organic flame retardants defaults from the ESD, as the loss scenarios will be different:

- the potential for dust formation is removed
- there may be volatilisation
- 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 less relevant. Having said that, it is noteworthy that ESD losses from the stage of conversion (e.g. foaming) are (for additive types where it is recognised that a range of substance types are used) dependent on the volatility of the additive.

Variation of loss rate based on volatility in the ESD/UCD

In the stages of compounding (e.g. formulation of systems) and conversion (e.g. 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 well established. This relationship is applied in some cases here (e.g. for some in-service loss stages) in the derivation of 'correction factors' to derive default rates of loss for TCPP (high 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.

3.1.2 Environmental releases

3.1.2.1 Release from production

3.1.2.1.1 Defaults

It is not considered necessary to seek default rates of loss. All manufacturing sites within the EU have been identified and site-specific release data have been provided by the industry.

3.1.2.1.2 Extent of site-specific data

Site-specific data provided by the producers of TCPP are set out in the Confidential Annex.

3.1.2.2 Release from formulation: Use A

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.

3.1.2.2.1 Overview

This life cycle stage has been divided as follows (with further information given in the Confidential Annex):

- large systems houses
- medium sized systems houses
- smaller systems houses
- systems houses using pre-formulated polyol, i.e. purchasing TCPP-containing polyols from others (1% of tonnage for large, medium and small systems houses).

The large, medium and small sized system houses each account for 30% to 35% of the TCPP consumed. System houses producing one-component foams are considered separately (Use E).

Rates of release from formulation (compounding) of systems for rigid foams and spray foams are evaluated together on the recommendation of the rigid foam industry (pers. comm. 31st July 2002, producers and downstream users). These processes are also effectively the same in terms of the default process.

3.1.2.2.2 Large systems houses: Use A1

Large systems houses are assessed on a partly site-specific basis. Information is provided in the Confidential Annex.

3.1.2.2.3 Medium systems houses: Use A2

Number and nature of sites

The B-tables (Table B2.3) give the following for IC11, formulation, loading rate 15%, with 10% in the main region.

Fraction of the main local source = 0.8
300 days' operation per year

The main systems houses have sites in six regions (IAL 2000). The following set of values is used in preference to the B-table defaults based on information set out in the Confidential Annex:

Fraction in the main region = 0.133 (gives sites in a minimum of 8 regions)
Fraction of the main local source = 1 (one site in the main region)
Number of days per year = 300

The main site handles just under 1000 tpa of TCPP and produces 22 tonnes of formulation per day (with an assumed loading rate of 15%).

Releases

The ESD defines separate rates of loss from handling of raw materials at compounding sites and from the compounding process itself. On the basis of a site visit and ISOPA data (ISOPA 2002b), releases from handling are set to 0%.

From the ESD:
Total losses from compounding = 0.025% to air
= 0.025% to wastewater

There is some evidence available, from an industry monitoring study undertaken at a European systems house (Tauw, 2007), to suggest that releases to air might be very much lower than those modelled, when best practice is followed[15].

[15] Sampling was undertaken at relevant emission points. In each case a suction probe was placed at the emission point and flue gas was led over an XAD-2 adsorption tube. TCPP was extracted using dichloromethane with ultrasonication; analysis was performed using GC-MS. Validation checks gave satisfactory results. The study has certain limitations. Monitoring was performed on one occasion only. The focus was on certain specific unit operations, associated with handling processes (believed to be the only potential sources of emission at the site).

3.1.2.2.4 Small systems houses: Use A3

Number and nature of sites

The B-tables (Table B2.3) give the following for IC11, formulation, loading rate 15%, with 10% in the main region.

Fraction of the main local source = 0.8
300 days' operation per year

There are at least 50 small to medium-sized systems houses across the EU (IAL 2000). Based on this and data set out in the Confidential Annex, the following set of values are used in preference to the B-table defaults:

Fraction in the main region = 0.1 (sites spread across the EU)
Fraction of the main local source = 0.45 (gives at least 3 sites in the main region)
Number of days per year = 300

The main site handles 250 tpa of TCPP and produces 5.5 tonnes of formulation per day (with an assumed content of TCPP in the formulation of 15%).

Releases

The ESD defines separate rates of loss from handling of raw materials at compounding sites and from the compounding process itself. On the basis of a site visit and ISOPA data (ISOPA 2002b), releases from handling are set to 0%.

From the ESD:
Total losses from compounding = 0.025% to air
= 0.025% to wastewater

There is some evidence available, from an industry monitoring study undertaken at a European systems house (Tauw, 2007), to suggest that releases to air might be very much lower than those modelled, when best practice is followed[15].

3.1.2.2.5 Systems houses using pre-formulated polyol: Use A4

In the absence of firm information, a loading rate of 10% is assumed.

Number and nature of sites

Industry indicates these systems houses account for less than 1% of the TCPP tonnage. No data are available on the number and distribution of these sites. It is thus assumed that these are spread across the EU as follows:

Emissions are given in terms of unit operations (<0.002 g TCPP per drum filling operation; <0.0002 g TCPP per pumping operation for transfer into production). The ESD default fraction released to air associated with blending processes was 2.5E-04. Release from the monitoring work for a component of this process (loading of blended product into drums) is equivalent to ~2E-08 and this could suggest that, assuming similar levels of control are in place for other aspects of the blending process, overall release fraction would be unlikely to be above 2E-06. These figures are subject to too much uncertainty to use in place of ESD defaults, but are indicative that the ESD could be significantly overestimating release potential at sites where best practice is applied. Further details are provided in the Confidential Annex.

Fraction in the main region = 0.1 (sites spread across the EU)
Fraction of the main local source = 1 (one site in the main region)
Number of days per year = 205
The main site handles just over 20 tpa of TCPP and produces 1 tonne of formulation per day (with an assumed loading rate of 10%).

Releases

Following the pattern of other systems houses, releases from handling are set to 0%. Thus,

Total losses from compounding = 0.025% to air
= 0.025% to wastewater

There is some evidence available, from an industry monitoring study undertaken at a European systems house (Tauw, 2007), to suggest that releases to air might be very much lower than those modelled, when best practice is followed[15].

3.1.2.3 Release from flexible foams: Use B

3.1.2.3.1 Foam production

Loading rates

The report on flame retardants by the German government (Leisewitz A, Hermann K and Schramm E, 2001) gives TCPP loading rates for flexible foams of between 3% to 5% of weight. Data provided by the producers of flexible foams in response to the questionnaire widens this range to between 2.5% and 14%, with two of the producers indicating a loading rate of around 7% to 8% TCPP on average.

Foamers indicate that variation is as a result of variations in the density of the foam, with different parts of the furniture requiring differing densities of foam. Seats need to be the hardest wearing and thus are of the highest densities. Seat backs are not subject to the same stresses and can thus be of lower densities. As low density foams are more difficult to flame retard, these are associated with a higher loading rate. Higher loadings of TCPP may also be used to maintain foaming properties and avoid the use of solids such as melamine.

Based on the information available, a loading rate of 8% in the foam is considered realistic and is used in the assessment.

Number of sites

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. Not all of these will be using flame retardants, and not all that use flame retardants will be using TCPP. EUROPUR have estimated that 390,000 tonnes of flexible slabstock PUR foams are produced in the EU each year, 60,000 tonnes of which are produced in the UK (RPA 2000). The low price of TCPP and the mature market for this product means that TCPP tends to be used by the larger sites producing flexible foam (pers. comm. 8th February 2002, Rhodia).

Data have been provided by the producers of TCPP and by companies using TCPP in the production of furniture. There are five manufacturers of flexible foam in the UK: Caligen Foam, Kay Metzler, Vita Foam (all British Vita companies), Recticel and Carpenter (pers. comm., not attributable). All five of these companies have provided information on TCPP consumption in the year 2000, and accounted for the consumption of 4,800 tonnes of TCPP, 71% of the TCPP used in this application[16]. Thus, most of the TCPP used in flexible foam production is consumed by very large UK-based sites.

The B-tables (Table B3.9) give the following, for IC11, processing, loading rate 8%, with 10% in the main region (Fraction in the main region = 0.1)
Fraction of main local source = 0.1
300 days' operation per year

This default is equivalent to a minimum of 100 foamers, maximum size 67 t of TCPP. It bears no relationship to practice.

Based on what is known, the following set of values would be preferable to the B-table defaults:

'Very-large' foamers (Based in the UK, accounting for around 70% of the TCPP used in flexible foam, with the largest site handling 1,920 tonnes per annum TCPP and an estimated 24,000 tonnes foam based on a loading rate of 8%).

Fraction in the main region = 1
Fraction of the main local source = 0.4
Number of days per year = 300

The remaining 30% of the flexible foam tonnage is split between 'large' and 'small' sites; some of the latter use systems rather than TCPP directly. The basis of the split is described in the Confidential Annex.

'Large' foamers using TCPP direct (spread around the rest of Europe, with the largest site handling around 350 tonnes per annum TCPP and an estimated 4300 tonnes foam based on a loading rate of 8%)

Fraction in the main region = 0.2
Fraction of the main local source = 1
Number of days per year = 300

'Small' foamers using TCPP direct (spread around the rest of Europe, with the largest site handling around 40 tonnes per annum TCPP and an estimated 500 tonnes foam based on a loading rate of 8%)

Fraction in the main region = 1
Fraction of the main local source = 1
Number of days per year = 300

[16] It has been confirmed with the British Rubber Manufacturers Association (BRMA), the UK trade association for flexible foams, that it is not breaching commercial confidentiality to reveal this tonnage (pers. comm. 17th July 2002).

Small foamers using TCPP in systems, in the absence of specific information, are assumed to be spread across Europe (with the largest site handling 75 tonnes per annum TCPP and an estimated 940 tonnes foam based on a loading rate of 8%):

Fraction in the main region = 0.32
Fraction of the main local source = 1
Number of days per year = 300

Major sources of release

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:

- the handling of the flame retardant prior to mixing with other ingredients (TCPP is a liquid)
- volatilisation from the foam while at elevated temperatures (curing)
- 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.

Mixing of the components required for the foam is usually carried out by a mixing head immediately prior to feeding into the moulding system. The flame retardant additives 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. methylene chloride) 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 are set out below.

Defaults

Although not used as the numerical basis of the risk assessment, it is of interest to explore the use of ESD defaults. Information on the release of flame retardants during the processing of plastics and foams is also given in the Emission Scenario Document (OECD, 2004). One source of release for liquid (flame retardant) additives is associated with the handling of the raw material (e.g. splashes, spills, etc.) prior to the foaming process. The ESD estimates releases to wastewater to be of the order of 0.01% (i.e. 0.1 kg/tonne).

Handling losses at foam producers: 0.01% to wastewater.

This route of release does not apply for the foamers using TCPP in systems, only for those adding TCPP direct.

The ESD sets out rates of release of various additive types from various types of process. To select the correct value some subtlety is necessary, as prescriptive application of the ESD default losses for flame retardants from foaming is not appropriate in the present case.

- Flame retardants are considered by the ESD to be solids and therefore of 'low' vapour pressure. Therefore it is necessary to multiply by a factor of 25 to derive equivalent rates of loss for a 'high' vapour pressure (in the context of the ESD) substance such as TCPP. The use of this correction factor is in accordance with relative rates of loss from 'low' and 'high' volatility additives given in the ESD for all types of polymer processing. The corrected rate of loss is equal to the rate given by the ESD for open processes and foamed articles for various 'high'-volatility additive types (e.g. antioxidants).
- Another correction factor can be seen in the relative rates of loss from open and closed processes given in the table. Like for like, the rate of loss from foaming (always considered an open process in the ESD) is ten times higher than from closed processes. However, consultation and site visits indicate that foaming of polyurethane in particular is always closed, in order to prevent workers being exposed to di-isocyanate.

Therefore the appropriate default to use is the rate of loss of a 'high'-volatility additive (in the context of the ESD) from a closed process. This is a rate of 0.05% (i.e. 0.5 kg/tonne) lost in equal proportions to air and wastewater.

Reasonable worst case emissions

In the case of polyurethane, the evidence is that due to the high levels of vapour controls in the workplace, it is not appropriate to differentiate between different site sizes, since controls must be equally stringent at all sites. Therefore the factor of ten is not applied for polyurethane related processes in this risk assessment.

Discussions with foam producers and their UK and European representatives – the British Rubber Manufacturers Association (BRMA), which represents UK foamers, and EUROPUR, the European Flexible Polyurethane Foam Blocks Manufacturers Association - indicate that in practice emissions from foamers will be very much lower than the default emissions. This was confirmed through a visit to a very large foamer in the UK. On the basis of that visit the following emission rates have been developed for very large foamers and are used in the assessment. The applicability of these values to all large sites in the UK has been confirmed by BRMA.

Emissions from handling TCPP are considered to be effectively zero owing to the storage of TCPP in large vessels which are located in large bunded areas. TCPP is moved in a closed system and pumped direct from the storage vessels to the mixing head. No water washing is used anywhere on site.

Emissions to air from foaming are also effectively zero. The foaming process is enclosed, with all fumes emitted through an activated carbon filter or other abatement methods. Studies by the International Isocyanate Institute indicate that the concentrations of TCPP emitted in exhaust gases from laydown and cutting processes are detectable but below the level of quantification. All UK based foamers operate in a similar manner owing to worker safety legislation controlling exposure to di-isocyanates. 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).

Water is not used to clean the mixing head or other machinery. When mixing vessels require cleaning, the plant is shut down and a polyol or solvent flush is used. Methylene chloride is used, drummed and sent for re-distillation. Thus, emissions to water from foaming are also zero.

There are emissions to solid waste from foaming, arising from the disposal of the polythene used to line the sides of the foam blocks. Around 2 mm of foam adheres to the blocks when the polythene sides are removed. The paper used to line the base of the blocks is removed at the cutting stage with no loss of foam. Taking account of the area of the side panels, 0.2% of a block is lost with the disposal of the side panels to landfill (i.e. 2 kg/tonne of TCPP). Thus, for a site handling 1,920 tonnes per annum of TCPP, 3.67 tonnes of TCPP will be lost to landfill.

0.2% TCPP to solid waste

Releases from curing and storage

Peak exothermic conditions occur approximately one hour after foaming i.e. during the curing phase. There is thus the potential for TCPP release during curing, since the foam is at elevated temperatures, e.g. up to 150^{o}C for several hours (depending on the size of the block). Data provided by the foam producers indicate that at any one time, up to 2.5% of the TCPP used at the facility could be present in blocks undergoing curing and storage. This figure is based on data on the tonnage of foam present on the site and the loading rate of TCPP. Thus for a site handling 1,920 tonnes per annum TCPP, 48 tonnes could be present at any one time in blocks undergoing curing and in storage.

The proposed rate of release in curing and storage, accounting for the finding that for TCPP, only 40% of the substance present is available for release, is 1.2E-04% to air and to wastewater. This is based on a model which brings together theoretical modelling with the findings of various published studies (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 is included for small sites.

Releases to air: 1.2E-04%

Releases to wastewater: 1.2E-04% (large sites)

0.01012% (small sites)

3.1.2.3.2 Foam cutting and manufacture of furniture

There may also be losses to the environment associated with the cutting of slabstock foams during cutting and trimming processes and manufacture of furniture. 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 with industry 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 0.0002% to air and 0.0002% to water is proposed, accounting for the finding that for TCPP, only 40% 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).

In the absence of specific information the following set of values are used in the assessment:

Fraction in the main region = 0.75
Fraction of the main local source = 0.05 (from Table B3.9)
Number of days per year = 300

Thus, the largest site handles ~3200 tonnes foam, i.e. approximately 250 tonnes TCPP per year, consistent with approximately 11 tonnes foam being cut per day. This combination of factors reflects the focus of TCPP foam processing in the UK.

3.1.2.3.3 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 10.5% of the TCPP in furniture foams (see section 2.2.2.2.5) will be rebonded in the EU (this is based on the combination of 20% of foam being available for recycling; 75% remaining in EU for recycling; and 70% of recycling being in the form of rebonding[17]). (Neither the quantity of TCPP-containing foam that is rebonded nor the concentration of TCPP in the rebond is relevant to this assessment as releases are estimated on the total amount of TCPP 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 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 4E-04% to air, accounting for the finding that for TCPP, only 40% of the substance present is available for release. There are no releases to wastewater (Appendix B).

A survey carried out by EUROPUR has produced results in the form of numbers 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. Conclusions have been drawn for TCPP-containing foam, taking into account the known concentration of

[17] 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.

foam manufacture in the UK and Ireland. The following set of values is used in the risk assessment:
Fraction in the main region = 0.9
Fraction of the main local source = 0.55
Number of days per year = 300

As for the foaming stage, a loading rate of 8% is used.

Thus the largest site handles 353 tonnes of TCPP, which is consistent with 14.7 tonnes of rebonded foam produced per day.

Loose crumb

It is assumed that 4.5% of the TCPP in furniture foams (see section 2.2.2.2.5) will be recycled as loose crumb in the EU (this is based on the combination of 20% of foam being available for recycling; 75% remaining in EU for recycling; and 30% of recycling being in the form of loose crumb[16]).

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 4E-04% to air, accounting for the finding that for TCPP, only 40% 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 06). 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.

In the absence of specific information the following set of values are used in the assessment:
Fraction in the main region = 0.9
Fraction of the main local source = 0.55
Number of days per year = 300 (from Table B3.9)

As for the foaming stage, a loading rate of 8% is used.

Thus the largest site handles 151 tonnes of TCPP, which is consistent with 6.3 tonnes of loose crumb foam produced per day.

3.1.2.3.4 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 multiply these rates by 5 (in accordance with the correction applied to rates of loss from conversion) to obtain the rate of loss of TCPP. Therefore the default release rates can be taken to be:

Indoor service:

Loss to air	0.25% over lifetime
Loss to wastewater	0.25% over lifetime

Outdoor service:

Loss to air	0.25% over lifetime
Loss to wastewater	0.75% per year

Values used in the risk assessment: Furniture and mattresses

It is known that the vast majority of flame-retarded furniture containing TCPP is used in the UK and Ireland. Therefore a fraction of 0.9 in the main region is used.

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 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 9.6E-03% per year to air is proposed, accounting for the finding that for TCPP, only 40% 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 TCPP 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 2.2.2.2.5). 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 9.6E-03% per year to air is proposed, accounting for the finding that for TCPP, only 40% 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 0.096% per year to air is proposed, accounting for the finding that for TCPP, only 40% 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 TCPP is limited by the available fraction (for TCPP, only 40% 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 0.8% 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 TCPP used in flexible foams used for furniture, rebonded foam and loose crumb furniture. The releases are evaluated on a regional scale, with the same 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 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.

3.1.2.4 Release from rigid foams: Use C

3.1.2.4.1 Loading rate

Data on the loading rate for TCPP levels in rigid foam are given in **Table 3.1**. One reason for the variation in loading rates could be that TCPP can be used alone or in combination with other flame retardants (e.g. brominated polyols or other organic phosphoric acid esters) in polyurethane insulation (Leisewitz A, Hermann K and Schramm E, 2001).

Table 3.1 Loading rates for rigid foam

Application	Loading rates	Source
Insulation board (flexible faced laminate)	2% to 25% (lowest 2% to 3%, highest 20% to 25%)	Questionnaires
	0% to 20% in the polyol component 0% to 10% of the foam	ISOPA 2003
	15% to 20% of the polyol component 7% to 10% of the PUR system	Schupp 2001
Insulation foams	approx. 5% of weight	Leisewitz A, Hermann K and Schramm E (2001)

3.1.2.4.2 Rate of release from board manufacture

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 rigid polyurethane foam are likely to be associated with:

- the handling of the flame retardant prior to mixing with other ingredients (TCPP is a liquid);
- 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.

Although not used as the numerical basis of the risk assessment, it is of interest to explore the use of ESD defaults. The ESD estimates a rate of 0.01% to wastewater from handling of raw materials. This route of release would theoretically still apply for the foamers using TCPP in systems, not only for those adding TCPP direct, to account for possible spillage of the formulation. However, ISOPA (2002b) states that >80% of rigid foamers "add the foam under closed loop conditions". This is taken to indicate that there is no need to account for handling losses for rigid foam, since controls are so widely applied.

The ESD sets out rates of release of various additive types from various types of process. As for flexible foam producers, the ESD notes that smaller sites may have up to ten times higher releases, but this is not applied in the case of the polyurethane industry, as explained in section 3.1.1.

Rigid foam producers:0.025% to air
0.025% to wastewater

Values used in the risk assessment

Foam blocks are large and the air around them would probably be saturated with TCPP vapour. The presence of facing panels will be an important additional retarding factor. The proposed rate is therefore 6.6E-06% 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-05% per year, for all sites. Accounting for the finding that, for TCPP, only 40% of the substance present is available for release, the resulting release rate for use in the risk assessment is 9.6E-06%. This is divided equally between air and wastewater, i.e. 4.8E-06% to each per year. This is based on a model which brings together theoretical modelling with the findings of various published studies (Appendix B).

An additional release of 0.01% to wastewater from handling of raw materials is included for small sites.

Releases to air: 4.8E-06%

Releases to wastewater: 4.8E-06% (large sites)

0.0100048% (small sites)

Number and nature of sites

The B-tables (Table B3.9) gives the following for IC11, processing, loading rate 10%, with 10% in the main region.

Fraction of the main local source = 0.1
300 days' operation per year

ISOPA data (ISOPA undated) indicates that 500 insulation foam manufacturers are involved in the production of construction materials from PUR in Europe each year, consuming 500,000 tonnes of polyurethane.

Questionnaires were returned by just nine producers of rigid foam for use in construction, one of which is located in Switzerland. Eight produce insulation board. The eight EU-based facilities account for 3,005 tonnes of TCPP, just 12% of the tonnage associated with construction applications. Further information is given in the Confidential Annex. A questionnaire was also returned by one further site using TCPP in the production of PIR rigid cell foam for insulation.

A loading rate of 10% is used in the risk assessment.

Of the 26,650 tonnes of TCPP that were used by rigid foamers in the year 2000, 70% was added via systems and the rest direct by rigid foamers. In general, large rigid foamers will tend to use TCPP direct, with systems used by smaller producers.

Based on the above and information in the Confidential Annex, the following set of values would be preferable to the B-table defaults for larger sites, accounting for 30% of the tonnage of TCPP, added directly by the foamers:

Fraction in the main region = 0.2
Fraction of the main local source = 1
Number of days per year = 300

Indicates 1,500 tonnes of TCPP consumed at the main site each year and 5 tonnes foam per day.

For smaller sites, accounting for 70% of the tonnage of TCPP present in systems:

Fraction in the main region = 0.1
Fraction of the main local source = 0.175
Number of days per year = 300

Indicates 300 tonnes of TCPP consumed at the main site each year and 1 tonne foam per day.

All rigid foam tonnage is treated as foam for building use.

Adhesive pressing Use Q

Approximately 400 tonnes of TCPP contained in rigid foam scrap go to adhesive pressing. Rates of release from the process of adhesive pressing are read across from the ESD in the absence of further information. The ESD sets out rates of release from various types of processing.

For open systems (worst case):
0.25% to air
0.25% to wastewater

Accounting for the finding that for TCPP, only 40% of the substance present is available for release, the resulting release rates for use in the risk assessment are 0.1% to air and to wastewater.

The site distribution is taken from the B-tables, assuming a fraction of 0.4 in the main region.

From Table B3.9:
Fraction of the main local source 0.15
Number of days 96
Implies 2.5 tonnes per day processed at the main region.

This number of sites is consistent with this use being associated with rigid foam production.

With regard to in-service loss, the main applications are furniture in kitchens and sailing boats, and flooring material, e.g. in gymnasiums, which need to have a certain elasticity (see ISOPA 2001b).

While use of adhesive pressed foam is in rigid panels, in-service loss cannot simply be read across from the value used for rigid foams (see section 3.1.2.4.3 below), as the panels are not sealed into the structure in the same way as sandwich panels. Rates of release are taken from the ESD, for indoor service (see section 3.1.2.3.4):

Loss to air 0.25% over lifetime
Loss to wastewater 0.25% over lifetime.

Assuming an average service life of 30 years and accounting for the finding that for TCPP, only 40% of the substance present is available for release, the resulting release rate for use in the risk assessment is 3.33E-03% per year to both air and wastewater.

The fraction in the main region is taken to be 0.1 for the service life.

In the absence of firm information it is assumed that the entire tonnage goes to landfill at the end of life.

3.1.2.4.3 In-service losses

Losses from foams for in-structural use in buildings

From TCPP applications such as insulation panels and window frame sealant foam, which are effectively sealed within building walls, the rates of loss from the ESD are far too high. Air circulation would be negligible around the exposed foam and edges of panels and hence releases from these panels in service need not be considered further in this risk assessment. A lifetime of 30 years is used, on the basis of information from ISOPA (1997).

3.1.2.5 Release from spray foams: Use D

These foams are formulated by systems houses and foamed at the point of use (i.e. on site) and not in purpose built foaming facilities. Thus emissions from foaming will be direct to the environment. Emissions from the formulation of spray foams in systems houses are accounted for in Use A.

Foaming on-site

It is inappropriate to apply the rates of loss for foaming from the ESD, as this is foaming at the point of service (often building sites or outside). In view of the uncharacterised nature of the sites of loss it is most appropriate that releases from these stages should be evaluated on a regional level.

Releases from foaming *in situ* are based on the rate of release in service. Based on an uncovered foam (at the time of spraying) the loss rate can be estimated as 0.00066% per day (i.e. 0.24% over the year) (Appendix B). Accounting for the finding that for TCPP only 40% of the substance present is available for release, the resulting release rate for use in the risk assessment is 0.096% to air. A fraction of 0.25 in the main region is used in the risk assessment.

In-service loss

Air circulation would be negligible around the foam and hence releases from spray foams in service need not be considered further in this risk assessment. The service life of spray foams is given as 25 years by Kraehling H and Zipfel L (2000).

3.1.2.6 Release from one-component foams: Use E

3.1.2.6.1 Compounding

Loading rates

The German report on flame retardants gives a 14% loading rate for one component foams (Leisewitz A, Hermann K and Schramm E, 2001). However, concentrations of TCPP in any initial polyol blend can be considerably higher. Data provided by manufacturers of PUR foam in cans indicate loading rates from 6% to 20% TCPP in the aerosol can. One reason for variation is that TCPP can be used alone or in combination with other flame retardants (e.g. brominated polyols or other organic phosphoric acid esters) in one-component foams (Leisewitz A, Hermann K and Schramm E, 2001). It has more recently been stated that the typical "highest level of TCPP containing formulations" contain <15% TCPP. Other formulations mainly contain Chloroparaffins (CP) or combinations TCPP/CP (Typical TCPP levels: 0 to 5%). There has been a trend away from TCPP to lower cost CP in one-component foams, and TCPP is now only used in formulations where it is really needed for certain performance reasons (ISOPA and the rigid polyurethane foam industry, 2006).

Based on the information presented above, a loading rate of 15% is considered appropriate, though recent information from industry suggests this may be an overly conservative model.

Rate of release

The ESD defines separate rates of loss from handling of raw materials at compounding sites and from the compounding process itself.

From the ESD:

Losses from handling	= 0.01% to wastewater
Total losses from compounding	= 0.025% to air
	= 0.025% to wastewater

Producers of one-component foams indicate that these are produced in completely sealed units. There is no process water. There are no site drains. Thus they suggest emissions to water should be zero (pers. comm. 31st July 2002, producers and downstream users). There is some evidence available, from an industry monitoring study undertaken at a European one-component foam plant (Tauw, 2007), to suggest that releases to air might be very much lower than those modelled, when best practice is followed[18]. However, without wide scale evidence including consideration of all processes on site, including handling and cleaning processes, this has not been accepted for the purposes of risk assessment.

The information from industry would suggest that the model used in the risk assessment may be over-conservative in respect of the life cycle stage of manufacture of one-component foams, both in terms of overestimated loading rate and possible emissions of TCPP to waste water. This may mean that PEC/PNEC ratios presented are conservative. However, in view

[18] The same sampling strategy was followed as for systems houses, and so the results have similar drawbacks. In this case, emissions are given in terms of unit operations (<0.02 g TCPP per truck unloading operation). The ESD default fraction released to air associated with blending processes was 2.5E-04. Release from the monitoring work could suggest that overall releases would be unlikely to be above 2E-06. These figures are subject to too much uncertainty to use in place of ESD defaults, but are indicative that the ESD could be significantly overestimating release potential, at sites where best practice is applied.

of the various uncertainties, it is considered that the model as it stands represents a realistic worst case.

The B-tables (Table B2.3) give the following for IC11, formulation, loading rate 15%, with 10% in the main region:
Fraction of the main local source = 0.8
300 days' operation per year

The TCPP industry has indicated that one component foams (i.e. can foams) tend to be made by large companies as making these is a difficult process (Pers. comm. 16/10/01). However, TCPP usage per site does not bear this out.

Based on the available information about systems houses (section 2.2.2.1), the following set of values would be preferable to the B-table defaults:
Fraction in the main region = 0.48
Fraction of the main local source = 1
Number of days per year = 300

This implies that the main site produces 20 t formulation per day.

3.1.2.6.2 Foaming

It is inappropriate to apply the rates of loss for foaming from the ESD, as this is foaming at the point of service (often building sites or outside). In view of the uncharacterised nature of the sites of loss it is most appropriate that releases from these stages should be evaluated on a regional level.

Releases from foaming *in situ* are based on the rate of release in service. Based on an uncovered foam (at the time of spraying) the loss rate can be estimated as 0.00066% per day (i.e. 0.24% over the year) (Appendix B). Accounting for the finding that, for TCPP, only 40% of the substance present is available for release, the resulting release rate for use in the risk assessment is 0.096% to air. A fraction of 0.20 in the main region is used in the risk assessment (total tonnage 2400 t including imports).

<u>In-service loss</u>

Air circulation would be negligible around the foam and hence releases from spray foams in service need not be considered further in this risk assessment.

3.1.2.7 Release from other uses

Rates of release and site sizes for other uses are discussed in the Confidential Annex.

3.1.2.8 Release from disposal

3.1.2.8.1 Releases of TCPP from landfill

Since TCPP is relatively soluble in water and leaching is a definite possibility, the likelihood of emissions from landfill sites should be considered. Due to the variety of landfills, in respect of their design, age and use, theoretical modelling of landfill emissions is difficult. Therefore, the Environment Agency of England and Wales conducted measurements of the concentration of TCPP in landfill leachate, during 2005 (pers. comm., 3rd August 2005). Fifty-eight data points from 22 locations were obtained. Of these, 16 were above the limit of quantitation of TCPP (10 µg/l). The data do not comply with every quality criterion of the TGD on a site-by-site basis but are of sufficient quality to use in the risk assessment, particularly when the data from the sites are combined.

Sources of FR-PUR in landfill in the risk assessment of TCPP include:

- Post-consumer (i.e. a proportion of end of life) flexible foam, rebond, and loose crumb foams
- Construction wastes (i.e. a proportion of end of life rigid foam, spray foam, one-component foam and adhesive pressed foam)
- Industrial wastes including solid wastes/sludges from industrial processes (virtually impossible to estimate quantitatively), peeled slabstock foam 'skins', etc.
- Confidential life cycle stages: end-of-life disposal.

Overall, the amount released to landfill can be estimated based on the tonnages of different types of end product containing TCPP and their distribution in service. Further justification for the quantities released to landfill is given in section 3.1.2.8 of the Confidential Annex. Annually, the RAR model suggests, approximately:

- Domestic wastes: ca. 5100 t TCPP per year in the main region
- Industrial/commercial wastes: at least ca. 12 t TCPP per year in the main region
- Construction wastes: ca. 2700 t TCPP per year in the main region.

Since some of these waste streams also include contributory volumes relating to confidential applications, it is not possible to break down these figures exactly here. (N.B. none of the above accounts for volatile releases in service or through waste remaining in the environment).

Screening with MOCLA model

The MOCLA model (Kjeldsen and Christensen, 2000) was applied to TCPP and the results suggested that landfill releases could be important. Disposal quantities of the order of magnitude indicated above were used in the MOCLA modelling. MOCLA is not well validated for adsorbing organic substances, and therefore these results should be treated with caution. Furthermore, there is uncertainty over the degradation rates to use. Absolute results from this model are therefore not useful but relative results are likely to be of use. TCPP has been in use for some years, and so it is not surprising that it can be detected. General

consideration of chemicals in landfill suggests that TCPP meets the expected criteria for being found in leachate: it is fairly stable, has appreciable water solubility and low volatility.

Although the MOCLA model is not fully validated, its use does suggest that at the time of writing (2006), we are entering or have already entered the period of maximum TCPP release, on the basis of the known use pattern and dates for implementation of the Landfill Directive. Hence, the results of recent monitoring (see below) could be considered representative of the expected highest levels.

Information derived from landfill monitoring of TCPP releases

Summary of data

The results are listed in **Table 3.2**. Most are less than the limit of quantification (LOQ), 10 µg/l. Three results reported in the spreadsheet, which were less than this value, are considered questionable but for simplicity they have been set to half the LOQ. It should be noted that all individual data points (concentrations in µg/l), including <LOQ results, are used in the analysis.

Table 3.2 Environment Agency data for UK landfill sites

Location	Concentration data	Leachate volume	Other details
Thames Region			
Pickeridge Farm	Three data points, one at 19.6 μg/l.	No data on leachate volume.	
Rainham Clearaway	Three data points, 11.6, 13.3 and <10 μg/l.	No data on leachate volume.	
Prospect Park	No values above LOQ		
Hatfield Quarry	No values above LOQ		
Wood Farm	Three data points out of eleven above LOQ - 15.5, 21.1, 17.7 μg/l.	No data on leachate volume.	
Patterson Court	Three data points, one at 19.8 μg/l.		Dilute and disperse landfill, used to use fragmentiser waste (including shredded foam) as cover material.
Norlands Lane	Three data points, one at 12.8 μg/l.	No data on leachate volume	
Trumps Farm	Three data points, two above LOQ - 38.5 and 21.9 μg/l (avg. = 23.5).	Leachate volume 427 000 gallons per year = 95 000 L, giving a total of **2.2 g TCPP per year**.	
Beddington Farmlands	Three data points, one at 66.6 μg/l.		Clay lined and capped
Ardley	Three data points, one at 24 μg/l.	Maximum leachate = 100 m^3/d. Release rate of TCPP = 24 x 10^{-6} x 100 x 10^3 g/d = **2.4 g/d**	
Purton	Three data points, two above LOQ – 25.3 and 55.5 μg/l (avg. = 30.3 μg/l).	Maximum leachate flow rate = 140 m^3/d. Release rate of TCPP = 30.3 x 10^{-6} x 140 x 10^3 g/d = **4.2 g/d**.	
Chapel Farm	Three data points, one at 14.7 μg/l.	Information on leachate but time scale not indicated.	
High Heavens	No values above LOQ		
Anglian Region			
Folly Farm	Concentration of TCPP unclear.	No data on volume.	
Gayton	No values above LOQ		
Bluewater	No values above LOQ		
Southern Region			
Efford	One value at 39.3 μg/l.	Produced at 100 m^3/d. Release rate of TCPP = 39.3 x 10^{-6} x 100 x 10^3 = **3.9 g/d**.	
Wales			
Abernant	No values above LOQ		
Giants grave	No values above LOQ		

Results are considered in terms of mass flow and absolute concentration. The highest concentration of 67 μg/l in a flow of 100 m^3/day (a typical maximum) would give 6.7 g/d.

There is a wide spread of landfill leachate volumes, reflecting the variety of landfill types and practices at the different locations. The volume data was provided by local inspectors and is considered to be reliable.

Interpretation

There is insufficient data about leachate volumes to calculate many mass flow values. However, the raw concentrations can be analysed and compared to the cadmium concentrations obtained in the risk assessment of cadmium, which also considered landfill releases (EC 2005). By setting '<LOQ' values equal to 5 μg/l and taking the arithmetic mean across the data set, a reasonable worst case for TCPP is 11±4 μg/l, where the ± represents a 95% confidence interval assuming a normal distribution. However, statistical analysis of a data set containing mostly '<LOQ' values is difficult. It cannot be determined whether the data do or do not fit a normal distribution. Use of a geometric mean would underestimate the importance of the definite values; use of a 90th percentile would give a high value unsuitable for extrapolation for estimation of regional releases. The highest concentration was 67 μg/l.

Comparison to Cadmium RAR

A similar approach to that outlined in the cadmium risk assessment report is used to predict emissions from landfill (EC 2005). Landfill sites which could contain cadmium battery waste are likely to be municipal, with the batteries arising from domestic consumers. Industrial scale batteries, or plating waste, would not ordinarily be found there. Therefore it is reasonable to expect that such sites would be comparable with landfills receiving PUR waste (containing TCPP) from domestic sources. Commercial sources of FR-containing wastes would be treated differently. Construction waste is also important for TCPP: it is understood that co-disposal of construction with domestic waste is not common (e.g. the TGD refers to them as separate types of site). Domestic waste disposal dominates the model for disposal in the main region (i.e. in the UK).

Extrapolation from the cadmium report

The Cadmium RAR uses 5 μg/l cadmium as the concentration in landfill leachate.

A factor of 2.2 (11 μg/l / 5 μg/l) is applied to cumulative cadmium emissions expressed as mass flow, to estimate equivalent TCPP emissions. The figures, at the local level, are derived from the 20-year figures for emissions to water given in Table 3.1.50 of the cadmium report.

The report describes two landfill profiles,

- Landfill profile 1: landfill with bottom liner and final top layer consisting of a single compacted clay liner
- Landfill profile 2: landfill with bottom liner and final top liner consisting of a single composite liner

and scaled up from cadmium rates these give TCPP releases as follows, assuming a leachate emission of 100 m^3/d:

Profile 1: 0.46 kg collected (0.062 g/d)

11.1 kg fugitive (30 g/d)

Profile 2: 5.5 kg collected (7 g/d)

0.23 kg fugitive (0.031 g/d)

These are not contradictory to the measured data derived in **Table 3.2**. Any fugitive releases can be ignored in local scale assessment, because they are unlikely to reach surface water, although there is a possibility of release to groundwater.

Local risk assessment

Landfill leachate should be disposed of via a municipal wastewater treatment plant, in accordance with the Landfill Directive 1999/31/EC. The highest available release extrapolated from the cadmium report is 30 g/d in total, which potentially could all be collected. If this were released to a standard (i.e. TGD default) WWTP then the PEC water would be

$$\frac{30 \times 10^3 \times 0.98}{2 \times 10^7} = 1.5 \times 10^{-3} \text{ mg/l}$$

A rate of 4.2 g/d is the highest available from the Environment Agency measured data set. If this were released to a standard WWTP then the PEC water would be

$$\frac{4.2 \times 10^3 \times 0.98}{2 \times 10^7} = 2.1 \times 10^{-4} \text{ mg/l}$$

The PNEC is 0.64 mg/l. Hence there is no apparent likelihood of local risk, expressed as PEC/PNEC and a local risk assessment of TCPP need not be performed in detail in the RAR.

Regional risk assessment

At the regional level, the cadmium RAR gives a UK flux of cadmium of 272 kg/y, which can be approximated (scaled up) to (2.2 x 272 kg/y)/365 = 1.64 kg/d of TCPP to water. For the rest of the EU, an equivalent figure can be derived from the proportion of wastes expected to find their way annually into municipal landfills, based on annual tonnages in service in relevant applications. Approximately three-quarters of the total volume of TCPP in waste landfilled in EU are disposed of in the main region. In other words, 1 tonne TCPP enters municipal landfill outside the main region (at the EUSES 'continental' scale) for every 3 tonnes inside the main region. Therefore the equivalent release at the continental level would be approximately one third of the regional release, i.e. 0.55 kg/d (i.e. an EU total of 2.19 kg/d). These would be taken as the Regional and Continental contributions respectively. In accordance with the TGD defaults, and supported by the available information about landfill operation, it is assumed that for a minority (20%) of sites the release will be direct to surface water rather than entering the municipal system. Whilst this is a somewhat arbitrary assumption, it allows for a realistic treatment of the fact that older landfills may have leakage.

This should be compared to the releases from other life cycle stages. In the current version, regional releases from all the other life cycle stages are modelled to be around 17.5 kg/d and continental scale around 23.5 kg/d (a total of 41 kg/d) to wastewater for TCPP. Therefore the

releases from landfill are significant enough to include, which has been done, but are relatively small contributors (ca. 7%) to the total regional release.

An alternative method would be to consider the number of landfills in the UK that might be releasing TCPP. There are a total of ca. 2500 landfills; take a flow of (for example) 3 g/d; 700 such domestic landfills (a reasonable estimate) would give 2.1 kg/d, which is a reasonable agreement with 1.64 kg/d.

Conclusion

A limited amount of monitoring data on concentrations of TCPP in landfill leachate have been interpreted to provide a generic worst case local release, and a separate calculation of total regional and continental releases. Landfill leachate makes a significant contribution (ca. 7%) to the total regional releases of TCPP to wastewater, and this has been included in the risk assessment.

3.1.2.8.2 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)
- 22% used for energy recovery.

Industry indicates that at end of life most furniture goes to landfill (see section 2.2.2.2.5). For the purposes of this risk assessment it is assumed that all furniture is landfilled at end of life.

3.1.2.8.3 End of life for rigid foams

Table 3.3 gives details of lifetimes for construction products for a range of publications.

The ESD considers losses from disposal of polymer additives. For both incineration (air and water) and landfill (air) the emission factor for flame retardants is 0%. With respect to losses to water from landfill, the ESD indicates that these will depend on many factors relating to the type of landfill as well as the properties of the additive and the nature of the polymer in which it was used. The maximum potential loss could be calculated from the total amount of additive remaining in the plastic at disposal.

Table 3.3 Lifetimes for construction products

Application	Lifetime	Comments/Source
Buildings and construction	>10 years	Emission Scenario Document (OECD 2004)
Construction (PUR specific)	> 25 years	ISOPA (1997)
Insulation foams	> 50 years	When insulation foams are salvaged from buildings, are expected to have served > 50 years. ISOPA (ISOPA 1996b)
Insulation board	50 years	Used in cavity walls and warm pitched roofs of domestic houses Kraehling H and Zipfel L (2000)
Insulation waste arisings	2% in < 2years 10% in 10 to 20 years 50% in 20 to 40 years 38% in > 40 years	APME (undated) uses these lifetimes to estimate insulation waste arisings

The ESD indicates that plastics constitute 5% of construction/demolition waste arisings. Of this waste stream, 10% is mechanically recovered, and the remaining 90% is landfilled or incinerated.

Data from APME (2000) for 1998 indicate that of the 585,000 tonnes of plastic present in building and construction waste in Europe 91% is landfilled and the rest mechanically recovered.

3.1.2.8.4 End of life for spray foams

The ESD considers losses from disposal of polymer additives. For both incineration (air and water) and landfill (air) the emission factor for flame retardants is 0%. With respect to losses to water from landfill, the ESD indicates that these will depend on many factors relating to the type of landfill as well as the properties of the additive and the nature of the polymer in which it was used. The maximum potential loss could be calculated from the total amount of additive remaining in the plastic at disposal.

The ESD indicates that plastics constitute 5% of construction/demolition waste arisings. Of this waste stream, 10% is mechanically recovered, and the remaining 90% is landfilled or incinerated.

Data from APME (2000) for 1998 indicate that of the 585,000 tonnes of plastic present in building and construction waste in Europe 91% is landfilled and the rest mechanically recovered.

3.1.2.8.5 End of life for one-component foams

The ESD considers losses from disposal of polymer additives. For both incineration (air and water) and landfill (air) the emission factor for flame retardants is 0%. With respect to losses to water from landfill, the ESD indicates that these will depend on many factors relating to the type of landfill as well as the properties of the additive and the nature of the polymer in which it was used. The maximum potential loss could be calculated from the total amount of additive remaining in the plastic at disposal.

The ESD indicates that plastics constitute 5% of construction/demolition waste arisings. Of this waste stream, 10% is mechanically recovered, and the remaining 90% is landfilled or incinerated.

Data from APME (2000) for 1998 indicate that of the 585,000 tonnes of plastic present in building and construction waste in Europe 91% is landfilled and the rest mechanically recovered.

3.1.2.9 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 3.4** below.

Table 3.4 Total releases to the regional and continental environmental compartments

Endpoint	Emission in kg/d
Total regional emission to air	134.85
Total regional emission to wastewater	18.70
Total regional emission to surface water	4.68
Total regional emission to industrial soil	0.86
Total continental emission to air	89.56
Total continental emission to wastewater	24.09
Total continental emission to surface water	6.02
Total continental emission to industrial soil	7.78

3.1.3 Environmental fate

3.1.3.1 Degradation in the environment

Results are summarised in **Table 3.5**.

3.1.3.1.1 Atmospheric degradation

Photodegradation

A half-life in air of 8.6 hours has been proposed based on an OH radical concentration of 5 x 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 44.76 x 10^{-12} cm^3/molecule.sec. With the TGD model for photodegradation, this is equivalent to a half-life of 8.6 h, suggesting that the rate reported by industry was estimated using AOPWIN.

```
SMILES : O=P(OC(CCL)C)(OC(CCL)C)OC(CCL)C
CHEM   : 2-Propanol, 1-chloro-, phosphate (3:1)
MOL FOR: C9 H18 CL3 O4 P1
MOL WT : 327.57
------------------- SUMMARY (AOP v1.90): HYDROXYL RADICALS ----------------
Hydrogen Abstraction       =  44.7631 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 =  44.7631 E-12 cm3/molecule-sec
```

3.1.3.1.2 Aquatic degradation

Abiotic degradation

In a GLP-compliant report (Geurts and van Veenendaal, 2001), preliminary hydrolysis testing was undertaken at 50ºC for five days. A decrease in concentration of less than 1% was observed at pH 4, 7 and 9. Based on this result, it is concluded with no need for further testing in accordance with the OECD guideline, that TCPP is stable in water at pH 4, 7 and 9 at 25ºC, with a half-life greater than or equal to one year.

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, and discussion is general only.

Phosphate esters are known to hydrolyse although this is expected to be slow under environmentally relevant conditions.

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.

Biodegradation studies

Sludge was collected from ten sites in Japan: four sewage plants and six surface waters (rivers, a lake and 'bays') for a MITI study (MITI, undated). Samples were taken regularly and fresh and old samples were mixed. Sludge was present at 30 or 100 mg/l in test vessels. No information is given on the purity of test substance. The test substance was present at 30 or 100 mg/l. No degradation (0%) was observed with sludge present at 30 mg/l and test substance at 100 mg/l. The report does not set out results in any detail and does not report values for other loading rates. Aniline was used as the reference substance, present at 100 mg/l. The rate and extent of aniline degradation fulfilled the validity criteria for the test.

In a GLP-compliant study (Bayer, 1991a; test report in German) sludge (mixed population) was obtained from communal lab outflow/sewage drain, not from municipal sewage plant. Sludge level in test vessels was unclear. The test substance was present in test vessels at 23 mg/l DOC at the start. 14% degradation had occurred by day 28. However the measurements during the test period are inconsistent, recording 19% degradation on day 7 and 14. Aniline was used as the reference substance, present at 20 mg/l DOC. The rate and extent of aniline degradation fulfilled the validity criteria for the test.

Activated sewage sludge was sampled from ten sites in the UK for a study of inherent biodegradability (SafePharm, 1996). The report is a summary and does not claim compliance with GLP. Sludge was present in test vessels at 100 mg/l. The test substance was present in the test vessels at a concentration of 30 mg/l. 21% degradation was observed by day 28. Degradation was assessed on the basis of a ThOD of 1.17 mg O_2/mg since the test substance was considered to be too insoluble to use DOC. Aniline was used as the reference substance, present at 100 mg/l. Aniline degradation fulfilled the test validity criteria, based upon DOC measurements.

The test is reported to provide 'evidence of inherent biodegradability'. This conclusion would not be reached today because stricter criteria now apply. The conclusion would also appear to be unsound; there appears to have been an acclimation period of around 13 days at the start of the test, followed by rapid degradation over three days (up to 13%). There then followed a period of slow degradation that had not reached a plateau by the end of the 28-day exposure period, although it had reached 21%. More information could have been obtained from a study with a longer exposure period, but the evidence from this summary indicates that the substance is susceptible to partial degradation.

A mixed inoculum from soil, activated sludge and raw influent sewage was used in a study of biodegradation (Madsen, 1993). The substance was present at 10 mg C/l (this concentration is valid only for the degradation phase, and not for the acclimation phase). There was a two-week acclimation phase, and then degradation was monitored over 28 days, using DOC and CO_2 evolution. The test substance and reference substances were the sole carbon sources in their respective tests. The extent of degradation was close to zero at the end of 28 days. The reference substance (dextrose) was degraded completely.

A SCAS inherent test has been submitted (Van Ginkel and Stroo, 2001). The acclimation period was long (approximately 3 weeks), and was followed by a period of rapid degradation. The result suggests that aqueous environments exposed regularly to TCPP could support strains capable of degrading the substance. This might apply to some larger sites should releases occur. The SCAS result is an indicator of inherent biodegradability; however, the TGD does not allow biodegradation rate constants greater than zero (i.e. no degradation) to be set on the basis of this test.

A prolonged closed bottle test has been performed which confirms that TCPP can be classed as inherently biodegradable under aerobic conditions (van der Togt and van Ginkel, 2002). Secondary activated sludge was obtained from a wastewater treatment plant treating predominantly domestic wastewater. It was used at 4 mg dw/l, and TCPP was present at 4 mg/l. The sludge was aerated for one week prior to the start of the test. Oxygen consumption was measured at 7 day intervals up to 28 days (at which point the degradation had reached 13%), and thereafter at 42, 56 and 84 days. After Day 21 degradation started and reached 60% by day 50. At the end of the study the oxygen consumption was equivalent to complete mineralisation.

Whilst not a standard study, the prolonged closed bottle test appears acceptable. The test conditions were unfavourable relative to standard inherent tests such as the Zahn-Wellens test (e.g. much lower concentration of inoculum). Significant degradation was observed, and the positive result of the SCAS test supports this outcome.

Based on the weight of evidence, some biodegradation in the environment should be allowed for, though the criteria for degradation in the WWTP are not met. Therefore, TCPP is evaluated as 'inherently biodegradable, not fulfilling the criteria' in this risk assessment.

3.1.3.1.3 Degradation in soil

No soil degradation data are available for review.

3.1.3.1.4 Summary of environmental degradation

Table 3.5 summarises the results of studies described in section 3.1.3.1, and **Table 3.6** shows the implications for the rate of degradation.

Table 3.5 Summary of environmental degradation for TCPP

Endpoint		Year test completed	Protocol cited	Results	Reliability[1]	Study reference
	Hydrolysis of TCPP	2001	EC method C10	$t_{1/2}$ >1 year at pH 4, 7 and 9 at 25ºC	(1) valid without restriction	Geurts and van Veenendaal, 2001
	Phosphate ester hydrolysis	1992		Discussion of mechanisms only. Rates not given	(4) not assignable	Gerlt (1992)
	Stability in soil	-	-	-	-	-
	Distribution	-	-	-	-	-
28d	Ready biodegradability	Unknown	MITI	Not readily biodegradable	(4) not assignable	MITI, undated
28d	Ready biodegradability	1991	OECD 301e	Not readily biodegradable	(4) not assignable	Bayer, 1991a
28 d	Effectively a 'ready test' preceded by an acclimation phase	1993	USEPA TSCA 796.3100	Not biodegradable	(1) valid without restriction	Madsen, 1993
28d	Inherent biodegradability	1996	Modified MITI (II)	"evidence of inherent biodegradability"	(4) not assignable	SafePharm, 1996
84 d	Prolonged closed bottle test	2002	EC method C6 modified	Inherently biodegradable	(2) valid with restrictions	van der Togt and van Ginkel, 2002
64 d	Inherent biodegradability	2001	OECD 302A (SCAS)	Inherently biodegradable	(2) valid with restrictions	van Ginkel and Stroo, 2001

[1] Klimisch code

Table 3.6 Summary of estimated ultimate biodegradation rate constants for use in the EUSES model

Compartment		Reaction rate constant	Half-life
Wastewater treatment plant		0 d^{-1}	Infinite
Surface water		4.62 x 10^{-3} d^{-1}	150 d
Soil	Kp_{soil} = 11.5 l/kg	2.31 x 10^{-3} d^{-1}	300 d
Sediment	Kp_{sed} = 28.8 l/kg	2.31 x 10^{-4} d^{-1}	3000 d

3.1.3.2 Distribution

Table 3.7 summarises the results of studies described in section 3.1.3.2.

Table 3.7 Summary of results of distribution studies

Endpoint	Year test completed	Protocol cited	Results	Reliability	Study reference
Adsorption to soil[1]	2002	Method C.19 of 2001/59/EC	Log K_{oc} = 2.76±0.22	(1) valid without restriction[2]	Cuthbert and Mullee, 2002a

Notes: 1 – Test sample was a composite sample of purity 97.9% (total of the four isomers), derived from recent representative commercial products from the main producers.

2 – 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.

3.1.3.2.1 Adsorption

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

- Measured adsorption coefficient in 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.

Application of findings of OECD 106 study for a structurally-related substance

The K_{oc} of the structurally-related substance TDCP has been determined to be 1780 in a reliable study (Schaefer and Ponizovsky, 2006).

The K_{oc} of TDCP predicted using the TGD equation for phosphates is 950 and using the 'hydrophobics' equation is 1230. These are somewhat lower than the measured value, suggesting that TDCP is adsorbing to organic matter more strongly than predicted by these equations. The TGD methods are discussed in more detail below.

From the OECD 106 study on TDCP, a regression equation was derived from a plot of log K_d versus log OC (organic carbon concentration), in order to derive a K_{oc} from the whole data set. Further details are reported in the TDCP risk assessment report. 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 derived: log K_{oc} = -0.44 + log K_{ow}. It is assumed that this same relationship can be applied to TCPP. Applying the same relationship for TCPP (log K_{ow} = 2.68), gives the result log K_{oc} = 2.24, K_{oc} = 174. The basis of such an approach is the structural analogy between the substances, and is justified because the most reliable information in the whole data set is the measured K_{oc} of TDCP. The robustness of this approach is reviewed below.

For the substance TDCP it was found that the HPLC 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, possibly involving the phosphate group, had occurred; binding to the natural substrates in the OECD 106 test system was much lower than to the HPLC column substrate.

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 as expected, suggesting that adsorption to components other than organic carbon was not significant.

HPLC estimation method

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} = 576, log K_{oc} = 2.76±0.22. The ± value is the 95% confidence interval. It should be noted that the calibration substances were general substances, not related structurally to TCPP, 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.

QSAR methods from the TGD

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 TCPP is 2.68 ± 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} = 304.2 (range 202.7 – 456.7).

The HPLC-estimated K_{oc} value is somewhat higher than the predicted value from the TGD method. This is consistently true for this group of substances. Within the ESR assessment of other chloroalkyl phosphates (4th priority list; Rapporteur UK/Ireland and 2nd priority list; Rapporteur Germany) measured K_{oc} values exceed K_{oc} values calculated, in accordance with the TGD, on the basis of log K_{ow}, using the QSAR for phosphates. These values are summarised in **Table 3.8**. Estimates made using the hydrophobics equation are also provided for reference.

Table 3.8 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

The estimates from HPLC are consistently out of line with other approaches. Both the phosphates and hydrophobics equations predict statistically similar K_{oc} values for TCPP to the value derived using the OECD 106 measured value for TDCP. It is considered that the uncertainty in reading across from TDCP to TCPP is less than or similar to the uncertainty in

applying the QSAR methods, especially given the relatively low value of r^2 for the phosphates equation.

The value of K_{oc} = 174 is used in the risk assessment of TCPP.

The coefficients in **Table 3.9** are derived from this value, using default conversion factors.

Table 3.9 Adsorption coefficients used in the environmental risk assessment

Partition coefficient	Symbol	Values used
Organic carbon - water partition coefficient	K_{oc}	174 l/kg
Solids – water partition coefficient for soil	Kp_{soil}	3.48 l/kg
Solids – water partition coefficient for sediment	Kp_{sed}	8.7 l/kg
Solid – water partition coefficient for suspended matter	Kp_{susp}	17.4 l/kg
Soil - water partition coefficient	$K_{soil\text{-}water}$	5.42 m^3/m^3
Sediment – water partition coefficient	$K_{sed\text{-}water}$	5.15 m^3/m^3
Suspended matter - water partition coefficient	$K_{susp\text{-}water}$	5.25 m^3/m^3

3.1.3.2.2 Precipitation

The relatively low volatility and moderate solubility and adsorption coefficient suggest that most TCPP 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.

3.1.3.2.3 Volatilisation

A Henry's Law constant of 3.96 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.

3.1.3.2.4 Distribution in wastewater treatment plants

Based on the physico-chemical properties of TCPP (vapour pressure = 1.4 × 10^{-3} Pa, water solubility = 1080 mg/l, Henry's law constant = 3.96 x 10^{-4} Pa m^3/mole, K_{oc} = 174 l/kg), and the weight of evidence supporting a conclusion of inherent biodegradability (not meeting the criteria), 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	97.9%
Fraction to sludge	2.1%
Fraction degraded	0%

<u>Adaptation in industrial WWTP</u>

A number of inherent-type biodegradation studies were performed with TCPP. While not standard tests, the studies showed that degradation of the test substance started after an

acclimation period of 2 weeks or more, indicating possible adaptation of the activated sludge. At industrial sites where releases are made regularly to on-site wastewater treatment, an adapted microbial population may be maintained, and could result in a higher removal rate than estimated using the SIMPLETREAT model (though at present the risk assessment does not allow for any such acclimation).

3.1.3.2.5 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 196, which is sufficiently close to the value of 174 used in the assessment 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 3.10**.

Table 3.10 Environmental distribution of TCPP for various models

	EQC Level I	Level III
% in air	0.005	0.0015
% in soil	29.6	86.4
% in water	69.8	13.6
% in sediment	0.66	0.044

The results for EQC level I (the simplest model) indicate that water, soil and sediment are all significant should TCPP 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 3.11**.

Table 3.11 Output of fugacity model for various release scenarios

Release:	To air, water and soil	To air	To water	To soil
% in air	0.0015	0.0036	0	0
% in soil	86.4	90.5	0.034	90.9
% in water	13.6	9.46	99.6	9.09
% in sediment	0.044	0.03	0.32	0.029

The results reflect that most TCPP 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, for a substance of low volatility. In water, the modelled adsorption to sediment is very low.

3.1.3.3 Accumulation and metabolism

3.1.3.3.1 Aquatic organisms

Table 3.12 Bioaccumulation of TCPP

Endpoint		Year test completed	Protocol cited	Results	Reliability	Study reference
42 d	Bioaccumulation in fish (*Cyprinus carpio)*	1992	MITI (OECD 305C)	BCF 0.8 – 4.6 at two concentrations over 6 weeks	(2) valid with restrictions. Toxicity to carp was not established.	MITI undated

One measurement of bioconcentration is available (MITI, undated), summarised in **Table 3.12**. Test concentrations appear to be acceptable, being nominally 0.2 and 0.02 mg/l, 0.4 and 0.04% of the lowest LC_{50} for other species (see section 3.3.1.1.1). Fish were kept in flow-through conditions for 28 days prior to exposure to test substance. The exposure period was six weeks after which the concentration of TCPP in fish was determined (method not stated). BCFs of 0.8 – 2.8 and <1.9 – 4.6 were obtained for the two concentrations respectively. Bioconcentration is calculated as (concentration in fish)/(concentration in water).

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:

Log BCF_{fish} = 0.85 log K_{ow} – 0.70

The log K_{ow} for TCPP is 2.68 ± 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} = 37.8 (range 18.7 – 76.6).

The measured BCFs 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 possibility of rapid metabolism in the organism. There is evidence for mammalian metabolism of both TCPP (which is discussed in Section 4.1.2.1) and TDCP (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 2.7 l/kg is used in the risk assessment; this is the arithmetic mean of the range 0.8 to 4.6. Since the values are in a narrow range, a mean is considered acceptable and representative.

3.1.3.3.2 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 TCPP is 2.68 ± 0.36. On the basis of the uncertainty on this value, a range of BCF can be estimated. From the above equation, $BCF_{earthworm}$ = 6.58 (range 3.35 – 14.0).

3.1.4 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.

3.1.4.1 Calculation of predicted environmental concentrations (PEC_{local})

The PECs for TCPP 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 3.13**.

Table 3.13 Notes on the basis of PECs for specific life cycle stages

		Basis of release rates to the environment
	Producer 1	Site specific data
	Producer 2	Site specific data
	Producer 3	Site specific data
	Producer 4	Site specific data
A1a	Large systems houses	Site specific data; ESD for plastics additives
A2	Medium systems houses	ESD for plastics additives
A3	Small systems houses	ESD for plastics additives
A4	Systems houses using preformulated polyol	ESD for plastics additives
B1a	Flexible foam – furniture - very large sites	Site specific data; Appendix B
B1b	Flexible foam – furniture - large sites	Appendix B
B1c	Flexible foam – furniture - small sites	Appendix B
B1d	Flexible foam – furniture - small sites using systems	Appendix B
B2	Foam cutting	Appendix B
C1	Rigid foam - large sites	Appendix B
C2	Rigid foam - large sites	Appendix B
E1	One-component foams	ESD for plastics additives (compounding phase) Appendix B (foaming, in-service loss phases)
F1	CONFIDENTIAL	Estimates from relevant ESDs; read across from relevant previous published risk assessments; site specific info and WWTP details in some instances
G1	CONFIDENTIAL	
G2	CONFIDENTIAL	
H1	CONFIDENTIAL	
I1	CONFIDENTIAL	
J1	CONFIDENTIAL	
K1	CONFIDENTIAL	
K2	CONFIDENTIAL	
L1	CONFIDENTIAL	
M1	CONFIDENTIAL	
N1	CONFIDENTIAL	
P1	CONFIDENTIAL	
O1	Rebonding	Appendix B
Q1	Adhesive pressing	Read across from Appendix B
R1	Loose crumb	Appendix B

3.1.4.1.1 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 3.14**.

Table 3.14 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.kgwwt-1]
Producer 1	0.0641	6.41E-04	1.14E-03	5.21E-03
Producer 2	0.15	0.0103	0.0108	0.0492
Producer 3	0.0347	2.08E-05	5.20E-04	2.37E-03
Producer 4	0.0783	7.83E-04	1.28E-03	5.85E-03

3.1.4.1.2 Calculation of PEC_{local} for formulation

PEC_{local} for formulation of systems is based on the ESD for additives used in the plastics industry, with site specific, confidential details of effluent concentration and wastewater treatment plant size and function for large sites. Calculated PECs are summarised in **Table 3.15**.

Table 3.15 Values used in calculation of PEC for formulation

	$C local_{effluent}$ [mg.l-1]	$C local_{water}$ [mg.l-1]	PEC_{water} [mg.l-1]	$PEC_{sediment}$ [mg.kgwwt-1]
A1a: Large systems houses	0.597	5.32E-06	5.04E-04	2.30E-03
A2: Medium systems houses	0.408	0.0408	0.0413	0.188
A3: Small systems houses	0.102	0.0102	0.0107	0.049
A4: Systems houses using preformulated polyol	0.0122	1.22E-03	1.72E-03	7.86E-03

3.1.4.1.3 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 3.16**.

Table 3.16 Values used in calculation of PEC for industrial and professional use

	$Clocal_{effluent}$ [mg.l-1]	**$Clocal_{water}$ [mg.l-1]**	**PEC_{water} [mg.l-1]**	**$PEC_{sediment}$ [mg.kg wwt-1]**
B1a: flexible foam (furniture) very large	3.76E-03	3.76E-04	8.75E-04	3.99E-03
B1b: flexible foam (furniture) large	6.73E-04	6.73E-05	5.66E-04	2.59E-03
B1c: flexible foam (furniture) small - not using systems	6.93E-03	6.93E-04	1.19E-03	5.44E-03
B1d: flexible foam (furniture) small - users of systems	0.0124	1.24E-03	1.74E-03	7.93E-03
B2: flexible foam cutting	8.32E-04	8.32E-05	5.82E-04	2.66E-03
C1: rigid foaming large sites	1.25E-04	1.25E-05	5.12E-04	2.34E-03
C2: rigid foaming small sites	0.0533	5.33E-03	5.83E-03	0.0266
E1: one-component foams	0.514	0.0514	0.0519	0.237
F1: confidential	0.0783	7.83E-03	8.33E-03	0.038
G1: confidential	1.3	0.13	0.131	0.598
G2: confidential	1.22	0.122	0.123	0.561
H1: confidential	2.45	0.245	0.245	1.12
I1: confidential	0.122	0.0122	0.0127	0.0581
J1: confidential	0.621	0.0621	0.0626	0.286
K1: confidential	0.0813	8.12E-03	8.62E-03	0.0394
K2: confidential	0.488	0.0488	0.0493	0.225
L1: confidential	3.70E-03	3.69E-04	8.68E-04	3.96E-03
M1: confidential	0.0139	1.39E-03	1.89E-03	8.64E-03
N1: confidential	0.245	0.0245	0.025	0.114
O1: rebonding	0	0	4.99E-04	2.28E-03
P1: confidential	0.0404	4.04E-03	4.54E-03	0.0207
Q1: adhesive pressing	0.122	0.0122	0.0127	0.0581
R1: loose crumb	0	0	4.99E-04	2.28E-03

3.1.4.1.4 Calculation of PEC_{local} for private use

Not applicable. Non-industrial applications, in-service loss and waste remaining in the environment are characterised on a regional scale.

3.1.4.1.5 Calculation of PEC_{local} for disposal

Preliminary research suggests that local scale exposure is possible due to WWTP treatment of landfill leachate, however an example calculation suggests that no local scale risks would be anticipated (see section 3.1.2.8.1). The contribution of release via landfill leachate to the regional PEC has been accounted for in the risk assessment (see section 3.1.2.8.1).

3.1.4.2 Measured levels

All available data are summarised in **Table 3.36**.

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 TCPP can be detected in the environment.

3.1.4.2.1 Monitoring data provided by regulatory authorities in England and Wales

The Environment Agency WIMS database contains some data on the environmental concentration of TCPP in various media (EA 2001, pers. comm. 3rd August 2005 and pers. comm. 22nd December 2005). This information has been provided and comprises the following measurements, taken between 1995 and 1999 and between November 2003 and July 2005:

- A total of 220 measurements in fresh surface water
- 181 measurements in sewage final effluent
- 3 measurements in ground water (one is for an isomer of TCPP)
- 11 measurements in trade effluent.

Most of the data relate to the Midlands Region of Environment Agency responsibility. The sites are identified by grid reference and are mostly in the vicinity of the site previously owned by Courtaulds Acetate Ltd (now Acordis) near Derby, where TCPP used to be produced.

Fresh surface water in general contains less than 5 – 10 μg/l. The highest value is 304 μg/l. This is one of a few very high outliers which may possibly reflect the data having been recorded in incorrect units. These high values were measured at 'New Inlet Attenborough GP'. The more recent data include high values sampled in the river Severn, with a maximum of 150 μg/l. Values for other sampling locations (river Derwent) are <3 μg/l.

For purposes of risk assessment, a concentration in fresh surface water may represent a local or background concentration depending on whether it is up or down stream from a point of

effluent intake e.g. a sewage works. In almost all of the data provided, this important information is not given. However, for a handful of the data, this information was supplied separately by the Environment Agency. On the basis of these data, it is suggested that the regional background in the UK would be of the order of 0.56 µg/l. Concentrations downstream of a nearby wastewater treatment plant fit in with the general spread of values in freshwater.

In general, sewage final effluent contains less than 20 µg/l. Again there are some very high outliers, of which the highest recorded value is 3.32 mg/l recorded in the town of Kimberley on the river Erewash, near Nottingham. The more recent data are for two sites only; the TCPP concentrations are all below 10 µg/l.

The measurements for trade effluent showed concentrations of less than 2 µg/l.

The measurements for ground water were 199 ng/l, measured in open land north-west of Worcester, near the River Teme; a high value of 21 µg/l, measured in an unspecified location in the Grimsby area, is considered by the source to be invalid and should be discounted; and 0.56 µg/l, measured in an unspecified site of unknown location. The values were collected as part of a screening assessment. The reliability is not assignable.

Landfill leachate

As described in section 3.1.2.8.1, the Environment Agency of England and Wales has conducted some limited studies of the concentration of TCPP in leachate from 22 landfills in southern England and Wales. The data are presented in full in section 3.1.2.8.1.

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 TCPP (lower limit of quantitation 10 ng/g w/w for all matrices). TCPP was not detected in any samples.

3.1.4.2.2 Measured levels reported in the open literature

All measured data are summarised in **Table 3.36.**.

Measured levels in the EU

Water

TCPP has been measured in drinking water (Galassi, Guzzella and Sora, 1989). Samples of drinking waters were taken from three sites in northern Italy. Sampling strategy is not clear in the paper. Water taken from a public fountain (water originating from Lake Como) was found to be mutagenic in *S. typhimurium* and *S. cerevisiae*. "Higher than background" levels of TCPP (and TCEP) were found. Other contaminants were present.

Samples were analysed using GLC and HPLC. The levels found are summarised in **Table 3.17**.

Table 3.17 TCPP in drinking water

	Time period	Total DOC (mg/l)	TCPP (µg/l)
Turin Ia	9/86	0.39	0.02
I	9/86	0.41	<0.01
II	11/86	0.33	<0.01
III	2/87	3.57	<0.01
Ferrara Ia	9/86	0.60	<0.01
I	9/86	1.71	<0.01
II	11/86	0.39	<0.01
III	2/87	2.05	<0.01
Como Ia	9/86	1.09	0.08
I	9/86	1.74	0.09
II	11/86	0.79	0.03
III	2/87	1.27	0.02

a – raw water extracts

Water was extracted from the River Po at a site in Ferrara, at the closing section of the river basin (Guzzella and Galassi, 1993). Samples were taken from May 1988 – September 1989. A bacterial assay using the *Vibrio fischeri* photobacterium (also known as *Photobacterium phosphoreum*) was used to determine toxicity of the samples; chemical analysis was performed using GC with an N/P-selective detector. The detection limit for organophosphorus compounds in water was 1 ng/l. Number of tests per sample is not stated. The results are summarised in **Table 3.18**.

Table 3.18 Concentration of TCPP recorded (ng/l)

May '88	Sept	Nov	Jan '89	Feb	May	Jun	Jul	Aug	Sept
68	28	23	52	0	33	42	19	16	27

The report indicates that the origin of the pollutants is likely to be urban/industrial. This suggests that the measurement represents a local concentration.

A LUMIStox bacteriological assay was used. The micropollutants were removed from the water and redissolved.

Several pesticide and non-pesticide organophosphorus compounds were screened for in the river Po and shortly after its point of discharge into the Adriatic sea (Galassi, 1991). Water was sampled at three locations: station A on the Po, at Ferrara; B and C in the Adriatic sea some way from the coastline, presumably following the plume of Po river water. Samples were taken four times between April and August 1988. A detection limit of 10 ng/l applied for TCPP (yet two samples are quoted as being an exact value less than 10). The results are summarised in **Table3.19**.

Table 3.19 Measured levels of TCPP (ng/l)

20/4/88			18/5/88	16/6/88			2/8/88		
A	B	C	A	A	B	C	A	B	C
27	5	<10	68	92	<10	<10	64	31	9

The results do not imply a consistent rate of downstream dilution, but the levels of TCPP do decline with distance.

River water, effluents and sediments in the region of the Elbe were sampled between 1996-99 (Reincke *et al.*, 2000). Surface waters were examined for chloroalkyl phosphates in 1996 and 1998; suspended sediments were investigated in 1998-99 (sampling twice monthly) and at the end of 1999 there was a special monitoring programme for water, wastewater and sediments in the governmental districts of Leipzig and Halle.

In 1996, samples were taken six times over the year at seven sites. In 1998, samples were taken in February and in July, at ten sites. TCPP was detected in all samples at levels of 20-780 ng/l. The results are summarised in **Table 3.20**.

Table 3.20 TCPP concentration in surface water samples (Reincke *et al.*, 2000)

		1996 (ng/l)		1998 (ng/l)	
	Min	**Median**	**Max**	**Min**	**Max**
Schmilka	20	72	160		<25
Dommitzsch	-	-	-	<25	27
Gorsdf. (Schw. Elster)	-	-	-	33	720
Dessau (Mulde)	160	284	450	71	79
Rosenburg (Saale)	130	305	780	<25	140
Magdeburg	90	217	520	<25	80
Schnackenburg	120	197	310	<25	77
Bunthaus	-	-	-	<25	88
Seemannshöft	70	207	370	29	61
Grauerort	75	169	260	<25	68

The 1999 samples in Leipzig and Halle districts were taken in December of that year. The one-off results are summarised in **Table 3.21**.

Table 3.21 TCPP concentration in further surface water samples (Reincke *et al.*, 2000)

	TCPP concentration (ng/l)
Saale catchment area	
Faule Pfütze, uh. Klärteiche Gaulis	200
Neue Gösel, Gütepegel	31000
Pleiße, Gütepegel	1800
Neue Luppe, Gütepegel	900
Weiße Elster, Gütepegel	1300
Mulde catchment area	
Lober, Gütepegel	2400
Mulde, Gütepegel	100

Overall the highest concentration in surface water was 31 µg/l in surface water at Neue Gösel, Gütepegel in December 1999.

This paper also cites some other monitoring data, given in **Table 3.22**, that are new for this RAR. The cited references are not available for review.

Table 3.22 TCPP concentrations in river waters

Location	Year	Number of samples	Result type	Value
Rhein at Köln	1996	103	50%, max	190, 790 ng/l
Rhein at Köln	1998	90	50%, max	50, 160 ng/l
Rhein at Köln	1998	104	50%, max	80, 240 ng/l
15 Fließgewässer (Hessen)	1997-98	2	range	<100-700 ng/l
13 Fließgewässer (Me.-Vorp.)	1996	12	range	20-3670 ng/l

Twenty-nine bath lakes and 573 house wells in Mecklenburg-Vorpommern were sampled and analysed for TCPP in 1999 and 1997-98 respectively (Prösch *et al.*, 2002). Additionally, bath lakes were sampled (16 samples) during summer 2000. Analysis was by GC-FPD. The results are summarised in **Table 3.23**.

Table 3.23 TCPP concentrations in bath lakes and house wells

Bath lakes					
Of the 29 samples taken in 1999:					
	<0.02 µg/l	0.02-0.1 µg/l	>0.1 µg/l	Median (µg/l)	Max (µg/l)
Number	2	23	4	0.03	0.37
Of the 16 samples taken in 2000:					
	<0.02 µg/l	Min (µg/l)	Median (µg/l)	Max (µg/l)	
Number	0	0.03	0.04	0.05	
House wells					
Of the 573 samples taken in 1997-98:					
	<0.02 µg/l	0.02-0.1 µg/l	>0.1 µg/l	Max (µg/l)	
Number	560	7	6	1.0	

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.

River water of the Ruhr and its tributaries 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 and TCPP had a recovery rate of 101% and a limit of quantification of 4.9 ng/l. In river waters (Ruhr, Möhne, Lenne, and

other tributaries) the concentration of TCPP varied between a few ng/l up to ~300 ng/l. Samples of river water were also taken from the Rhine and Lippe rivers, for comparison with the above results. Analysis showed that TCPP was present at 80-100 ng/l and 100 ng/l respectively in Rhine and Lippe river waters.

Lake waters were sampled and analysed in three lakes in Italy in a study by Galassi *et al.* (1992). Samples were taken between 1986 and 1988 in the area of maximum depth of the lakes (Varese, Comabbio and Monate). It is noted that the watersheds of the three lakes are densely populated with a high density of industries in the area between lake Varese and Comabbio. Analysis of TCPP was by GC-MS, after extraction of the samples with hexane. The detection limit was 20 ng TCPP per 1 ml of hexane extract. TCPP was detected at 4.5 µg/l in lake Varese water, 17.8 µg/l in lake Comabbio water, and 0.04 µg/l in lake Monate water.

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

For TCPP, monitoring data are available from the internet-database Waterbase (V&W). The concentrations of TCPP at Amsterdam, Belfeld, Eemmeerdijk, Eijsden, Haringvlietsluit, IJmuiden, Lobith, Maassluis, Schaar van Ouden Doel, and Steenbergen in 2002 and 2003 were all lower than the detection limit of 5 µg/L. However, this limit seems rather high and lower values have indeed been reported. The average values are 1.93 µg/L in the effluents of STPs discharging into the river Meuse (**Table 3.24**) and 0.27 and 0.55 µg/L for two isomers in water of from the river Lek (**Table 3.25**). The 90$_{th}$ percentiles were 0.07 µg/L in the river Roer (tributary of the river Meuse) in 2002/2003 (**Table 3.24**) and 0.31 and 0.61 µg/L for two isomers in water of from the river Lek at Nieuwegein in 2002 (**Table 3.25**).

Table 3.24 Monitoring data for several phosphate ester in the river Meuse and tributaries and discharging effluents

Location	Date	Max [µg/L]	90$_{th}$ P [µg/L]	Avg [µg/L]	Med [µg/L]	Min [µg/L]
STP effluents (5) Meuse basin	12/2002-3/2003	4.2		1.93	1.57	0.11
Roer	3/2002-2/2003		0.07			

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 3.25 Monitoring data for several phosphate esters near the river Lek at Nieuwegein

Location	Date	Max [µg/L]	90$_{th}$ P [µg/L]	Avg [µg/L]	Med [µg/L]	Min [µg/L]
Nieuwegein	4/2002-6/2002	1.72	0.31	0.27	0.09	0.05
Nieuwegein	4/2002-6/2002	0.62	0.61	0.55	0.49	0.48

(data from RIWA (2003)); data represent two different isomers

TCPP 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 TCPP were reduced from 54 ng/l in the river Ruhr to 2.9 ng/l in the finished water at site A, 95 to 50 ng/l at site B, ca. 74 ng/l to ca. 4 ng/l at site C. Filtration with activated carbon was found to be the most effective treatment method for removal of TCPP and related substances.

Andresen *et al.* (2007) monitored for TCPP 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 90 ng TCPP/l was measured in the River Elbe (near the town of Stade).

WWTP and other effluents

TCPP was detected in various waters (Puchert and Prösch, undated). The time of sampling is not given.

Industrial outflows, receiving rivers and water-works waters were examined. The discussion is brief. Tris(chloroisopropyl)phosphate was one of three phosphates identified (by GC/MS and GC/FPD). No CAS number is given, but the substance is thought to be the TCPP which is the subject of this risk assessment. The results are summarised in **Table 3.26**.

Table 3.26 Levels of TCPP reported (ng/l)

Warnow	Nebel	Elbe	Wasserwerk 1 (direkte Entnahme)	Wasserwerk 2 (Uferfilträt)	Wasserwerk 3 (Uferfilträt)
280	830	280	3.7	2.6	<1

No validation data relating to the analytical recovery or storage conditions are presented.

Effluents from thirteen wastewater treatment plants in the Baltic Sea catchment area were analysed in a study by Prösch, Puchert and Gluschke (2000). These are municipal or industrial sites, as indicated in the table below. Effluents were sampled once monthly during 1998. Samples were analysed by GC and all isomers of TCPP are included in the results. TCPP was detected in all thirteen samples at average concentrations of 0.18 – 26.7 µg/l (note that there is significant variation in size and hence total annual emission of TCPP). The results are summarised in **Table 3.27**.

Table 3.27 TCPP concentrations in effluents (Prösch, Puchert and Gluschke, 2000)

			TCPP concentration in µg/l			TCPP releases in kg/year	
WWTP	**Throughput (m^3/year)**	**Industrial**	**Min**	**Median**	**Max**	**Min**	**Max**
Frankfurt/Oder	5 780 000		0.65	0.93	1.43	3.76	8.27
Eisenhüttenstadt	3 100 000	Y	6.11	14.19	26.72	18.94	82.83
Schwedt	2 070 000		0.61	0.87	1.38	1.26	2.86
Altfriedland	763 000		0.92	1.23	1.98	0.70	1.51
Rostock	16 200 000		0.81	1.21	1.88	13.12	30.46
Stralsund	6 260 000		0.96	1.25	1.93	6.01	12.08
Wismar	3 520 000		0.28	0.80	1.21	0.99	4.26
Güstrow	2 800 000		0.28	0.64	1.08	0.78	3.02
Görlitz	3 440 000		0.58	0.93	1.31	2.00	4.51
Zittau	3 480 000	Y	0.61	2.33	6.92	2.12	24.08
Rothenburg	226 000		0.68	1.03	1.40	0.15	0.32
Kiel	23 100 000		0.63	1.16	1.99	14.55	45.97
Osterby	50 000		0.18	0.42	0.80	0.01	0.04

Taking the maximum average concentrations and average throughputs, i.e. assuming a steady rate of release over the year, the largest release of TCPP would be from Eisenhüttenstadt, a total of approximately 83 kg/year. The smallest release is from Osterby, with a maximum average of 0.04 kg/year. The level of dilution and hence implications for PEC are not made clear. The mean effluent concentration suggests an influent concentration to the WWTP of around 1 µg/l. The $PEC_{regional}$ is 2.2 µg/l, a value not inconsistent with these findings.

Effluents in the region of the Elbe were sampled between 1996-99 (Reincke *et al.*, 2000). At the end of 1999 there was a special monitoring programme for wastewater in the governmental districts of Leipzig and Halle. Samples were taken in December 1999 in Leipzig and Halle districts. The one-off results were as follows. The highest concentration of TCPP was 74 µg/l in effluent from Kläranlage MUE GmbH. The results are summarised in **Table 3.28**.

Table 3.28 TCPP concentrations in effluents (Reincke *et al.*, 2000)

	TCPP concentration (ng/l)
Saale catchment area	
Kläranlage MUE GmbH	74000
Kläranlage Leipzig-Rosental	1300
Fabrikabwasser-Kanal, BUNA	8100
Kühl- und Regenwasser-Kanal 2, BUNA	200
Mulde catchment area	
Kläranlage Delitzsch	7300

20 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. Concentrations of TCPP in treated effluent were up to 2.3 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, TCPP was eliminated up to 21%. 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 9 µg/l TCPP were measured in treated effluent. Raw data are not presented. Median and maximum concentrations are shown in **Table 3.29**.

Table 3.29 TCPP concentrations in wastewater streams (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	1.49	1.01	
Effluent	12	12	0.01	1.74	0.92	9%
Köln						
Influent	12	12	0.01	12.9	3.5	
Effluent	12	12	0.01	9.0	3.5	0%

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 the Düsseldorf WWTP, TCPP was present at approximately 1.0 µg/l in influent, and approximately 0.9 µg/l in effluent. In the Köln WWTP, TCPP was present at approximately 3.4 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 3.30** and the results are summarised in **Table 3.31**.

Table 3.30 WWTP sampling locations (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 TCPP of 570-5800 ng/l in influent and 1700-6600 ng/l in effluent.

Table 3.31 TCPP concentrations in wastewater streams (Meyer and Bester, 2004)

	STP A (ng/l)	STP B (ng/l)
Influent	Max 5800 Mean 2000	Max 940 Mean 650
Intermediate settling tank / primary settling tank	Max 5900 Mean 2500	Max 780, Mean 950 [sic]
Final sedimentation tank	Max 4500 Mean 2600	Max 1400, Mean 820
Effluent	Max 6600 Mean 3000	Max 1100 Mean 820

There is no evidence of removal of either substance at either WWTP.

Other findings were that at STP A, the load of TCPP was discernibly lower at weekends than on weekdays. This suggests that this WWTP was receiving TCPP-containing effluent associated with some kind of industrial activity. In the absence of identification of the STP it is not possible to judge the significance of this information in the context of the risk assessment. 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 TCPP 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 TCPP concentrations of ca. 1 µg/l in influent and ca. 0.9 µg/l in effluent of Düsseldorf-Sud, and ca. 3.5 µg/l in both influent and effluent of Köln-Stammheim.

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 and TCPP had a recovery rate of 101% and a limit of quantification of 4.9 ng/l. In STP effluents, concentrations of ~20-~380 ng/l were analysed.

Influent and effluent water and sludge were sampled at a WWTP site located in a major city (Bester, 2005). TCPP was identified by GC-MS. The concentration in influent and effluent were 520 ng/l and 380 ng/l respectively (mean values). The concentrations of TCPP in the

wastewater inflow exhibited a high variability. Rates of elimination in the sewage treatment plant were also variable but were not high. Concentrations in sewage sludge of the same plant were also analysed: mean value 5100 ng/g dwt., 1700 ng/g wwt., equivalent to 5.1 mg/kg dwt and 1.7 mg/kg wwt respectively. For purposes of comparison, sludge samples from twenty other plants were analysed. In these samples, concentrations ranging from 1000-20 000 ng/g (dry wt.), equivalent to 1-20 mg/kg dwt, were detected; the average concentration was similar to the main WWTP site analysed. Authors calculated that 0.1% of TCPP sold in Germany reaches the sewage system, using a rather simplistic calculation. It is notable that one of the isomers of TCPP behaved similarly to the main isomer.

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 is presented as well as concentration data (for most sites data are available for single samples only). Analysis was by GC-NPD. For TCPP, the data presented represent the sum of three isomers detected. The data are presented in **Table 3.32**.

Table 3.32 TCPP 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	1800	2200 [1]	64
	4700	170			61
2	140900	5800	2900	1800	850
	140900	5800	1600	1700 [1]	610
3	46100	3500	3400	2400	1900
	46100	3500			1500
4	317500	13900	2800	1500	650
	317500	13900	1500	1600 [1]	840
5 [2]	500	-	1100	-	200
6	10300	790	18000	24000 [1]	1900
7	14900	770	2500	2300	790
	14900	770			1300
8	-	800			1200
	-	800			1300
9	-	240			250
10	-	14400			750
11	-	1900			700

Notes 1 – The authors noted the increases in TCPP concentration in effluent compared to influent in some cases. It was concluded that this was due to day-to-day variations in influent concentration, and that effluent concentrations could be expected to be more stable due to extensive mixing processes inside WWTP.

2 – no biological treatment at site 5.

Rodil *et al.* (2005) reported concentrations of TCPP 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. TCPP concentrations varied from 3.1 µg/l (raw wastewater), 2.4 µg/l (primary effluent) to 2.6 µg/l (tertiary effluent).

Two WWTPs in the Frankfurt area were sampled in a study reported by Höhne and Püttmann (2006). TCPP was among a number of flame retardants analysed. The maximum influent concentrations were 10.4 µg/l TCPP (Niederrad/Griesheim) and 4413 ng/l (Sindlingen); reducing to 6646 ng/l and 2634 ng/l respectively. Minimum and median concentrations suggest significant variability in levels of TCPP entering the Sindlingen plant as reported concentrations increase significantly in treated effluent (min. 333 ng/l increasing to 736 ng/l; median 1004 increasing to 1616 ng/l).

Rainwater and snow

Chloroalkyl phosphates were identified and determined as part of a larger study into the occurrence of chloro-organics in samples of rainwater and snow (Laniewski, Börenand and Grimvall, 1998). It had previously been reported that TCEP was found in rain and snow, and in this study TCEP was found, together with lower levels of other chloroalkyl phosphates. GC-MS analysis identified three isomers of tris(chloropropyl) phosphate, of which TCPP was one. TCPP was positively identified in snow in southern Sweden (rural area), Gdansk (Poland; densely built-up area), in glacial ice in northern Sweden, and in rainwater in Mace Head, Ireland (both remote areas). The maximum concentration of TCPP found was 3.0 ng/l but it is not stated which sample medium this referred to.

In snow samples collected in northern Sweden, TCPP concentrations dominated in the analysis (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. The authors reported that TCPP (sum of all isomers) showed a pattern of decrease in concentration with distance from the road intersection, and concluded that traffic could be a source of TCPP in outdoor environments, though since TCPP is not used in automotive applications this seems unlikely. Results are presented in **Table 3.33**.

Table 3.33 TCPP concentrations in snow (Marklund *et al.*, 2005a)

	Concentration (ng/kg snow)
Road 1	170
Road 2	130
Road 3	110
Airport 1	120
Airport 2	100
Airport 3	210

Sediment

Sediment from three lakes in northern Italy were sampled and analysed for various chemical classes of pollutants (Galassi, Provini and De Paolis, 1990). The number of samples taken reflects the size of the lake. Samples were taken in May 1986 and September 1987. 'TCPP'

(tris(3-chloropropyl)phosphate) was detected at levels of 0.600-1 and 0.3 µg/g dry weight among other phosphate esters at two of the lakes. A structure is not presented and it seems unlikely from the nomenclature that this is the TCPP that is the subject of this risk assessment.

The substance detected is likely to be present as a result of outflow from "point sources of pollution in the aquatic environments considered". The paper refers to an industrial site on the lake where it was detected at the higher level, so this may represent a local measurement. Detection was by GC with an N/P selective detector.

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, the intention being to obtain a pollution profile along the river. TCPP ('technical mixture') was one of nine FRs analysed for, and the tris-isopropyl structure (i.e. TCPP as described in section 1.1) and an isomer (Bis(1-chloro-2-propyl)-2-chloropropyl phosphate, though the structure depicted is incorrect) were analysed for separately. Analysis was by GC/MS. TCPP was detected in twenty samples (it is presumed that two samples were taken at each point though this is not stated; no FR occurs in more than 20 samples), at a concentration range of 15 – 540 micrograms/kg (mean 302 micrograms/kg).

Sediments in the region of the Elbe were sampled between 1996-99 (Reincke et al., 2000). Suspended sediments were investigated in 1998-99 (sampling twice monthly) and at the end of 1999 there was a special monitoring programme for sediments in the governmental districts of Leipzig and Halle. Suspended sediments were measured bi-monthly in 1998 and 1999, at ten and eleven sampling sites respectively. TCPP was detected in all samples at levels of 2-1100 µg/kg. The results are summarised in **Table 3.34**.

Table 3.34 TCPP concentrations in sediments from the river Elbe

	1998 (µg/kg)			1999 (µg/kg)		
	Min	**Median**	**Max**	**Min**	**Median**	**Max**
Schmilka	92	172	220	110	235	420
Dommitzsch	160	297	400	150	300	540
Gorsdf. (Schw. Elster)	140	312	420	22	319	480
Bad Düben (Mulde)	-	-	-	230	390	690
Dessau (Mulde)	290	527	1100	160	292	510
Rosenburg (Saale)	340	500	690	2	290	500
Magdeburg	350	432	520	280	408	690
Schnackenburg	89	212	340	27	158	250
Bunthaus	28	186	280	10	127	230
Seemannshöft	15	72	210	19	33	45
Grauerort	10	25	48	5	17	31

The 1999 samples in Leipzig and Halle districts were taken in December of that year. The one-off results are shown in Table 3.35.

Table 3.35 TCPP concentrations in sediments

	TCPP concentration (µg/kg)
Saale, at Meuschau	160
Saale, at Planena	20
Weiße Elster, at Ammendorf	38
Saale, at Trotha	350

Overall the highest concentration in surface water was 1100 µg/kg in Mulde sediments at Dessau in 1998.

Sediments were taken from the rivers Danube, Neckar and Rhine, as part of annual monitoring by the local environmental protection authority. The results were reported 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. High concentrations in the sediments of the three rivers (up to 1.3 mg/kg dry weight) are noteworthy, since this suggests accumulation.

A review of findings for many FRs (BAG/ERZ, 2000) notes that TCPP was found at up to 160 µg/kg in freshwater sediments (Lach and Steffen 1997).

Sediments were sampled and analysed in three lakes in Italy in a study by Galassi *et al.* (1992). Sediment core samples were taken between 1986 and 1988 in the area of maximum depth of the lakes (Varese, Comabbio and Monate). It is noted that the watersheds of the three lakes are densely populated with a high density of industries in the area between lake Varese and Comabbio. Analysis of TCPP was by GC-MS, after extraction of the samples with hexane. The detection limit was 20 ng TCPP per 1 ml of hexane extract. TCPP was detected at 0.30 µg/g dw in lake Varese sediment, 0.86 µg/g dw in lake Comabbio sediment, and not detected in lake Monate sediment. The two measurements are equivalent to 0.30 mg/kg and 0.86 mg/kg dwt respectively.

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 TCPP ranged between 5.9-311 µg/kg dwt, median 57 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. The chemical analyses were complemented by results of ecotoxicological studies with two sediment organisms (*Chironomus riparius* and *Potamopyrgus antipodarum*).

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 TCPP was below this level in the groundwater samples.

Measured levels in Asia

Water

TCPP was sampled in surface waters (Fukushima, Kawai and Yamaguchi, 1992). Monitoring data for organophosphoric acid triesters 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".

TCPP (tris(chloropropyl)phosphate) was found, but without structural representation or CAS number it is not confirmed that it is 13674-84-5. Samples were analysed for organophosphoric acid triesters using GC/MS and determined by GC with a flame photometric detector. Maps showing distribution of different levels are presented in the paper. Particularly high levels of TCPP (13.1 μg/l) were determined in the Yamato river. The nature of local industries in the areas surrounding the sampling sites is not set out.

Effluents

TCPP was detected in samples of effluents (Ishikawa *et al.*, 1985). Neither article nor abstract are translated from the Japanese. There is reference to TCPP but there is no full chemical name or diagram so it is not clear whether this is the TCPP that is the subject of this risk assessment. Factory effluent from food, chemical, steel, metal and 'others' industries were sampled. TCPP was detected at a level of 60 ng/l in the effluent from only one site ('other' industries). TCPP was also detected in four of 14 river waters, the maximum level being 180 ng/l.

In domestic miscellaneous effluent TCPP was not detected (i.e. <30 ng/l). In sewage treatment plant effluent, TCPP was detected at only one of six sampling sites, at which 980 ng/l was measured in the influent and 320 ng/l in the effluent (implying 67% removal, although any time delay between the measurements is not made clear). Any relationship between the different effluent release sites and the river water sampling sites is not made clear.

Samples were taken from degradation ponds at a sea-based disposal site (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 TCPP was approximately 70 μg/l. TCPP was relatively stable under all conditions over 78 days.

Measured levels in North America

Packaged foods

As part of a major study (Kan-Do, 1995), packaged foods were prepared according to the manufacturers' recommendations and then screened for the presence of around 300 different chemical substances (nutrients, toxic elements and pesticides) using established methods appropriate to the substance (not named). TCPP (not clearly identified as the substance that is the subject of this risk assessment) was found in three of the 234 food items investigated (raw peach and pear, and catsup), at an average level of 0.0093 μg/l.

Table 3.36 TCPP concentrations in the environment: Freshwater and related data

Sample type	Location	Sample period	Analytical method	Results	Scale represented	Reliability	Study reference
Drinking water and surface water	EU: Northern Italy	1986-87	GLC/HPLC	Max 0.09 μg/l	Unclear but probably regional	(4) not assignable. No validation of storage and analysis	Galassi, Guzzella and Sora, 1989
River water	EU: River Po at Ferrara	1988-89	GC	0 – 68 ng/l	Local	(2) valid with restrictions.	Guzzella and Galassi, 1993
River and sea water	EU: River Po and Adriatic	1988	GC	Max 92 ng/l	Sample point A likely to be local for private use stage: level of industrialisation is not known.	(4) not assignable. No validation of storage and analysis	Galassi, 1991
Surface waters and suspended sediments	EU: River Elbe and various other surface waters (max conc. in Neue Gösel, Gütepegel)	1996-99		ND – 780 ng/l (Elbe); ND – 31 μg/l (other waters)	Unclear but probably regional	(4) not assignable. No validation of storage and analysis	Reincke *et al.*, 2000
Rive r waters	EU: River Ruhr and its tributaries including Möhne and Lenne; also Rhine and Lippe	Sept 2002	GC-MS	Ruhr and tributaries: max ~300 ng/l Rhine 80-100 ng/l Lippe 100 ng/l	Unclear but probably regional	(4) not assignable. No validation of storage and analysis	Andresen *et al.*, 2004
River water and treated drinking water	EU: River Ruhr	Not clear	GC/MS	Concentrations in river water 54, 95 and ca. 74 ng/l Concentrations in treated drinking water 2.9, 50 and ca. 4 ng/l	Unknown without further information.	(4) not assignable.	Andresen and Bester, 2006
Fresh surface water	EU: UK Midlands region	1995-99, 2004-2005		Largely 5 – 10 μg/l. Highest value 304 μg/l	Not known	(2) valid with restrictions. Acceptable, though possible some data points may be in incorrect units	Environment Agency WIMS database

Sample type	Location	Sample period	Analytical method	Results	Scale represented	Reliability	Study reference
Fresh surface water	EU: UK Midlands region	1995-99		0.56 μg/l	Regional	(2) valid with restrictions. Acceptable, though possible some data points may be in incorrect units	Environment Agency WIMS database
Freshwater sediments	EU: England and Wales	2002 or earlier	LC-MS	Not detected (<10 μg/kg wwt)	Unclear	(2) valid with restrictions	CEFAS, 2002
Bath lakes and house wells	EU: Mecklenburg-Vorpommern	1997-99	GC-FPD	Bath lakes: max 0.37 μg/l House wells: max 1.0 μg/l	Unclear	(4) not assignable.	Prösch *et al.*, 2002
River/estuarine water	EU: R. Elbe estuary	May-June 2005	GC/MS	90 ng/l	Unclear	(4) not assignable.	Andresen *et al.* (2007)
WWTP receiving waters	EU: Germany	Not stated	GC/FPD, GC/MS	Max 830 ng/l	Local pre- and post-wastewater treatment.	(4) not assignable.	Puchert and Prösch, undated
WWTP receiving waters	EU: Germany	Not stated	Not stated	Trace concentrations	Local pre- and post-wastewater treatment.	(4) not assignable.	Kuch *et al.*, undated
Lake waters	EU: three Italian lakes	1986-88	GC/MS	Varese: 4.5 μg/l Comabbio: 17.8 μg/l Monate: 0.04 μg/l	Varese and Comabbio are local sites though the type of industry is not indicated in the report	(4) not assignable.	Galassi *et al.*, 1992
Sewage final effluent	EU: UK Midlands region	1995-99		Largely <20 μg/l. Highest value 3.32 mg/l	Local (though the sources of TCPP are not made clear, and cannot be linked to specific life cycle stages)	(2) valid with restrictions. Acceptable, though possible some data points may be in incorrect units	Environment Agency WIMS database

Sample type	Location	Sample period	Analytical method	Results	Scale represented	Reliability	Study reference
Municipal and industrial WWTP effluents	EU: Baltic Sea catchment area	1998	GC	Municipal sites: max 0.8-1.99 µg/l Industrial sites: max 6.9-26.7 µg/l	Local (though the sources of TCPP are not made clear, and cannot be linked to specific life cycle stages)	(4) not assignable.	Prösch, Puchert and Gluschke, 2000
WWTP effluents	EU: Elbe region and Leipzig and Halle districts	1996-99		Max 74 µg/l	Local (though the sources of TCPP are not made clear, and cannot be linked to specific life cycle stages)	(4) not assignable.	Reincke *et al.*, 2000
WWTP and disposal site effluents	EU: Germany			Treated effluent: max 2.3 µg/l Disposal site effluent: in mg/l range	Local (though the sources of TCPP 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 9 µg/l	Local (though the sources of TCPP are not made clear, and cannot be linked to specific life cycle stages)	(4) not assignable.	NRW, 2003
WWTP effluents	EU: Germany			Treated effluent: 0.9-3.4 µg/l	Local (though the sources of TCPP are not made clear, and cannot be linked to specific life cycle stages)	(4) not assignable.	Fahlenkamp *et al.*, 2004
WWTP effluents	EU: North Rhine-Westphalia	Spring 2003		Treated effluent: 1.7 – 6.6 µg/l	Local (though the sources of TCPP 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-~380 ng/l	Local (though the sources of TCPP are not made clear, and cannot be linked to specific life cycle stages)	(4) not assignable.	Andresen *et al.*, 2004

Sample type	Location	Sample period	Analytical method	Results	Scale represented	Reliability	Study reference
WWTP effluents	EU: Germany	Not clear	Not clear	Düsseldorf-Sud ca. 1 µg/l in influent and ca. 0.9 µg/l in effluent Köln-Stammheim ca. 3.5 µg/l in both influent and effluent	Unknown	(4) not assignable	Friedrich *et al*. (2005)
WWTP effluents	Not clear	2004	LC-ESI-MS/MS	3.1 µg/l (raw wastewater), 2.4 µg/l (primary effluent) 2.6 µg/l (tertiary effluent)	Unknown	(4) not assignable	Rodil *et al.* (2005)
WWTP effluents	EU: Germany (Frankfurt area)	Not clear	Not clear	Niederrad/Griesheim: Max 10.4 µg/l (influent) Max 6646 ng/l (effluent) Sindlingen Max 4413 ng/l (influent) Max 2634 ng/l (effluent)	Unknown	(4) not assignable	Höhne and Püttmann (2006)
Trade effluent	EU: UK Midlands region	1995-99		<2 µg/l.	Unknown	(2) valid with restrictions. Acceptable, though possible some data points may be in incorrect units	Environment Agency WIMS database

Sample type	Location	Sample period	Analytical method	Results	Scale represented	Reliability	Study reference
WWTP effluent and sludge	EU: Dortmund, Germany	April 2002	GC-MS	WWTP influent: 520 ng/l Effluent: 380 ng/l (means) Sludge: 5.1 mg/kg dwt 20 other sludge samples: 1-20 mg/kg dwt	Local (though the sources of TCPP are not made clear, and cannot be linked to specific life cycle stages)	(4) not assignable.	Bester, 2005
WWTP effluents and sludges	EU: Swedish WWTPs	2003	GC-NPD	1100 – 18,000 ng/l measured in influent wastewater 1500 – 24,000 ng/l measured in treated wastewater 61 – 1900 ng/g dw measured in sludge	Local (though the sources of TCPP are not made clear, and cannot be linked to specific life cycle stages)	(4) not assignable.	Marklund *et al.*, 2005b
Landfill leachate	EU: UK (Environment Agency Thames, Anglian and Wales Regions)	2005	Not stated	21 sites with analysis for TCPP: range of results 0.4 - 66.6 µg/l; mean 24.6 µg/l	Local	(2) valid with restrictions	Pers. comm., 3rd August 2005
Ground water	EU: UK	1995-2005		56 ng/l 199 ng/l	Unknown	(4) not assignable. Acceptable, though possible some data points may be in incorrect units	Environment Agency WIMS database and pers. Comm. 22nd December 2005
Ground waters	EU: Germany			Trace concentrations		(4) not assignable.	Kuch *et al.*, undated
Rainwater and snow	EU: Sweden, Poland and Ireland	1996-97	GC/AED and GC/MS/ SIM	Max 3.0 ng/l	Gdansk likely to be local, others are regional	(4) not assignable.	Laniewski, Börenand and Grimvall, 1998

Sample type	Location	Sample period	Analytical method	Results	Scale represented	Reliability	Study reference
Snow	EU: Northern Sweden	March 2003	GC-NPD and GC/MS	Near road intersection: 110-170 ng/kg snow Airport: 100-210 ng/kg snow	Unclear	(4) not assignable	Marklund *et al.*, 2005a
Lake sediments	EU: Northern Italy	1986-87	GC	0.600-1 and 0.3 μg/g dry weight, but may not be the relevant substance	Unclear	(4) not assignable.	Galassi, Provini and De Paolis, 1990
River sediments	EU: River Elbe	Jan-Feb 2001	GC/MS	Max 540 μg/kg, mean 302 μg/kg		(4) not assignable	Heemken, Kuballa and Stachel, undated
Suspended sediments	EU: River Elbe region and Leipzig and Halle districts	1996-99		Elbe region: 2-1100 μg/kg Leipzig and Halle districts: 20-350 μg/kg	Unclear	(4) not assignable.	Reincke *et al.*, 2000
River sediments	EU: Danube, Neckar and Rhine			Max 1.3 mg/kg dry weight	Unclear	(4) not assignable.	Kuch *et al.*, undated
Freshwater sediments	Unclear			Max 160 μg/kg	Unclear	(4) not assignable.	Lach and Steffen 1997, in BAG/ERZ, 2000
Lake sediments	EU: three Italian lakes	1986-88	GC/MS	Varese: 0.30 mg/kg dwt Comabbio: 0.86 mg/kg dwt Monate: ND	Varese and Comabbio are local sites though the type of industry is not indicated in the report	(4) not assignable.	Galassi *et al.*, 1992
River sediments	EU: River Elbe and tributaries	2002	GC-FPD	5.9-311 μg/kg dwt, median 57 μg/kg dwt	Presumably local	(4) not assignable	Stachel *et al.*, 2005

Sample type	Location	Sample period	Analytical method	Results	Scale represented	Reliability	Study reference
River water	Asia: Various rivers, Japan	1976-90	GC/MS and GC/FPD	<13.1 μg/l	Maximum concentration is probably downstream from a facility but this is not explicitly stated.	(2) valid with restrictions	Fukushima, Kawai and Yamaguchi, 1992
Effluents and river water	Asia: Kitakyushu City, Japan	Unknown	Unknown	Max 980 ng/l (sewage treatment influent)	Local (factory effluents) and unclear (other samples)	(4) not assignable.	Ishikawa *et al*, 1985
Degradation ponds at sea-based disposal site	Asia: Japan			approximately 70 μg/l	Presumably represents local environment for disposal	(4) not assignable.	Kawagoshi *et al.*, 2002
Packaged foods	North America: USA	1982-91		0.0093 μg/l	N/A	(4) not assignable	Kan-Do, 1995

3.1.4.3 Comparison between predicted and measured levels

UK monitoring data provided by the Environment Agency result in a regional background concentration of 0.56 µg/l in water, which compares well with the modelled value of 0.50 µg/l. UK data are particularly relevant, since the largest volume of TCPP-containing furniture foam is believed to be in service in the UK and Ireland.

The existence of EU measurements of comparable magnitude to the modelled $PEC_{regional}$ value of 0.50 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.

It is notable that the data suggest that TCPP is detectable in a wide range of non-industrial indoor environments. This supports the modelling of releases by volatilisation in service and from waste remaining in the environment.

The finding from studies of WWTP effluents that removal of TCPP in treatment plants was not significant supports the SimpleTreat model, which estimates that over 90% of the substance would be directed to water in a biological treatment plant.

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.

3.1.5 Terrestrial compartment

3.1.5.1 Calculation of PEC_{local}

The most significant contribution to $PEClocal_{, soil}$ comes from spreading of WWTP sludge onto agricultural land. The PECs for TCPP 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.

3.1.5.1.1 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 3.37**.

Table 3.37 PECsoil values 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	5.75E-03	5.75E-03	5.75E-03
Producer 2	5.75E-03	5.75E-03	5.75E-03
Producer 3	5.75E-03	5.75E-03	5.75E-03
Producer 4	0.0153	0.0136	8.53E-03

3.1.5.1.2 Calculation of PEC_{local} for formulation

PEC_{local} for formulation of systems is based on the ESD for additives used in the plastics industry, with site specific, confidential details of effluent concentration and wastewater treatment plant size and function for large sites. Calculated PECs are given in **Table 3.38**.

Table 3.38 PECsoil values for formulation

	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: Large systems houses	0.0825	0.0695	0.0335
A2: Medium systems houses	0.0564	0.0475	0.0217
A3: Small systems houses	0.0185	0.0162	9.74E-03
A4: Systems houses using preformulated polyol	7.26E-03	6.99E-03	6.21E-03

3.1.5.1.3 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 given in **Table 3.39**.

3.1.5.1.4 Calculation of PEC_{local} for private use

Not applicable. Non-industrial applications, in-service loss and waste remaining in the environment are characterised on a regional scale.

3.1.5.1.5 Calculation of PEC_{local} for disposal

Not included in the present assessment, though preliminary research suggests that low levels of 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.

3.1.5.2 Measured levels

No data are available for review.

3.1.5.3 Comparison between predicted and measured levels

No data are available for review.

Table 3.39 PECsoil values 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.)
B1a: flexible foam (furniture) very large	6.21E-03	6.13E-03	5.89E-03
B1b: flexible foam (furniture) large	5.83E-03	5.81E-03	5.77E-03
B1c: flexible foam (furniture) small - not using systems	6.59E-03	6.44E-03	5.99E-03
B1d: flexible foam (furniture) small - users of systems	7.26E-03	6.99E-03	6.19E-03
B2: flexible foam cutting	5.85E-03	5.83E-03	5.78E-03
C1: rigid foaming large sites	5.76E-03	5.76E-03	5.75E-03
C2: rigid foaming small sites	0.0123	0.0111	7.64E-03
E1: one-component foams	0.0693	0.0581	0.0253
F1: confidential	0.0154	0.0137	8.74E-03
G1: confidential	0.165	0.137	0.0523
G2: confidential	0.155	0.129	0.0493
H1: confidential	0.305	0.251	0.0928
I1: confidential	0.0208	0.0181	0.0102
J1: confidential	0.0824	0.0688	0.0291
K1: confidential	0.0159	0.0141	9.06E-03
K2: confidential	0.0654	0.0547	0.0231
L1: confidential	6.20E-03	6.12E-03	5.88E-03
M1: confidential	7.48E-03	7.17E-03	6.29E-03
N1: confidential	0.0357	0.0303	0.0144
O1: rebonding	5.75E-03	5.75E-03	5.75E-03
P1: confidential	0.0108	9.88E-03	7.32E-03
Q1: adhesive pressing	0.0208	0.0181	0.0102
R1: loose crumb	5.75E-03	5.75E-03	5.75E-03

3.1.6 Atmosphere

Given the low levels of releases, the relatively low volatility and moderate solubility and adsorption coefficient of TCPP, 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 TCPP in the atmosphere have been estimated using EUSES 2.0.3. The predicted local and regional atmospheric concentrations are shown in **Table 3.40**.

Table 3.40 Estimated air concentrations of TCPP

Scenario	Air concentrations (C_{local}) (mg/m³)		$PEC_{local(air),\ ann}$ (mg/m³)
	Emission episode	Annual average	
Producer 1	2.50E-09	2.06E-09	1.42E-07
Producer 2	4.73E-07	3.89E-07	5.29E-07
Producer 3	0	0	1.40E-07
Producer 4	3.06E-08	1.78E-08	1.58E-07
A1a: Large systems houses	1.07E-03	8.76E-04	8.76E-04
A2: Medium systems houses	2.32E-04	1.90E-04	1.91E-04
A3: Small systems houses	5.82E-05	4.78E-05	4.79E-05
A4: Systems houses using preformulated polyol	6.95E-06	3.90E-06	4.04E-06
B1a: flexible foam (furniture) very large	2.14E-06	1.75E-06	1.90E-06
B1b: flexible foam (furniture) large	3.83E-07	3.14E-07	4.55E-07
B1c: flexible foam (furniture) small - not using systems	4.67E-08	3.84E-08	1.79E-07
B1d: flexible foam (furniture) small - users of systems	8.34E-08	6.85E-08	2.09E-07
B2: flexible foam cutting	4.73E-07	3.88E-07	5.29E-07
C1: rigid foaming large sites	7.11E-08	5.85E-08	1.99E-07
C2: rigid foaming small sites	1.45E-08	1.19E-08	1.52E-07
E1: one-component foams	2.09E-04	1.71E-04	1.72E-04
F1: confidential	4.17E-05	2.86E-05	2.87E-05
G1: confidential	9.27E-05	1.52E-05	1.54E-05
G2: confidential	6.95E-06	6.85E-07	8.26E-07
H1: confidential	1.39E-05	7.62E-07	9.02E-07
I1: confidential	6.95E-05	1.52E-05	1.54E-05
J1: confidential	3.50E-04	1.63E-04	1.63E-04
K1: confidential	2.31E-04	5.69E-05	5.71E-05
K2: confidential	3.47E-07	6.46E-08	2.05E-07
L1: confidential	2.49E-08	4.57E-09	1.45E-07
M1: confidential	7.65E-06	6.28E-06	6.42E-06
N1: confidential	1.08E-09	1.18E-10	1.40E-07
O1: rebonding	1.31E-06	1.08E-06	1.22E-06
P1: confidential	2.29E-05	1.89E-05	1.90E-05
Q1: adhesive pressing	6.95E-05	1.83E-05	1.84E-05
R1: loose crumb	5.61E-07	4.61E-07	6.02E-07

Some monitoring data for indoor air and environments have been obtained and these are presented in section 3.1.6.1 below. 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.

3.1.6.1 Measured levels reported in the open literature

The following measured data relate to indoor environments. All results are summarised in **Table 3.47**.

Measured levels in the EU

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.

TCPP was detected in indoor air at levels of up to 261 ng/m^3. The levels of TCPP were not found to be correlated with dust levels, although it would be expected that these substances would be found mostly in particle-bound form. It is of interest to note that this concentration represents 0.13% of saturation (based on the vapour pressure).

Settled dusts were collected from 15 environments including workplaces, domestic and public buildings in a recent study (Marklund *et al.*, 2003). 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. TCPP was detected at the concentrations shown in **Table 3.41**.

These findings are very interesting. The highest levels of TCPP were detected in office, university lobby, hotel, prison and hospital office (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). TCPP was found at significant concentration on the surface of computer screen/casing. It is unclear how this could have arisen as TCPP 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, and it is possible that statutory requirements may exist requiring higher levels of flame retardancy in some specific types of location, such as prisons. 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 TCPP 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 TCPP 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).

Table 3.41 TCPP concentrations in settled dusts (Marklund *et al.*, 2003)

	TCPP[a] **mg/kg dust or ng/m² for computer screen and computer cover**
Home 1[b]	0.47
Home 2	0.93
Day care centre	2.5
Hospital wards[b]	2.3
Hospital office[c]	5.3
Radio shop[c]	2.3
Textile shop	1.4
Hotel[b]	8.9
Prison[c]	8.9
University lobby	50
Office[c]	73
Library[b]	2.9
Aircraft[c]	2.2
Cinema[c]	2.4
Public dance hall[c]	1.5
Computer screen	370
Computer cover	220

Notes
[a] Sum of isomers.
[b] Average of three replicates.
[c] Average of two parallel samples.

Indoor air has been sampled in similar 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 3.42**.

Table 3.42 TCPP concentrations in indoor air (Marklund *et al*., 2005c)

	TCPP (ng/m^3) (sum of three isomers)
Home 1	210
Home 2	38
Day care centre	28
Hospital ward	69
Radio shop	10
Textile shop	32
Hotel	69
Prison	570
University lobby	440
Office	160
Library	40
Public dance hall	97
Furniture store	73
Plastics Factory 1	32
Plastics Factory 2	27
Bowling alley	93
Laboratory	31
Blank (n = 3)	5.1

In a recent study (Prösch and Puchert, 2003), cotton pieces were exposed to indoor air *in situ* e.g. in cars and rooms, then washed. Levels of up to 1400 ng TCPP were extracted from the wash water. Flats and houses, old and new-built, were included in the work. TCPP-containing materials were present (e.g. in the installation of windows, building foam and fixing foam around door frames). Automotive interiors were also included, though this is not believed to be a relevant application for TCPP foam.

The exposure period was at least one week. Pieces of 100% cotton cloth 8 x 8 cm (i.e. 64 cm^2) were used to take samples. Prior to use, these were soaked for 1 hour in acetone. The cloths were stored before and after exposure in sealed glass containers. After exposure the cloths were washed in the laboratory with vibration at 40ºC for 30 minutes. The cloths were dried carefully and the cooled wash-water analysed using solid phase extraction GC/flame photometer.

Results were as shown in **Table 3.43**.

Table 3.43 TCPP concentrations sampled from indoor air

	TCPP in ng/cloth				
	Room 1	**Room 2**	**Room 3**		
Dwelling 1	~20	~150	~300	Vehicle 1	~250
Dwelling 2	~100	~450	~900	Vehicle 2	1400
Dwelling 3	~100	~250	~930	Vehicle 3	~40
Dwelling 4	~20	~30	~30	Vehicle 4	<10
Dwelling 5	~40	~50	~80	Vehicle 5	~10
Dwelling 6	~30	~430	-	Vehicle 6	~10

Settled and suspended dusts were collected as part of 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. It also reports some findings from previous studies; TCPP was detected in the samples summarised in **Table 3.44**.

Table 3.44 Reported TCPP concentrations in dusts

TCPP in 436 house dusts (Ingerowski *et al*., 2001)	<0.1-375 mg/kg dust 95th Percentile 3.4 mg/kg
Organophosphate FR in dusts from a kindergarten with organophosphate-containing building materials	44 mg/kg dust
Organophosphate FR in dwellings in Munich (Carl, 1998)	0.4-25 mg/kg dust
Organophosphate FR in dusts from buildings with Organophosphate FR building materials (Hansen *et al*., 2001)	Not detected

A review of findings for many FRs (BAG/ERZ, 2000) notes that organophosphate esters were detected in indoor air in schools and offices (Carlsson *et al*. 1997).

Indoor air was sampled at twelve locations around Zurich (Hartmann *et al.*, 2004): car interiors, a theatre, two furniture stores, three offices and three electronics stores. 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 TCPP (no reason is given). The limits of detection and quantification are 0.12 and 1.2 ng/m^3 respectively for TCPP.

TCPP was detected at 260 ng/m^3 in the 9-year-old car (undisturbed sample; with 'occupation' – with people entering and leaving the car every 30 minutes, the concentration was 190 ng/m^3). Lower levels (23 ng/m^3) were analysed in the new car and TCPP was below the detection limit in the 1-year-old car. Both furniture stores, the theatre and one of the offices gave levels of TCPP ranging from 46 to 130 ng/m^3. TCPP was not detected in samples taken at any of the electronics stores or the other offices.

In the 9-year-old car, variation of TCPP concentration was within analytical uncertainty between the samples with high and low dust concentrations.

Five indoor environments were sampled and analysed in a study by Carlsson *et al.* (1997). Indoor air was sampled at three school buildings, a day-care centre and an office. Three separate TCPP isomers were detected but it was not possible to link the concentrations found with a specific isomer structure. Samples were analysed using GC-NPD, GC-AED, and GC/MS. The limit of detection of TCPP was 5 pg. Results are summarised in **Table 3.45**.

Table 3.45 Concentrations of TCPP isomers in indoor air (Carlsson *et al.*, 1997)

	Concentrations(ng/lm^3; mean values)				
	School 1	**School 2**	**School 3**	**Day Care Centre**	**Office**
'TCPP 1'	14	41	35	34	31
'TCPP 2'	5.1	15	12	16	12
'TCPP 3'	<0.5	1.5	1.1	2.9	1.4
Total [1]	19.1	57.5	48.1	52.9	44.4

Note: 1 – Total of all TCPP isomers calculated by the Rapporteur, not taken from the published paper

Another study investigated air concentrations of TCPP 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 TCPP are summarised in **Table 3.46**. 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.

It is surprising that TCPP was detectable in any instance, since it is known that TCPP tends not to be used in flexible PUR for automotive applications, owing to its volatility and fogging potential.

Table 3.46 Summary results of Wensing *et al.* (2004)

Vehicle	**Old**		**New (all vehicles)**		**New (single vehicle)**			
Temp (°C)	48[1]	20[2]	20[1]	65[1]	65[1]	65[2]	50[2]	40[2]
TCPP (µg/m^3)	1.7	< 0.2	< 0.01 – 0.48	0.07 – 11.1	0.60	< 0.39	< 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 TCPP among various organophosphate compounds in 29 indoor environments. TCPP concentrations ranged from 7-160 ng/m^3 in ten private homes; 5-2300 ng/m^3 in seven transport vehicles; 41-120 ng/m^3 in three offices; 12-22 ng/m^3 in three workshops; 1-96 ng/m^3 in four shops and 26-140 ng/m^3 in three healthcare facilities.

Table 3.47 TCPP concentrations in air and indoor environments

Sample type	Location	Sample period	Analytical method	Results	Scale represented	Reliability	Study reference
Indoor environments	EU: Indoor air			Max 261 ng/m^3 in indoor air	Presumably represents local environment for in-service loss	(4) not assignable.	Bürgi, 2002
Indoor air	EU: Indoor air		solid phase extraction GC/flame photometer	Dwellings: 20-450 ng/sample Vehicles: <10-1400 ng/sample	Dwellings presumably represent local environment for in-service loss TCPP not used in automotive applications;	(4) not assignable.	Prösch and Puchert, 2003
Indoor air				Detected			Carlsson, Nilsson *et al*. 1997 in BAG/ERZ, 2000
Indoor air	Europe: Zurich		GC/MS	Vehicles: <LOD – 260 ng/m^3 Buildings: 46 to 130 ng/m^3	Buildings presumably represent local environment for in-service loss TCPP is not used in automotive applications.	(4) not assignable.	Hartmann *et al.*, 2004
Indoor air	Europe: Sweden		GC-NPD, GC-AED and GC/MS	Totals across TCPP isomers (means): Schools max 57.5 ng/m^3 Day care centre 52.9 ng/m^3 Office 44.4 ng/m^3	Buildings presumably represent local environment for in-service loss	(4) not assignable.	Carlsson *et al.*, 1997

Sample type	Location	Sample period	Analytical method	Results	Scale represented	Reliability	Study reference
Indoor air	Europe: Sweden		GC-NPD	10-570 ng/m^3 Concentrations above 100 ng/m^3 seen in Home 1, Prison, University Lobby and Office (210, 570, 440 and 1600 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	1-2300 ng/m^3 in a range of indoor environments. Concentrations above 100 ng/m^3 seen in private homes, offices, transport vehicles, and healthcare facilities.	Buildings presumably represent local environment for in-service loss	(4) not assignable.	Staaf and Östmann (2005)
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	ND – 3.4 mg/kg dust (95%ile)	Presumably represents local environment for in-service loss	(4) not assignable.	Various, in Nagorka and Ullrich, 2003

3.1.7 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 3.48 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.

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

PEC$regional_{(water)}$ = 4.99E-04 mg/l from the EUSES v2.03 model.

PEC$regional_{(freshwater\ sediment)}$ = 2.42E-03 mg/kg wwt from the EUSES v2.03 model.

PEC $regional_{(soil)}$ = 2.65E-03 mg/kg wwt from the EUSES v2.03 model.

PEC$continental_{(water)}$ = 1.27E-05 mg/l from the EUSES v2.03 model.

PEC$continental_{(freshwater\ sediment)}$ = 6.17E-05 mg/kg wwt from the EUSES v2.03 model.

PEC $continental_{(soil)}$ = 1.19E-05 mg/kg wwt from the EUSES v2.03 model.

Table 3.48 PECs for secondary poisoning assessment

	$PEC_{oral,\ predator,\ fish}$ [mg.kgwwt-1]	$PEC_{oral,\ predator,\ earthworm}$ [mg.kg-1]
Producer 1	2.06E-03	8.22E-03
Producer 2	0.0128	8.22E-03
Producer 3	1.37E-03	8.22E-03
Producer 4	1.96E-03	0.0159
A1a: Large systems houses	1.35E-03	0.0706
A2: Medium systems houses	0.0466	0.0491
A3: Small systems houses	0.0127	0.0185
A4: Systems houses using preformulated polyol	2.27E-03	9.43E-03
B1a: flexible foam (furniture) very large	1.76E-03	8.59E-03
B1b: flexible foam (furniture) large	1.42E-03	8.28E-03
B1c: flexible foam (furniture) small - not using systems	2.12E-03	8.90E-03
B1d: flexible foam (furniture) small – users of systems	2.72E-03	9.43E-03
B2: flexible foam cutting	1.44E-03	8.30E-03
C1: rigid foaming large sites	1.36E-03	8.23E-03
C2: rigid foaming small sites	7.26E-03	0.0134
E1: one-component foams	0.0583	0.0594
F1: confidential	8.59E-03	0.016
G1: confidential	0.0303	0.136
G2: confidential	0.0176	0.128
H1: confidential	0.0194	0.248
I1: confidential	4.97E-03	0.0203
J1: confidential	0.0404	0.0699
K1: confidential	4.05E-03	0.0164
K2: confidential	0.0136	0.0561
L1: confidential	1.44E-03	8.58E-03
M1: confidential	2.89E-03	9.61E-03
N1: confidential	4.97E-03	0.0322
O1: rebonding	1.35E-03	8.22E-03
P1: confidential	5.83E-03	0.0123
Q1: adhesive pressing	5.69E-03	0.0203
R1: loose crumb	1.35E-03	8.22E-03

3.2 MARINE EXPOSURE ASSESSMENT

3.2.1 General discussion

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

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

3.2.2 Degradation

TCPP is considered inherently biodegradable on the basis of several non-standard freshwater tests, therefore a mineralisation half-life of 150 days can be assumed for the marine environment.

3.2.3 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.

3.2.3.1 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 3.49**.

Table 3.49 Marine PEC for production

	$PEC_{sea\ water}$ [mg.l-1]	$PEC_{marine\ sediment}$ [mg.kgwwt-1]
Producer 1	6.90E-04	3.15E-03
Producer 2	1.55E-03	7.08E-03
Producer 3	6.93E-05	3.16E-04
Producer 4	8.48E-04	3.87E-03

3.2.3.2 Calculation of PEClocal for formulation

PEC_{local} for formulation of systems is based on the ESD for additives used in the plastics industry, with site specific, confidential details of effluent concentration and wastewater treatment plant size and function for large sites. Calculated PECs are summarised in **Table 3.50**.

Table 3.50 Marine PEC for formulation

	$PEC_{sea\ water}$ [mg.l-1]	$PEC_{marine\ sediment}$ [mg.kgwwt-1]
A1a: Large systems houses	5.40E-05	2.46E-04
A2: Medium systems houses	4.21E-03	0.0192
A3: Small systems houses	1.09E-03	5.00E-03
A4: Systems houses using preformulated polyol	1.73E-04	7.92E-04

3.2.3.3 Calculation of PEClocal for industrial/professional use

PEC_{local} values for industrial and professional use is calculated for all life cycle stages. Calculated PECs are summarised in **Table 3.51**.

Table 3.51 Marine PEC for industrial and professional use

	$PEC_{sea\ water}$ [mg.l-1]	$PEC_{marine\ sediment}$ [mg.kgwwt-1]
B1a: flexible foam (furniture) very large	8.69E-05	3.97E-04
B1b: flexible foam (furniture) large	5.54E-05	2.53E-04
B1c: flexible foam (furniture) small - not using systems	1.19E-04	5.45E-04
B1d: flexible foam (furniture) small - users of systems	1.75E-04	7.99E-04
B2: flexible foam cutting	5.70E-05	2.60E-04
C1: rigid foaming large sites	4.98E-05	2.27E-04
C2: rigid foaming small sites	5.93E-04	2.71E-03
E1: one-component foams	5.30E-03	0.0242
F1: confidential	8.48E-04	3.87E-03
G1: confidential	0.0134	0.0611
G2: confidential	0.0125	0.0573
H1: confidential	0.025	0.114
I1: confidential	1.30E-03	5.93E-03
J1: confidential	6.40E-03	0.0292
K1: confidential	8.79E-04	4.01E-03
K2: confidential	5.04E-03	0.023
L1: confidential	8.63E-05	3.94E-04
M1: confidential	1.91E-04	8.72E-04
N1: confidential	2.55E-03	0.0116
O1: rebonding	4.85E-05	2.22E-04
P1: confidential	4.61E-04	2.10E-03
Q1: adhesive pressing	1.30E-03	5.93E-03
R1: loose crumb	4.85E-05	2.22E-04

3.2.3.4 Calculation of PEClocal for private use

Not applicable. Non-industrial applications, in-service loss and waste remaining in the environment are characterised on a regional scale.

3.2.3.5 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.

3.2.3.6 Measured levels

Seawaters were analysed in a study by Weigel *et al.* (2005). Seawater samples were taken in June-July 1998 in various locations in the North Sea, at a depth of 5 m. Samples were analysed using GC-MS. A number of contaminants were detected and quantified throughout the North Sea. Concentrations of TCPP attained values between 1-8 ng/l. The 8 ng/l concentration was from a sample location in the area of the German Bight.

Andresen *et al.* (2007) monitored for TCPP 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 around 28 ng TCPP/l was measured. In the Bight, concentrations of around 5 to 24 ng/l were measured, with lowest concentrations seen in waters furthest offshore.

Table 3.52 TCPP concentrations in the environment: Marine data

Sample type	Location	Sample period	Analytical method	Results	Scale represented	Reliability	Study reference
Sea water	EU: North Sea	June-July1998	GC-MS	Max 8 ng/l	Local – Regional (highest concentrations may reflect local)	(4) not assignable	Weigel *et al.* (2005)
Sea water	EU: North Sea	May-June 2005	GC-MS	Max 24 ng/l	Local – Regional (highest concentrations may reflect local)	(4) not assignable	Andresen *et al.* (2007)

3.2.3.7 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 around 5E-05 to 0.025 mg/l. The predicted regional PEC for marine water is 4.85E-05 mg/l (equivalent to around 50 ng/l). The measured data, derived from a relatively limited number of samples, range from about 1 to 24 ng/l. The measured data are therefore not inconsistent with the modelled regional concentration and lower range local concentrations.

3.2.4 Secondary poisoning

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

Table 3.53 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.

3.2.4.1 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 TCPP (lower limit of quantitation 10 ng/g ww for all matrices). TCPP was not detected in any samples.

3.2.4.2 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, most of which are between 0.1 – 1 µg/kg wwt, are in agreement with this finding.

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

$PEC_{regional(sea\ water)}$ = 4.85E-05 mg/l from the EUSES v2.03 model

$PEC_{regional\ (marine\ sediment)}$ = 2.22E-04 mg/kg wwt from the EUSES v2.03 model.

$PEC_{continental(sea\ water)}$ = 3.06E-08 mg/l from the EUSES v2.03 model.

$PEC_{continental\ (marine\ sediment)}$ = 1.4E-07 mg/kg wwt from the EUSES v2.03 model.

Table 3.53 PECs for marine secondary poisoning

	$PEC_{oral, predator, fish (marine)}$ [mg.kgwwt-1]	$PEC_{oral marine top predator}$ [mg.kgwwt-1]
Producer 1	8.42E-04	2.73E-04
Producer 2	1.80E-03	4.64E-04
Producer 3	1.54E-04	1.36E-04
Producer 4	7.61E-04	2.57E-04
A1a: Large systems houses	1.37E-04	1.32E-04
A2: Medium systems houses	4.75E-03	1.06E-03
A3: Small systems houses	1.29E-03	3.63E-04
A4: Systems houses using preformulated polyol	2.26E-04	1.50E-04
B1a: flexible foam (furniture) very large	1.74E-04	1.40E-04
B1b: flexible foam (furniture) large	1.39E-04	1.33E-04
B1c: flexible foam (furniture) small - not using systems	2.10E-04	1.47E-04
B1d: flexible foam (furniture) small – users of systems	2.71E-04	1.59E-04
B2: flexible foam cutting	1.40E-04	1.33E-04
C1: rigid foaming large sites	1.32E-04	1.31E-04
C2: rigid foaming small sites	7.35E-04	2.52E-04
E1: one-component foams	5.95E-03	1.30E-03
F1: confidential	8.71E-04	2.79E-04
G1: confidential	3.09E-03	7.23E-04
G2: confidential	1.79E-03	4.64E-04
H1: confidential	1.98E-03	5.01E-04
I1: confidential	5.01E-04	2.05E-04
J1: confidential	4.12E-03	9.29E-04
K1: confidential	4.07E-04	1.86E-04
K2: confidential	1.39E-03	3.82E-04
L1: confidential	1.40E-04	1.33E-04
M1: confidential	2.89E-04	1.63E-04
N1: confidential	5.01E-04	2.05E-04
O1: rebonding	1.31E-04	1.31E-04
P1: confidential	5.89E-04	2.23E-04
Q1: adhesive pressing	5.75E-04	2.20E-04
R1: loose crumb	1.31E-04	1.31E-04

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

The following Sections review the available toxicity data for TCPP 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 TCPP 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 TCPP samples used in the tests reported below. It is acknowledged that the variation in composition of TCPP across manufacturers could lead to differing ecotoxicity profiles. All testing was conducted on composite samples and there is no evidence to indicate whether the isomers would have very different ecotoxicity profiles.

3.3.1 Aquatic compartment (incl. sediment)

Study reports have been submitted for consideration in respect of acute tests with fish, invertebrates, algae and micro-organisms and acute and chronic tests with aquatic invertebrates.

3.3.1.1 Toxicity test results

The contents of the test reports are summarised below and in **Table 3.54**.

The result of an acute toxicity test with *Oryzias latipes* reported in IUCLID has not been submitted for review. The test is assessed in IUCLID as being valid with restrictions. In addition, two 48-h LC_{50} values for tests with *Pimephales promelas* and *Lepomis macrochirus* are quoted in NICNAS (2001). These studies were also not submitted for review.

Table 3.54 Summary of aquatic toxicity test results for TCPP

Test species	Test protocol	Year test completed	Endpoint and exposure period	Result (mg/l)[1]	Reliability assessment	Comments	Study reference
Toxicity to fish							
Zebrafish (*Brachydanio rerio*)	Not given	1991	96-h LC_0 24-h LC_{50} 48-h LC_{50} 72-h LC_{50} 96-h LC_{50}	32 56 56 56 56	(1) valid without restriction	Fulfils all reliability criteria. Static test. Test results are presented as the geometric mean of 24, 48, 72 and 96-h LC_0 and LC_{100} values. LC_{50} values are therefore approximate	Kanne, 1991
Bluegill Sunfish (*Lepomis macrochirus*)	Not given	1985	96-h NOEC 96-h LC_{50}	6.3 84	(1) valid without restriction	Static test. The 96-h LC_{50} was determined by extrapolation outside the range of test concentrations. Comparison with a reported 120-h LC_{50} value of approximately 20 mg/l (based on mean measured concentrations) suggests that the 96 hour value is reasonable although perhaps a little high.	Meeks, 1985a
Fathead Minnows (*Pimephales promelas*)	Not given	1985	96-h NOEC 24-h LC_{50} 48-h LC_{50} 72-h LC_{50} 96-h LC_{50}	6.6 >51 >51 51 51	(1) valid without restriction	Fulfils all reliability criteria. Static test but with analysis of exposure concentrations.	Meeks, 1985b
Guppy (*Poecilia reticulata*)	OECD 203; ISO 7346-1	1997	48-h LC_{20} 48-h LC_{50} 96-h LC_{20} 96-h LC_{50}	22 (N*) 40 (N) 17 (N) 30 (N)	(4) not assignable	Static test. Only a summary report was available for review and there is insufficient information to fully evaluate the standard of the test. The test was not supported by chemical analysis and was not subject to GLP.	Griebenow, 1998
Killifish (*Oryzias*	Japanese	Not given	48-h LC_{50}	54 (N)	(4) not	Data are from a secondary source.	MITI, 1992

Test species	Test protocol	Year test completed	Endpoint and exposure period	Result (mg/l)[1]	Reliability assessment	Comments	Study reference
latipes)	Industrial Standard (JIS K 0102-1986-71)				assignable	Given that the data are not critical for deriving the PNEC the original test report has not been reviewed.	
Fish – acute QSAR (Esters)	ECOSAR (version 0.99g)		96-h LC_{50}	21		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}	11			
Fish – chronic QSAR (Esters)	ECOSAR (version 0.99g)		NOEC	5.2			
Toxicity to aquatic invertebrates							
Cladoceran (*Daphnia magna*)	DIN 38412/L11	1997	24-h EC_{20} 24-h EC_{50} 48-h EC_{20} 48-h EC_{50}	57 (N) 75 (N) 51 (N) 63 (N)	(4) not assignable	Only a summary report was available for review and there is insufficient information to fully evaluate the standard of the test. The test was not supported by chemical analysis and was not subject to GLP.	Griebenow, 1998
Cladoceran (*Daphnia magna*)	Not given	1985	48-h NOEC 48-h EC_{50}	33.5 131	(1) valid without restriction	Fulfils all reliability criteria.	Meeks, 1985c
Invertebrate - acute QSAR (Esters)	ECOSAR (version 0.99g)		48-h LC_{50}	63		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.	

Test species	Test protocol	Year test completed	Endpoint and exposure period	Result (mg/l)[1]	Reliability assessment	Comments	Study reference
Cladoceran (*Daphnia magna*)	OECD 202	1995	21-day NOEC (parent mortality) 21-day NOEC (reproduction) 14-day EC_{50} (parent immobilisation) 21-day EC_{50} (parent immobilisation) 21-day EC_{50} (reproduction)	32 (N) 32 (N) 42 (N) 40 (N) 32-56 (N)	(1) valid without restriction	Fulfils all reliability criteria. Analysis of exposure concentrations confirmed that they were close to nominal. Results are therefore expressed relative to nominal.	Sewell, Foulger and Bartlett, 1995
Invertebrate – longer term repro QSAR (Neutral organics)	ECOSAR (version 0.99h)		16-d EC_{50} (reproduction)	4.3		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. However the estimated value is within an order of magnitude of the measured value. 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 NOEC 72-h ErC_{10} (growth rate) 72-h EbC_{10} (biomass) 72-h ErC_{50} (growth rate)	13 (N) 42 (N) 14 (N) 82 (N) 33 (N)	(1) valid without restriction	Fulfils all the reliability criteria. Results are expressed relative to nominal concentrations because measured concentrations were within 80-120% of nominal. The study was subject to GLP.	Desjardins (2004)

Test species	Test protocol	Year test completed	Endpoint and exposure period	Result (mg/l)[1]	Reliability assessment	Comments	Study reference
			72-h EbC_{50} (biomass)				
Freshwater alga (*Scenedesmus subspicatus*)	DIN 38412/L33	1997	72-h EC_{20} (chlorophyll concentration) 72-h EC_{50} (chlorophyll concentration)	25 (N) 45 (N)	(4) not assignable	Only a summary report was available for review and there is insufficient information to fully evaluate the standard of the test. The test was not supported by chemical analysis and was not subject to GLP.	Griebenow, 1998
Freshwater alga (*Selenastrum capricornutum*) – note: now known as *Pseudokirchneriella subcapitata*	OECD 201; EEC DOC 89/88/XI, Directive 79/831, Annex V-C3	1991	96-h NOEC 96-h LOEC 96-h ErC_{50} (growth rate) 96-h EbC_{50} (biomass)	6 (N) 18 (N) 73 (N) 47 (N)	(3) invalid	The test was not supported by chemical analysis and test media were prepared by dilution of a stock suspension. These are significant inadequacies that invalidate the data for the purposes of risk assessment.	Kroon and van Ginkel, 1992
Algae QSAR (Esters)	ECOSAR (version 0.99g)		96-h EC_{50} 96-h NOEC	1.8 1.4		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	ISO 8192		EC_{50}	784	(2) valid with restrictions	The test is of an overall acceptable standard, despite some limitations in the test report.	Bayer, 1990
Photobacterium (*Vibrio fischeri*)			15-minute EC_{50}	171.5 ppm (95% confidence interval of 149.0 – 197.0 ppm	(3) invalid	Result of a LUMIStox bacteriological assay as part of a monitoring study from the open literature. The organism is also known as *Photobacterium phosphoreum*	Guzzella and Galassi, 1993

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

3.3.1.1.1 Fish

Acute toxicity

Study data

Reports have been submitted for five acute fish tests – one each with *Brachydanio rerio* (Zebrafish; Kanne, 1991), *Lepomis macrochirus* (Bluegill sunfish; Meeks, J.R., 1985a), *Pimephales promelas* (Fathead minnow; Meeks, J.R., 1985b), *Poecilia reticulata* (Guppy; Griebenow, 1998) and *Oryzias latipes* (Killifish; MITI, 1992). The tests with *B. rerio*, *L. macrochirus and P. promelas* all gave results that were acceptable for determining a PNEC – the lowest 96-h LC_{50} of approximately 51 mg/l was determined in the test with *P. promelas*. The 96-h LC_{50} or 48-h LC_{50} (*O. latipes*) values determined in the other 4 tests are in the range 30 to 84 mg/l and are therefore supportive of the *P. promelas* value.

Other test results reported elsewhere but not submitted for review

Although not in IUCLID, for completeness it is noted that two 48-h LC_{50} values of 98 and 180 mg/l and corresponding NOECs of 9.8 and 9.8 mg/l are quoted in NICNAS (2001). The values relate to acute tests carried out with *Pimephales promelas* and *Lepomis macrochirus* respectively. The results are referenced to *IPCS Environmental Health Criteria 209: Flame retardants: tris (chloropropyl) phosphate and tris (2-chloroethyl) phosphate* (1998) and there is no assessment of their validity. These results are supportive of the result obtained with *B. rerio* in the submitted study report.

QSAR estimated acute toxicity

Estimated values of 21 and 11 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 5.2 mg/l has been derived for chronic fish toxicity using an ECOSAR QSAR applicable to esters.

3.3.1.1.2 Aquatic invertebrates

Acute toxicity

Study data

Reports have been submitted for two acute invertebrate tests with *Daphnia magna* (Griebenow, 1998 and Meeks, 1985c). One test fulfilled the criteria for acceptability for

determining a PNEC, giving a 48-h EC_{50} value of 131 mg/l. The 48-h EC_{50} value obtained in the other test was 63 mg/l.

QSAR estimated acute toxicity

An estimated value of 63 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

Study data

A report has been submitted for one chronic invertebrate test with *Daphnia magna* (Sewell, Foulger and Bartlett, 1995). The test fulfilled all the acceptability criteria for determining a PNEC and gave a 21-day NOEC for reproduction of 32 mg/l.

QSAR estimated chronic toxicity

An estimated value of 4.3 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).

3.3.1.1.3 Algae

Acute toxicity

Study data

Reports have been submitted for three algal growth inhibition tests – two with *Pseudokirchneriella subcapitata* (also referred to as *Selenastrum capricornutum* and *Raphidocelis subcapitata*; Desjardins, 2004 and Kroon and van Ginkel, 1992) and one with *Scenedesmus subspicatus* (Griebenow, 1998). One of the tests with *P. subcapitata* fulfilled all the reliability criteria. The test gave a 72-h E_rC_{50} value for effects on growth rate of 82 mg/l, a 72-h E_rC_{10} of 42 mg/l and a NOEC of 13 mg/l. These results are below the reported water solubility value for TCPP of 1080 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. The results of the other test (Griebenow, 1998) were, however, supportive of those obtained in the reliable test (Desjardins, 2004).

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.

3.3.1.1.4 Micro-organisms

A translated report has been submitted for one microbial inhibition test (Kanne *et al.*, 1990). The report did not include information on dissolved oxygen concentrations, inhibition rates

for controls or reference substance or duration of the study. Dates for the start and end of the study indicate that the test duration may have been up to 2 days. No inhibition curve was presented in the translated report, although it is indicated that this was included in the original. Although the IC_{50} for the reference substance was not included in the original report, a supplementary document was provided indicating that the validity criterion was satisfied. Dissolved oxygen concentrations and respiration rates of controls were not reported. Despite some limitations in the test report, the test was conducted to GLP and according to ISO guidelines, therefore it is considered acceptable for determining the PNEC for micro-organisms. The IC_{50} was determined to be 784 mg/l.

As part of a monitoring study (Guzzella and Galassi, 1993; see section 3.1.4.2.2), water was extracted from the River Po at a site in Ferrara, at the closing section of the river basin. A bacterial assay using the *Vibrio fischeri* photobacterium (also known as *Photobacterium phosphoreum*) was used to determine toxicity of the samples; chemical analysis was performed using GC with a N/P-selective detector. The detection limit for organophosphorus compounds in water was 1 ng/l.

A LUMIStox bacteriological assay was used. The micropollutants were removed from the water and redissolved. Over a 15-minute exposure period, the EC_{50} for TCPP was found to be 171.5 ppm (95% confidence interval of 149.0-197.0).

3.3.1.1.5 Endocrine disrupting effects

Oestrogenic/anti-oestrogenic effects have been investigated by Föllmann and Wober (2006) using the recombinant yeast reporter gene assay and by induction of the alkaline phosphatase enzyme in human endometrial cancer Ishikawa cells. The original study report has not been reviewed and therefore a reliability rating of 4 (not assignable) is applicable to the results. No induction of oestrogenic or anti-oestrogenic effects was detected in either of the test systems.

Prediction of oestrogen receptor binding and reporter gene response made by the Environmental Protection Agency of Denmark gave a negative prediction for oestrogen receptor binding and no robust prediction for reporter gene response. The predictions were made using the Multicase model based on data from the Japanese METI test presented at the 6th Meeting of the Task Force on Endocrine Disrupters Testing and Assessment (EDTA) held in Tokyo from 24-25 June 2002.

3.3.1.1.6 Amphibians

No amphibian effects data were available for review.

3.3.1.2 Calculation of Predicted No Effect Concentration (PNEC)

Study data

The lowest values are as follows:

Acute toxicity to fish	96-hr LC_{50}	= 51 mg/l
Acute toxicity to invertebrates	48-hr EC_{50}	= 131 mg/l
Acute toxicity to algae	72-hr E_rC_{50}	= 82 mg/l

Chronic toxicity to invertebrates (repro test)	21-day NOEC	= 32 mg/l
Chronic toxicity to algae	72 hr E_rC_{10}	= 42 mg/l (NOEC 13 mg/l)
Toxicity to WWTP micro-organisms	EC_{50}	= 784 mg/l

QSAR estimates

Acute toxicity to fish	96-hr LC_{50}	= 11 - 21 mg/l
Chronic toxicity to fish	NOEC	= 5.2 mg/l
Acute toxicity to invertebrates	48-hr LC_{50}	= 63 mg/l
Chronic toxicity to invertebrates	16-d EC_{50}	= 4.3 mg/l
Acute toxicity to algae	96-hr EC_{50}	= 1.8 mg/l
Chronic toxicity to algae	96-hr NOEC	= 1.4 mg/l

PNEC$_{aquatic}$

Fish were marginally more susceptible to TCPP 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 32 mg/l and an E_rC_{10} value of 42 mg/l (NOEC 13 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 0.64 mg/l has been derived from the *Daphnia* test data by dividing the NOEC of 32 mg/l for effects on *Daphnia magna* reproduction by an assessment factor of 50.

This value is the $PNEC_{aquatic}$ considered by the Rapporteur as the most appropriate value. However, for the purposes of comparison, an alternative PNEC is derived from the algal NOEC. This is in accordance with guidance received from TC NES I 05, because 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 of the study. The *Daphnia* result is more sensitive than the algal E_rC_{10}, hence, the PEC/PNEC ratios presented in the report are based on the PNEC value shown above.

An alternative $PNEC_{aquatic}$ of 0.26 mg/l can be derived from the algal test data by dividing the NOEC of 13 mg/l for effects on *P. subcapitata* by an assessment factor of 50.

This suggests that, using this alternative analysis of the test results, the risks to fresh water could be up to 2.46 times greater than the values presented in the report. This is commented upon in the Conclusions to the risk assessment.

Micro-organisms

The PNEC for waste-water treatment is 7.84 mg/l based on the IC_{50} of 784 mg/l and an assessment factor of 100.

Sediment-dwelling organisms

No toxicity data are currently available for sediment-dwelling organisms, therefore it is not possible to determine a $PNEC_{sed}$ based on measured data. According to the Technical Guidance Document, $PNEC_{sed}$ can be calculated by the equilibrium partitioning method using the following equation:

$$PNEC_{sed} = \frac{K_{susp\text{-}water}}{RHO_{susp}} * PNEC_{water} * 1000$$

For TCPP this is:

$$PNEC_{sed} = \frac{5.25}{1150} * 0.64 * 1000$$

$$= 2.92 \text{ mg/kg wwt}$$

Hence, $PNEC_{sed}$ = 2.92 mg/kg will be used for risk characterisation.

3.3.2 Terrestrial compartment

3.3.2.1 Toxicity test results

Short and long-term tests have been conducted with the earthworm, *Eisenia foetida* and long-term tests with the plant species *Triticum aestivum* (Wheat), *Sinapis alba* (Mustard) and *Lactuca sativa* (Lettuce) for TCPP. A 14-day LC_{50} of 97 mg/kg has been determined in the short-term test with *E. foetida*. Lowest NOECs of 53 and 17 mg/kg soil dry weight have been determined in the long-term tests *with E. foetida* and *L. sativa* (Lettuce) respectively. The results of a test with soil micro-organisms (nitrogen transformation) for TDCP have been read across to TCPP. The results are summarised in **Table 3.55**.

Table 3.55 Summary of terrestrial toxicity test results for TCPP

Test species	Test protocol	Year test completed	Endpoint and exposure period	Result (mg/kg dry weight)[1]	Reliability assessment	Comments	Study reference
Toxicity to earthworm							
Earthworms (*Eisenia foetida*)	OECD 207	1996	14-day NOEC 7-day LC_{50} 14-day LC_{50}	32 (N) 131 (N) 97 (N)	(2) valid with restrictions	The test was not subject to GLP. The test is of an overall acceptable standard although there are inadequacies in some elements. Organic matter content in the test soil was 10%.	Wetton, 1996
Earthworms (*Eisenia foetida*)	OECD draft guideline (January 2000): Earthworm Reproduction Test	2003	28 day NOEC (mortality) 28 day NOEC (biomass) 28 day LOEC (biomass) EC_{50} for reproduction 56 day NOEC for reproduction 56 day LOEC for reproduction	≥ 196 (N) 116 (N) 151 (N) 71 (N) 53 (N) 69 (N)	(1) valid without restriction	Fulfils all reliability criteria. A fully valid GLP study. Organic matter content in the test soil was 10%.	Servajean, 2003a
Toxicity to higher plants							
Wheat (*Triticum aestivum*), Mustard (*Sinapsis alba*), Lettuce (*Lactuca sativa*)	OECD Guideline 208	2003	NOEC (emergence): Wheat Mustard Lettuce NOEC (dry weight): Wheat	≥98 (N) 30 (N) 17 (N) 22 (N) 29 (N)	(1) valid without restriction	Fulfils all reliability criteria. A fully valid GLP study. Organic matter content in the test soil was 1.4%.	Servajean, 2003b

Test species	Test protocol	Year test completed	Endpoint and exposure period	Result (mg/kg dry weight)[1]	Reliability assessment	Comments	Study reference
			Mustard Lettuce	18 (N)			
Toxicity to soil micro-organisms							
Nitrifying micro-organisms in sandy loam soil (TDCP)	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	Study conducted using a similar test substance (TDCP) Fulfils all the reliability criteria. The study was subject to GLP. Organic matter content in the test soil was 1%.	van Ginkel (2005)

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

3.3.2.1.1 Earthworm

Acute toxicity

A report has been submitted for one short-term acute test with the earthworm *Eisenia foetida* (Wetton, P.M. 1996). The test fulfilled the criteria for acceptability for determining a PNEC. A 14-day LC_{50} of 97 mg/kg dwt has been determined in the test along with a 14-day NOEC of 32 mg/kg dwt.

The organic matter content 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 is therefore applied, giving standardised results of:

14-day $LC_{50standardised}$ 33.0 mg/kg dry weight.
14-day $NOEC_{standardised}$ 10.9 mg/kg dry weight

Long-term toxicity

A report has been submitted for one long-term test with the earthworm *Eisenia foetida* (Servajean, E. 2003a). The test fulfilled the criteria for acceptability for determining a PNEC. A 56-day NOEC (28-day adult plus 28-day juvenile exposure period) of 53 mg/kg dwt for earthworm reproduction has been determined in the test.

The organic matter content 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 is therefore applied, giving standardised results of:

56-day $NOEC_{standardised}$ 18.02 mg/kg dry weight

3.3.2.1.2 Higher plants

Long-term toxicity

A report has been submitted describing the results of emergence and growth tests with the plant species *Triticum aestivum* (Wheat), *Sinapis alba* (Mustard), *Lactuca sativa* (Lettuce) (Servajean, E. 2003b). The tests fulfilled the criteria for acceptability for determining a PNEC. The lowest NOEC determined in the tests was 17 mg/kg dry weight for emergence of *L. sativa* seedlings. The lowest NOECs determined for *S. alba* and *L. sativa* were 28 and 18 mg/kg respectively based on 21-day post emergence plant wet weight.

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 = 17 mg/kg dry weight

3.3.2.1.3 Terrestrial micro-organisms

Inhibition of soil nitrogen transformation by soil micro-organisms was examined in a study with TDCP conducted voluntarily by industry (van Ginkel, 2005). A 28-day NOEC of

≥128 mg/kg wet weight (no inhibition at the highest concentration tested) was determined in the test. The only other data relevant are for WWTP micro-organisms and these suggest a consistent low order of acute toxicity for TCPP (and structurally-related substances TDCP and V6). Due to the structural similarity of TDCP to TCPP, their similar physico-chemical properties and their lack of toxicity to WWTP micro-organisms, it is considered justifiable to read-across the long-term soil nitrogen transformation effects data from TDCP to TCPP. This was agreed at TCNES III 05.

This read-across is further supported by reference to the effects on other terrestrial organisms of TCPP and TDCP from high-reliability studies. The two substances have a very similar level of toxicity to higher plants ($NOEC_{emergence}$ of 19 mg/kg wwt for TDCP compared to 17 mg/kg wwt for TCPP). Earthworms show less sensitivity to TCPP than to TDCP in both short- and long-term studies (14-day LC_{50} of 23 mg/kg wwt for TDCP compared to 33 mg/kg wwt for TCPP; chronic $NOEC_{repro}$ of 3.3 mg/kg wwt for TDCP compared to 18 mg/kg wwt for TCPP).

3.3.2.2 Calculation of Predicted No Effect Concentration (PNEC)

The lowest values available are as follows:

Toxicity to earthworms	14 d LC_{50}	= 33 mg/kg dwt
Chronic toxicity to earthworms	56 d NOEC	= 18 mg/kg dwt
Toxicity to higher plants	NOEC	= 17 mg/kg dwt
Toxicity to soil micro-organisms (nitrifying micro-organisms in sandy loam soil) by read-across from TDCP	28 d NOEC	= 128 mg/kg wwt

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 17/10 = 1.7 mg/kg soil dry weight, equivalent to 1.5 mg/kg soil wet weight.

3.3.3 Atmosphere

No data are available on the toxicity of TCPP to plants or other organisms exposed via air. Based on its structure, TCPP 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 a similar substance (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 TCPP contributing to atmospheric effects such as global warming, ozone depletion and acid rain is likely to be very small.

3.3.4 Secondary poisoning

3.3.4.1 Effect data

The most relevant data for derivation of the PNEC for secondary poisoning for TCPP are from a 13-week study in the rat. The lowest dose tested resulted in effects and hence no dose-based NOAEL is available. The LOAEL is 52 mg/kg bw/day, based on liver effects (increase in absolute and relative liver weights, accompanied by mild thyroid follicular cell hyperplasia, observed in males of all dose groups). For full details refer to Section 4.1.2.6.1.

Using the conversion factors given in the Technical Guidance Document:
LOAEL = 52 mg/kg bw/d
NOAEL <52 mg/kg bw/d

NOEC mammal = NOAEL mammal x CONV mammal

NOEC = <52 mg/kg bw/d x 20 (animal age >6 weeks)
= <1040 mg/kg food

Toxicokinetics data show that there is 80% absorption by the oral route.

3.3.4.2 Calculation of $PNEC_{oral}$

According to the Technical Guidance Document an assessment factor of 90 is appropriate for the results of a study of this duration. Therefore, applying this assessment factor:
PNEC oral = NOAEL/AF

PNEC oral = <1040/90
= <11.6 mg/kg food

A PNEC for secondary poisoning of <11.6 mg/kg food will be used. This value is also applicable for the assessment of secondary poisoning in the marine environment.

3.3.5 MARINE EFFECTS ASSESSMENT

3.3.5.1 Calculation of Predicted No Effect Concentration (PNEC)

PNEC*seawater*

No measured data are currently available for marine organisms therefore the marine PNEC is derived from data obtained for freshwater species (NOEC = 32 mg/l), applying an assessment factor of 500 to give PNEC*seawater* = 0.064 mg/l.

PNEC*marine sediment*

No measured data are currently available for marine sediment organisms therefore the PNEC is derived by equilibrium partitioning to give PNEC*marine sediment* = 0.292 mg/kg.

3.4 RISK CHARACTERISATION

PEC values for fresh and marine water, sediment and soil, and for predators are given in **Tables 3.14** to **3.16**, **3.37** to **3.40** and **3.48** to **3.53**. PEC/PNEC ratios are given in **Tables 3.57** to **3.62**.For ease of reference, the PNECs used in the risk assessment are summarised in **Table 3.56** below.

Table 3.56 PNECs used in the risk assessment of TCPP

Compartment	Value of PNEC
Freshwater	0.64 mg/l *0.26 mg/l (alternative value for comparison)*
Freshwater sediment	2.92 mg/kg wet weight (equilibrium partitioning)
WWTP micro-organisms	7.84 mg/l
Seawater	0.064 mg/l (extrapolation from freshwater)
Marine sediment	0.292 mg/kg wet weight (extrapolation from freshwater)
Soil	1.5 mg/kg wet weight
Secondary poisoning	<11.6 mg/kg food

3.4.1 Aquatic compartment (incl. sediment)

3.4.1.1 Water and sediment

Table 3.57 PEC/PNEC ratios for surface water and freshwater sediments

Scenario	$PEC/PNEC_{water}$	$PEC/PNEC_{sediment}$
Producer 1	1.78E-03	1.78E-03
Producer 2	0.0168	0.0168
Producer 3	8.12E-04	8.12E-04
Producer 4	2.00E-03	2.00E-03
A1a: Large systems houses	7.88E-04	7.88E-04
A2: Medium systems houses	0.0645	0.0645
A3: Small systems houses	0.0168	0.0168
A4: Systems houses using preformulated polyol	2.69E-03	2.69E-03
B1a: flexible foam (furniture) very large	1.37E-03	1.37E-03
B1b: flexible foam (furniture) large	8.85E-04	8.85E-04

Scenario	PEC/PNEC$_{water}$	PEC/PNEC$_{sediment}$
B1c: flexible foam (furniture) small - not using systems	1.86E-03	1.86E-03
B1d: flexible foam (furniture) small - users of systems	2.71E-03	2.71E-03
B2: flexible foam cutting	9.10E-04	9.10E-04
C1: rigid foaming large sites	7.99E-04	7.99E-04
C2: rigid foaming small sites	9.10E-03	9.10E-03
E1: one-component foams	0.081	0.081
F1: confidential	0.013	0.013
G1: confidential	0.205	0.205
G2: confidential	0.192	0.192
H1: confidential	0.383	0.383
I1: confidential	0.0199	0.0199
J1: confidential	0.0979	0.0979
K1: confidential	0.0135	0.0135
K2: confidential	0.077	0.077
L1: confidential	1.36E-03	1.36E-03
M1: confidential	2.96E-03	2.96E-03
N1: confidential	0.039	0.039
O1: rebonding	7.80E-04	7.80E-04
P1: confidential	7.09E-03	7.09E-03
Q1: adhesive pressing	0.0199	0.0199
R1: loose crumb	7.80E-04	7.80E-04

PEC/PNEC$_{regional(water)}$ = 7.80E-04 from the EUSES v2.03 model.

PEC/PNEC $_{regional(freshwater\ sediment)}$ = 8.28E-04 from the EUSES v2.03 model.

Conclusions to the risk assessment for the aquatic compartment:

PEC/PNEC ratios for water and sediment are reported in **Table 3.57**. No risks are identified. As noted in the derivation of PNECaquatic (see section 3.3.1.2), the use of the algal NOEC as the basis of PNEC would lead to PEC/PNEC ratios 2.46 times higher than those calculated here. This would not have any implications for the conclusions of the assessment based on the data in **Table 3.57**.

Due to the use of the equilibrium partitioning method, values for the sediment are identical to those for the water column[19]. The use of the equilibrium partitioning method is supported by evidence from the related substance TDCP where the PNEC from sediment studies is similar to that from equilibrium partitioning.

[19] Use of the equilibrium partitioning method to derive PNEC for sediment means that both PECsediment and PNECsediment are derived from the respective values for the associated aquatic compartment using the same factor, K*susp*water. This direct proportionality means that PEC/PNEC ratios are the same for sediment as for water.

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.

This applies to all local life cycle stages and also at the regional scale.

3.4.1.2 Wastewater treatment processes

Table 3.58 PEC/PNEC ratios for wastewater treatment plants

Scenario	$PEC/PNEC_{WWTP}$
Producer 1	0.0082
Producer 2	0.019
Producer 3	0.0044
Producer 4	9.99E-03
A1a: Large systems houses	0.0762
A2: Medium systems houses	0.052
A3: Small systems houses	0.0131
A4: Systems houses using preformulated polyol	1.56E-03
B1a: flexible foam (furniture) very large	4.79E-04
B1b: flexible foam (furniture) large	8.59E-05
B1c: flexible foam (furniture) small - not using systems	8.84E-04
B1d: flexible foam (furniture) small - users of systems	1.58E-03
B2: flexible foam cutting	1.06E-04
C1: rigid foaming large sites	1.60E-05
C2: rigid foaming small sites	6.80E-03
E1: one-component foams	0.0655
F1: confidential	9.99E-03
G1: confidential	0.166
G2: confidential	0.156
H1: confidential	0.312
I1: confidential	0.0156
J1: confidential	0.0793
K1: confidential	0.0104
K2: confidential	0.0623
L1: confidential	4.71E-04
M1: confidential	1.78E-03
N1: confidential	0.0312
O1: rebonding	0
P1: confidential	5.15E-03
Q1: adhesive pressing	0.0156
R1: loose crumb	0

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

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.

This applies to all life cycle stages.

3.4.2 Terrestrial compartment

PEC/PNEC ratios for the terrestrial compartment are presented in **Table 3.59**.

Table 3.59 PEC/PNEC ratios for soil

Scenario	$PEC/PNEC_{soil}$
Producer 1	3.83E-03
Producer 2	3.83E-03
Producer 3	3.83E-03
Producer 4	0.0102
A1a: Large systems houses	0.055
A2: Medium systems houses	0.0376
A3: Small systems houses	0.0123
A4: Systems houses using preformulated polyol	4.84E-03
B1a: flexible foam (furniture) very large	4.14E-03
B1b: flexible foam (furniture) large	3.89E-03
B1c: flexible foam (furniture) small - not using systems	4.40E-03
B1d: flexible foam (furniture) small - users of systems	4.84E-03
B2: flexible foam cutting	3.90E-03
C1: rigid foaming large sites	3.84E-03
C2: rigid foaming small sites	8.17E-03
E1: one-component foams	0.0462
F1: confidential	0.0103
G1: confidential	0.11
G2: confidential	0.104
H1: confidential	0.203
I1: confidential	0.0138
J1: confidential	0.0549
K1: confidential	0.0106
K2: confidential	0.0436
L1: confidential	4.13E-03
M1: confidential	4.99E-03
N1: confidential	0.0238
O1: rebonding	3.83E-03

Scenario	$PEC/PNEC_{soil}$
P1: confidential	7.18E-03
Q1: adhesive pressing	0.0139
R1: loose crumb	3.83E-03

$PEC/PNEC_{regional(soil)}$ = 1.77E-03 from the EUSES v2.03 model.

Conclusions to the risk assessment for the terrestrial compartment:

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.

This applies to all local life cycle stages and also at the regional scale.

3.4.3 Atmosphere

Neither biotic nor abiotic effects on the atmosphere are likely because of the low predicted environmental concentrations of TCPP (all concentrations are below 1 $\mu g/m^3$).

Conclusions to the risk assessment for atmosphere:

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.

This applies to all life cycle stages.

3.4.4 Secondary poisoning

PEC/PNEC ratios for secondary poisoning are presented in **Table 3.60**.

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, no values are close to 1 (they are all at least one order of magnitude below 1) and due to the lack of any significant bioaccumulation potential of TCPP, it is reasonable to conclude that there are no risks.

Table 3.60 PEC/PNEC ratios for secondary poisoning

Scenario	$PEC/PNEC_{fish\ eating}$	$PEC/PNEC_{worm\ eating}$
Producer 1	>1.78E-04	>7.11E-04
Producer 2	>1.1E-03	>7.11E-04
Producer 3	>1.19E-04	>7.11E-04
Producer 4	>1.7E-04	>1.38E-03
A1a: Large systems houses	>1.17E-04	>6.11E-03
A2: Medium systems houses	>4.03E-03	>4.25E-03
A3: Small systems houses	>1.1E-03	>1.6E-03
A4: Systems houses using preformulated polyol	>1.97E-04	>8.16E-04
B1a: flexible foam (furniture) very large	>1.53E-04	>7.44E-04
B1b: flexible foam (furniture) large	>1.23E-04	>7.17E-04
B1c: flexible foam (furniture) small - not using systems	>1.83E-04	>7.7E-04
B1d: flexible foam (furniture) small - users of systems	>2.35E-04	>8.16E-04
B2: flexible foam cutting	>1.25E-04	>7.18E-04
C1: rigid foaming large sites	>1.18E-04	>7.12E-04
C2: rigid foaming small sites	>6.28E-04	>1.16E-03
E1: one-component foams	>5.05E-03	>5.14E-03
F1: confidential	>7.43E-04	>1.39E-03
G1: confidential	>2.62E-03	>0.0118
G2: confidential	>1.53E-03	>0.0111
H1: confidential	>1.68E-03	>0.0215
I1: confidential	>4.3E-04	>1.76E-03
J1: confidential	>3.5E-03	>6.05E-03
K1: confidential	>3.51E-04	>1.42E-03
K2: confidential	>1.18E-03	>4.86E-03
L1: confidential	>1.25E-04	>7.42E-04
M1: confidential	>2.5E-04	>8.32E-04
N1: confidential	>4.3E-04	>2.79E-03
O1: rebonding	>1.17E-04	>7.11E-04
P1: confidential	>5.04E-04	>1.06E-03
Q1: adhesive pressing	>4.92E-04	>1.76E-03
R1: loose crumb	>1.17E-04	>7.11E-04

Conclusions to the risk assessment for secondary poisoning:

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.

This applies to all local life cycle stages.

3.4.5 Marine environment

3.4.5.1.1 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). No biodegradation simulation tests are available for TCPP. TCPP is not readily biodegradable but there is some evidence for ultimate biodegradation. It showed inherent biodegradability in a SCAS test and prolonged closed bottle test (See Section 3.1.3.1.4) but does not meet the TGD criteria for inherent biodegradability. TCPP is therefore considered to be potentially persistent, the screening criterion for persistence is met.

Bioaccumulation

The criterion used in the marine risk assessment for bioaccumulation is a bioconcentration factor (BCF) >2,000 l/kg. TCPP has a measured fish BCF of 0.8-4.6 in a reliable study and hence does not meet the B criterion.

Toxicity

The toxicity criterion used in the marine risk assessment guidance 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 TCPP is 32 mg/l measured in 21-day *Daphnia* study. Regarding human health effects, the possibility of read across of carcinogenicity data from structurally similar compounds is currently under consideration and has not yet been discussed at TC NES. Further mutagenicity testing is being carried out to determine the genotoxic potential of TCPP and a two-generation fertility study is also underway. 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. This conclusion may need to be re-visited once the mutagenicity and fertility testing have been completed.

Summary of PBT assessment

PBT assessment

For the PBT assessment, TCPP can be considered to meet the screening criteria as persistent (P) or potentially very persistent (vP) based on its ultimate mineralisation. The available information on bioaccumulation shows that TCPP does not meet the B or vB criterion. The T criterion is not met, though this should be reviewed once the human health data set is completed.

3.4.5.2 Marine risk characterisation

Table 3.61 PEC/PNEC ratios for sea water and marine sediments

Scenario	$PEC/PNEC_{sea\ water}$	$PEC/PNEC_{marine\ sediment}$
Producer 1	0.0108	0.0108
Producer 2	0.0242	0.0242
Producer 3	1.08E-03	1.08E-03
Producer 4	0.0133	0.0133
A1a: Large systems houses	8.43E-04	8.43E-04
A2: Medium systems houses	0.0658	0.0658
A3: Small systems houses	0.0171	0.0171
A4: Systems houses using preformulated polyol	2.71E-03	2.71E-03
B1a: flexible foam (furniture) very large	1.36E-03	1.36E-03
B1b: flexible foam (furniture) large	8.66E-04	8.66E-04
B1c: flexible foam (furniture) small - not using systems	1.86E-03	1.86E-03
B1d: flexible foam (furniture) small - users of systems	2.73E-03	2.73E-03
B2: flexible foam cutting	8.91E-04	8.91E-04
C1: rigid foaming large sites	7.78E-04	7.78E-04
C2: rigid foaming small sites	9.26E-03	9.26E-03
E1: one-component foams	0.0828	0.0828
F1: confidential	0.0133	0.0133
G1: confidential	0.209	0.209
G2: confidential	0.196	0.196
H1: confidential	0.391	0.391
I1: confidential	0.0203	0.0203
J1: confidential	0.0999	0.0999
K1: confidential	0.0137	0.0137
K2: confidential	0.0787	0.0787
L1: confidential	1.35E-03	1.35E-03

Scenario	PEC/PNEC$_{sea\ water}$	PEC/PNEC$_{marine\ sediment}$
M1: confidential	2.98E-03	2.98E-03
N1: confidential	0.0398	0.0398
O1: rebonding	7.58E-04	7.58E-04
P1: confidential	7.20E-03	7.20E-03
Q1: adhesive pressing	0.0203	0.0203
R1: loose crumb	7.58E-04	7.58E-04

PEC/PNEC $_{regional(sea\ water)}$ = 7.58E-04 from the EUSES v2.03 model

PEC/PNEC $_{regional\ (marine\ sediment)}$ = 7.59E-04 from the EUSES v2.03 model.

Conclusions to the risk assessment for the marine environment:

PEC/PNEC ratios for sea water and marine sediments are presented in **Table 3.61**.

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.

This applies to all local life cycle stages and also at the regional scale.

Secondary poisoning in the marine environment

PEC/PNEC ratios for secondary poisoning are presented in **Table 3.62**.

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

Table 3.62 PEC/PNEC ratios for secondary poisoning in the marine environment

Scenario	$PEC/PNEC_{marine\ predator}$	$PEC/PNEC_{marine\ top\ predator}$
Producer 1	>7.29E-05	>2.37E-05
Producer 2	>1.56E-04	>4.02E-05
Producer 3	>1.33E-05	>1.17E-05
Producer 4	>6.59E-05	>2.22E-05
A1a: Large systems houses	>1.19E-05	>1.14E-05
A2: Medium systems houses	>4.11E-04	>9.13E-05
A3: Small systems houses	>1.12E-04	>3.14E-05
A4: Systems houses using preformulated polyol	>1.95E-05	>1.3E-05
B1a: flexible foam (furniture) very large	>1.5E-05	>1.21E-05
B1b: flexible foam (furniture) large	>1.2E-05	>1.15E-05
B1c: flexible foam (furniture) small - not using systems	>1.81E-05	>1.27E-05
B1d: flexible foam (furniture) small - users of systems	>2.35E-05	>1.38E-05
B2: flexible foam cutting	>1.22E-05	>1.15E-05
C1: rigid foaming large sites	>1.15E-05	>1.14E-05
C2: rigid foaming small sites	>6.36E-05	>2.18E-05
E1: one-component foams	>5.15E-04	>1.12E-04
F1: confidential	>7.53E-05	>2.41E-05
G1: confidential	>2.67E-04	>6.25E-05
G2: confidential	>1.55E-04	>4.01E-05
H1: confidential	>1.71E-04	>4.33E-05
I1: confidential	>4.33E-05	>1.77E-05
J1: confidential	>3.57E-04	>8.04E-05
K1: confidential	>3.53E-05	>1.61E-05
K2: confidential	>1.2E-04	>3.3E-05
L1: confidential	>1.21E-05	>1.15E-05
M1: confidential	>2.5E-05	>1.41E-05
N1: confidential	>4.33E-05	>1.77E-05
O1: rebonding	>1.13E-05	>1.13E-05
P1: confidential	>5.09E-05	>1.93E-05
Q1: adhesive pressing	>4.97E-05	>1.9E-05
R1: loose crumb	>1.13E-05	>1.13E-05

Conclusions to the risk assessment for secondary poisoning in the marine 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.

This applies to all life cycle stages.

3.4.5.3 Areas of uncertainty in the environmental risk assessment

There is always statistical uncertainty regarding all the property inputs to the modelling of PEC/PNEC ratios, but none of these would have a large enough effect to change the conclusions. The risk assessment uses a reliable property data set, including some well-supported read-across data, and justifiable use pattern and release rate parameters. The Rapporteur has no reason to anticipate significant tonnage increases in the near future, based on industry information and general research.

For sediment dwelling organisms, the use of the equilibrium partitioning method as the basis of the PNEC is not considered to be a significant uncertainty, because there is evidence from the related substance TDCP that the PNEC from sediment studies is similar to that from equilibrium partitioning.

With regard to secondary poisoning, 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, due to the low ratios and lack of any significant bioaccumulation potential of TCPP, it is reasonable to conclude that there are no risks.

With regard to the marine risk assessment, extrapolation of data measured for the freshwater environment to the marine environment has been done in accordance with the TGD.

4 HUMAN HEALTH

4.1 HUMAN HEALTH (TOXICITY)

4.1.1 Exposure assessment

4.1.1.1 Occupational exposure

General introduction

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.

The general discussion summarises the important issues arising from the exposure assessment and brings together measured exposure data and predictions from the EASE (Estimation and Assessment of Substance Exposure) model. EASE is a general purpose predictive model for workplace exposure assessments. EASE is essentially a series of decision trees. For any substance, the system asks a number of questions about the physical properties of the substance and the circumstances of its use. For most questions, the EASE user is given a multiple-choice list from which to select the most appropriate response. Once all the questions have been answered, the exposure prediction is determined absolutely by the choices made. EASE can be used to estimate inhalation and dermal exposure – dermal exposure is assessed as the potential exposure rate to the hands and forearms (a total skin area of approximately 2000 cm^2). The output ranges generated by EASE for inhalation exposure relate to steady-state conditions, and estimate the average concentration of the substance in the atmosphere over the period of exposure.

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

Overview of exposure

TCPP is a liquid at room temperature with a low vapour pressure of 1.4×10^{-3} Pa at 25^0C and a calculated saturated vapour concentration (SVC) of 0.19 mg/m^3 at 21^0C.

EASE modelling will not be used to predict inhalation exposure to TCPP due to the low volatility of the substance, as EASE has limitations estimating the inhalation exposure to such a substance. For a substance with such a low vapour pressure EASE always predicts an exposure range of 0-0.1 ppm (0-1.3 mg/m^3 for TCPP). The upper level is clearly much too high with respect to a SVC of 0.19 mg/m^3. EASE has been used to estimate inhalation exposure to dust containing TCPP where appropriate.

Occupational exposure to TCPP may occur during its manufacture and during the manufacture and cutting of flexible and rigid polyurethane (PUR) foam. Inhalation of vapours and liquid aerosols and skin contact are the predominant routes of exposure during manufacture of TCPP and manufacture of foam, while inhalation of dust and skin contact are

thought to be the predominant routes of exposure during foam conversion and cutting of rigid foam. Oral exposure is not considered to be a significant route of exposure under normal working practices. The total number of people occupationally exposed to TCPP is not known but it is likely to be thousands if the foam cutting companies and construction workers using laminates are taken into account.

Descriptions of the processes and sources of occupational exposure are discussed below along with a discussion of exposure levels. All of the measured data used in this assessment has been supplied by industry, either directly or through trade organisations. The occupational exposure scenarios are:

1. Manufacture of TCPP
2. Manufacture of flexible PUR foam
3. Cutting of flexible PUR foam
4. Production of foam granules and rebonded PUR foam
5. Formulation of systems and manufacture of spray foam
6. Use of spray foams
7. Manufacture of rigid PUR foam
8. Use of rigid PUR foam
9. Manufacture of one-component foams
10. Use of one-component foams

Following manufacture, most TCPP (over 98%) produced in the EU is used as a flame retardant in the production of polyurethane (PUR) for use in construction and furniture. PUR is produced from the reaction of di-isocyanates with polyols. TCPP can be added to polyols in the production of PUR systems (around 50-60% - see section 4.1.1.1.5 below) or added directly at the point of foaming.

Most TCPP is used in rigid PUR foam (over 80%), mainly for construction applications. The remaining PUR applications are accounted for by flexible foam (over 17%), used in upholstery and bedding for the UK market.

Use of TCPP in products other than PUR tends to be associated with single users who have tried the product of their own accord and decided to use it. Industry has indicated that other possible applications include paints, unsaturated polyester resins and epoxy resins. No further information is available on these uses or the number of workers potentially exposed to TCPP through these uses. The very low tonnage involved confirms that TCPP is not widely used outside the PUR industry and so the uses are not considered further for the purpose of this risk assessment.

The total number of workers potentially exposed to TCPP during the production of PUR foam in the EU is difficult to estimate. Industry has informed the rapporteur that for flexible foam, EUROPUR members (representing about 85% of the market) have about 68 plants in the EU. Some plants use TCPP more frequently than others. A fair assumption may be that approximately 5 operators per plant can be around the foaming tunnel during production, bearing in mind the frequency of use of TCPP will vary somewhat from plant to plant. This gives an estimated total of 340 workers exposed to TCPP through the manufacture of flexible polyurethane foam in the EU.

For the production of rigid foam, a recent survey has shown that there are about 190 rigid foam manufacturing plants in the EU (ISOPA survey, 2003). Again, it is difficult to estimate the total number of operators potentially exposed to TCPP in these plants, as not all plants use TCPP. A reasonable estimate would be that about 10 workers or 2 per shift would work in the

foam production area. This gives an estimated total of 1,900 workers exposed to TCPP through the manufacture of rigid polyurethane foam in the EU.

Occupational exposure limits

There are no occupational exposure limits set for TCPP

4.1.1.1.1 Scenario 1: Occupational exposure during the manufacture of TCPP

TCPP is manufactured by four producers in the EU. In the year 2000, the total EU production was 36,000 tonnes. Between 1998 and 2003, production has increased significantly but the total EU sales tonnage has remained reasonably stable within approximately 10%. 8,304 tonnes of TCPP were imported into the EU in 2001 and 6,211 tonnes were exported from the EU in the year 2000. A further quantity of 1,200 tonnes of TCPP is believed to be imported into the EU in finished goods.

In all production facilities, TCPP is produced by reacting phosphorous oxychloride with propylene oxide followed by purification. The crude product is washed and dehydrated to remove acidic impurities and residual traces of catalyst. The product is then filtered, transferred to storage tanks for despatch in road tankers or packed into drums. There are some slight differences in procedures between the four different production plants. A brief description of production processes is given below for each facility and comments made in the summary part regarding the differences and typical procedures.

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 TCPP manufacturing plants were evaluated under actual working conditions. At this plant, TCPP is produced in a closed system. It is produced in batches, with 3 batches being run simultaneously. All transfers are done using closed lines. Storage is in closed vessels under nitrogen to exclude moisture and air. 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 TCPP during the production steps. Only one operator per shift is assigned to the plant and he spends most of his 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. At the fluids plant, blending and drumming occurs. There are 2 filling stations and both are semi-automatic and equipped with local exhaust ventilation. The plunger is also designed in such as way as 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.

In total it has been estimated that the total time spent on maintenance in a year for the three production lines is between 20 and 40 hours per year. The PPE worn depends on the type of

maintenance being carried out, but is a minimum of gloves, hard hat, safety spectacles, safety shoes and coveralls.

In total (including operators and supervisors, lab personnel and maintenance workers), there are approximately 30 people who could be potentially exposed to TCPP in this plant.

Operators monitored were involved in production and blend drumming (one operation of blend drumming was monitored; blend contained 10% TCPP). For inhalation monitoring, the method used was Akzo Nobel Method CG/6.089.3. Samplers were run at 1 L/min ± 10% in the breathing zone. The sample tube (XAD-2) 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 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 by industry for TCPP. The method for determination of TCPP concentration is Akzo Nobel Method CG/6.089.3. The limit of detection was evaluated to be 0.1 μg for TCPP and 3 μg on cotton gloves.

Table 4.1 below gives a summary of the results for the inhalation monitoring and **Table 4.2** summarises the results for the dermal monitoring.

Table 4.1 Results of personal inhalation monitoring carried out on operators involved in production of TCPP and blend drumming

Operator's Task	Length of time monitored (mins)	Inhalation exposure TCPP (μg/m^3)	8hr TWA (μg/m^3)
Production	500	8.2	8.2
Blend drumming	177	1.6	0.6

Table 4.2 Results of personal dermal monitoring carried out on operators involved in production of TCPP and blend drumming

Operator's Task	Length of time monitored (mins)	Dermal exposure TCPP (mg/kg bw)
Production	500	0.02
Blend drumming	177	0.20

During the monitoring period (for both dermal and inhalation), the production operator supervised the production of 3 batches, pumped TCPP into the tank and sampled TCPP three times (including from the funda filter and from the tank). During these activities, he wore protective gloves (Vygen plus PVC gloves, cotton lined). The operator carrying out the task of blend drumming filled 23 drums of 300 kg each for a period of 3 hours (this was equivalent to 690 kg of TCPP). He also attached labels to the drums. He was monitored for 177 minutes (3 hours), which is the length of time taken to carry out his work with TCPP. For the remainder of his shift he worked at the drumming station, but handled substances other than TCPP. He did not wear PPE while carrying out these tasks. Industry has indicated that

theoretically, an operator could be working with TCPP for a full 8-hour shift, depending on requirements.

In parallel to the personal monitoring, a static measurement, with the same equipment as for personal monitoring, was performed. In the TCPP plant, the static monitoring was carried out near a sampling valve; one sample of TCPP was taken during the monitoring period. The monitoring period was for 300 minutes (5 hours). This static measurement gave a concentration of TCPP of 4.8 μg/m^3 (this would correspond to an 8hr TWA also of 4.8 μg/m^3). However, it is likely that the source of the TCPP would have been episodes of short-term exposure during sampling with long periods of minimal or no exposure. These short term exposures would be made up of a series of peak exposures as the valve is opened and the sample drawn, with the concentration falling and rising depending on the proximity of the operator to the valve and the prevailing weather conditions If the only source of TCPP in this area was from sampling, the short-term exposure may have been 1.44 mg/m^3 (one sample collected taking 1 minute) over the period of sampling, with peaks of higher exposure. Industry have indicated that in this plant, the usual maximum for carrying out any particular function on the TCPP plant is twice per shift, so an operator would not normally be in the monitored area more than twice during his shift.

Production plant 2

In a second TCPP production plant, personal inhalation exposure of operators was measured by industry (2002). The method used for measuring TCPP was the same as that described for plant 1 above. The method (Akzo Nobel Method CG/6.089.3) was validated by industry specifically for TCPP. The exposure monitoring was carried out by an external, authorised and certified analytical laboratory. Monitoring was carried out on the chemical production area and the 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 (i.e. monitoring was for an 8 hour shift).

In this plant, TCPP 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. There are approximately 30 operators potentially exposed to TCPP in this production plant.

The monitoring indicated that the operator working in production and quality control was exposed to an airborne concentration of TCPP of 28 μg/m^3. This operator is reported to spend at least 80% of his 8-hour shift in the production hall. As the manufacturing system is closed, most of his exposure will come from quality control sampling. The operator takes several samples from the reactor, the washer, the dehydrator, the check tank and the storage tank. His total exposure time could be as much as 150 minutes per day from sampling and analysis. The second operator involved in the drumming of the final product into steel drums and IBCs was exposed to an airborne concentration of TCPP of 1.8 μg/m^3. This operator also takes quality control samples, but only 1- 5 per day, each taking a few seconds. Although there is LEV at the drumming point there may be some exposure in this area. Industry has indicated that the operators spend 80-90% of their shift in the work area, so the monitoring results obtained can be taken as 8hr TWAs.

There are 4 maintenance personnel on site, who work in conjunction with maintenance contractors, suppliers etc. It is estimated that up to 10 people may be exposed to TCPP in relation to their maintenance work activities (industry information). They may spend up to 7

hours per day carrying out work that could expose them to TCPP. They work under a permit to work regime and there are systems in place to ensure that pipework/vessels are purged prior to maintenance work. The personal protective equipment worn depends on the type of work being carried out but would include helmets, goggles and coveralls, and may also include gloves and respiratory protective equipment as required.

Production plant 3

In a third manufacturing facility, industry measured inhalation exposure to TCPP in the production plant (2002). Measurements were taken in accordance with TRGS (German Technical Rules for Hazardous Substances) Rule 402 to determine the concentration of substances in the air in working environments. The method used for measuring TCPP and analysis was comparable to that used in the monitoring at the 2 plants previously described.

At this production plant, some of the equipment is in an open-air plant and some in a closed building with ventilation (8 air changes per hour). The equipment is operated from a measuring station. TCPP is produced continuously in what industry has described as a substantially closed system. The manufactured TCPP is conveyed to receivers in the basement via fixed pipelines and from there to the storage tank. This is a closed transfer system. The product is decanted into drums, polyethylene containers and road tankers, as required. Drums and polyethylene containers are filled automatically by siphoning. The operator stages empty containers and monitors filling from a control console. Filling time depends on the order, but can last an entire shift. Road tankers are filled via fixed pipeline and a loading spout. The lid on the top of the tanker is covered by a conical hood through which the filling pipeline, level indicator and the pipe for displaced air are fed. (open-air). While the tanker is being filled, the operator performs follow-up and completion work (time < 15 mins). Samples are taken using an open flask (4 samples every 2 hours) by the operator during inspections for unit monitoring (time < 1 min). During filling and sampling the worker wears coveralls, safety glasses, safety shoes and helmets. A laboratory worker takes a sample from the pure product containers twice a day. The sampling time is < 2 mins and analysis takes about 15 mins. These samples are taken using an evacuated flask which is attached to the sampling point via tubing. There is a slight chance of exposure when the flask is withdrawn from the sampling point. Laboratory staff wear coveralls, gloves, goggles and respiratory protective equipment while taking samples. The analysis takes place in a fume cupboard. While carrying out the analysis the laboratory worker wears coveralls, gloves and goggles.

One plant operator and one laboratory worker were monitored (a total of 6 measurements were taken, 3 per person). The plant operator carried out sampling and plant analysis during the monitoring period. The operator was monitored during a full working shift on 3 occasions, on 3 separate days. The laboratory worker was monitored during sampling from pure product receivers (sample taken using an evacuated flask on the riser of the receiver) and during analysis (carried out in a fume cupboard). He was monitored for a short time period on 3 separate days. This time period that he was monitored for was the only time during which he could be exposed to TCPP during his shift (he could only be exposed to TCPP during sampling). **Table 4.3** below gives a summary of these monitoring results.

Table 4.3 Results of personal inhalation monitoring carried out on production operator and laboratory technician involved in production of TCPP

Operator monitored	Length of time monitored (mins)	TCPP (μg/m^3)	8hr TWA TCPP (μg/m^3)
Plant operator, day 1	460	< 50	< 50
Plant operator, day 2	480	< 50	< 50
Plant operator, day 3	460	< 50	< 50
Lab Technician, day 1	15	< 50	< 25
Lab Technician, day 2	20	< 50	< 25
Lab Technician, day 3	26	< 50	< 25

Note: 50 μg/m^3 was the limit of detection

As per the guidelines given in the TGD, the 8hr TWA for the laboratory worker was calculated as half the limit of detection.

In addition to the personal monitoring described above, static measurements were also carried out in this plant. A total of 4 static measurements were taken on 4 separate days in the region of the pure product receivers (at the outlet nozzle or in the line upstream of TCPP) at a height of 150 to 180 cm. The sampling time was on average 6 hours each day. The measured values were all less than 10 μg/m^3 (10 μg/m^3 was the limit of detection).

Production plant 4

One other production company monitored for potential worker exposure during the production of a flame retardant blend containing 50% TCPP. TCPP was mixed with one mass-equivalent of another flame retardant. The plant is a closed system, where the raw materials are pumped via pipes to the mixing vessels and from there to storage tanks. The operator spends about 50% of his time in a control room from where he monitors the process. The remaining 50% of the time, he spends in the plant.

During the process of blend production, overpressure is released via a safety valve. It occurs when the storage tanks are being filled, an event which occurs once daily (max) and takes about 10-15 mins. The TCPP concentration in the release air was monitored twice (both times for 4 hours) and the personal exposure of the worker running this operation over 4 hours was monitored once (there is only one operator involved in this work at any one time). The release of air via safety valves occurs at a level about 3-4 metres above the head of the operator. Industry has indicated that during this time, the operator is located in the control room, monitoring the process. Quality control samples are taken twice per day. The operator wears gloves when taking samples, with respiratory protective equipment available if required. Following manufacture about 50% of the blend is distributed exclusively by road tankers with the other 50% being transferred by pipeline for polyol blending. The TCPP blend is transferred via an automatic pumping station to the road tankers so there is little opportunity for exposure.

There is no daily maintenance carried out on the plant. Planned maintenance is carried out about once per year. Prior to maintenance starting, the TCPP is pumped out of the pipelines and the pipelines are flushed through with water. Checks are carried out to ensure that the OELs for methyl oxirane and phosphorus oxy-chloride are met. Maintenance staff is equipped

with chemical suits, goggles and nitrile rubber gloves to carry out their work. There are no sampling data for this activity.

Details on the analytical method were provided to the company by an EU polyurethane company and modified slightly. Briefly, air was passed through a silica-gel tube and the adsorbed TCPP desorbed with methanol, applying ultrasound for 10 mins. The methanol, containing the desorbed TCPP, was injected into a gas chromatograph and the detection performed via pulse flame-photometric detector.

The results show that the concentration of TCPP in the release air was <80 μg/m^3 while the operator was exposed to an airborne concentration of <8 μg/m^3 TCPP. This value is taken as an 8 hr TWA.

Summary of measured inhalation exposure data

Exposure monitoring was performed at all 4 TCPP production plants. For the measured data, there are few data points from each study carried out in each plant. 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 TCPP production industry. In the first plant, both inhalation and dermal monitoring was carried out, while in the other 3 plants, only inhalation monitoring was performed. **Table 4.4** below gives a summary of the measured inhalation exposure data.

Table 4.4 Summary of results of personal inhalation monitoring carried out on operators involved in TCPP production in the 4 EU TCPP production plants

Production Plant	Activity	8hr TWA TCPP (μg/m^3)
1	Production	8.20
	Blend Drumming	0.60
2	Production and Quality Control	28.0
	Drumming	1.80
3	Production	< 50.0
	Laboratory Testing	< 25.0
4	Blend Production	< 8.0

Summary of measured dermal exposure

For dermal exposure, measured in plant 1, an operator involved in production was exposed to 0.2 mg/kg bw TCPP while an operator involved in blend drumming was exposed to 0.2 mg/kg bw. The production operator wore protective gloves while carrying out his tasks, while the operator involved in blend drumming did not.

Modelled dermal exposure data

For workers involved in the manufacture of TCPP, 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 TCPP 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 $mg/cm^2/day$.

For drumming of TCPP and TCPP 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 $mg/cm^2/day$. 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.

Values taken forward to risk characterisation

For inhalation exposure, the reasonable worst case (RWC) taken forward to risk characterisation is 25 $\mu g/m^3$. This value is half the limit of detection from plant 3, in line with TGD guidance. It is likely that, generally, exposure levels are lower, although there was one higher result of 28 $\mu g/m^3$. It is taken in preference to the SVC of 0.19 mg/m^3, as the SVC does not appear to be realistic, when all of the measured data, both personal and static, from all production facilities, is taken into account. The typical inhalation exposure level to be taken forward to risk characterisation is 12.5 $\mu g/m^3$. This value was taken, as it is half the reasonable worst-case scenario, yet is still a somewhat precautionary value.

For dermal exposure, the reasonable worst case taken forward to risk characterisation is the EASE estimate of 1 $mg/cm^2/day$. This is for the processes of sampling and drumming during the production scenario. It is estimated that the area of exposure would be 210 cm^2. The RWC is therefore 210 mg/day. For typical exposure a value of 0.1 $mg/cm^2/day$, which is the lowest value predicted using EASE modelling, but still higher than the lower of the two real values obtained (assuming an 70 kg man and the area exposed is 210 cm^2). The typical dermal exposure is therefore 21 mg/day. Both of these estimates are higher than the real data obtained, but as there were only two data points it was decided to err on the side of caution.

4.1.1.1.2 Scenario 2: Occupational exposure during the manufacture of flexible PUR foam

6800 tonnes of TCPP was used in the production of flexible foam in Europe in the year 2000 (18% of total TCPP use). It is known that the vast majority of TCPP is added directly by foamers, although some systems are sold into this sector. TCPP is used in slabstock foam for upholstery and mattresses for the UK market. TCPP tends not to be used in the automotive industry owing to its potential for fogging.

TCPP is delivered to the foam manufacturers via road tankers in about three 20 tonne loads per year. In the UK however, the delivery frequency may be higher for large foamers (e.g. 1 road tanker per week). Unloading takes about half an hour and is direct to the storage tank. There was no information about the type of pipe connections and the potential for exposure during connecting and disconnecting pipe-work. One quality control sample is taken per year from an outlet tap before the filter. In the UK it is common practice that a sample is taken at every delivery. When taking the sample the person is equipped with gloves and safety glasses.

Slabstock 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 about 20^0C) 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, 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 TCPP used depends on the foam grade and is controlled by a meter. The range of TCPP used in flexible foam varies from 0 to 15%. 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 exposure during slabstock foam manufacture are at the mixing head where all ingredients are added and mixed together and when operators have to enter the tunnel to carry out various duties, such as controlling foam start-up and removing base paper or polythene. Exposure can also occur at the end of the foaming track during supervision of the block cut-off area. At the beginning of the production process, in order to form a barrier for the liquid and to ensure block shape from the very beginning, two operators may enter the tunnel to hold up a board. They remain in the tunnel until the foam is solid enough to be self-supporting. This typically takes 4 minutes. Due to the presence of isocyanate vapours, the operators wear PPE (including RPE) during this work. Some of the newer machines are equipped with automated start boards, which can reduce operator access to the foaming tunnel but does not eliminate it completely. Some machines operate by a wet purging of chemical streams prior to the start of foaming and at the end of a foaming run. This requires an operator to hold a bucket (or bag) under the mixing head to catch the first and last few kg of the formulation. The time taken for this is very short (typically 5 seconds) and PPE /RPE is worn on both occasions. Because foam machines can vary across the EU it is difficult to estimate the occurrence of these procedures, but in any case the tunnel area is always enclosed and extracted for the control of isocyanate emission from the production process.

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 if the operators have to enter the tunnel. In “automatic” (i.e. bigger) plants, operators hardly ever come in contact with TCPP. The only possibility for dermal contact is when they close the valve on the delivery truck. During this, heavy rubber gloves with sleeves, as well as a face-protecting shield are used. In smaller plants, either IBCs or drums are used for TCPP storage. Here, potential dermal contact exists if dripping occurs from the end of the pump used to empty the container. Again, heavy rubber gloves are routinely used during this work.

Measured inhalation exposure data

Studies have been carried out by industry in 2 plants to determine the inhalation exposure of operators to TCPP during flexible polyurethane foam production. Inhalation exposures were evaluated under actual working conditions. The study was conducted at 2 industrial sites located in the UK - one involved in foam production and cutting and the other one in foam production exclusively. Industry has indicated that the operations monitored were typical of a working day and no event occurred which might have affected the results. A total of 11 operators were involved in the studies in the 2 plants. They were monitored as they performed their tasks. 7 operators on the production line, 2 operators in the cutting area, 1 operator involved in foam sampling or cutting and 1 technician in the QC laboratory were monitored.

In parallel, some static measurements with the same equipment as for personal monitoring were performed in different areas of the plants. The method used to measure TCPP was the same as that described previously for inhalation exposure during production of TCPP. The limit of detection for TCPP was assessed at 0.1 μg TCPP for inhalation.

Table 4.5 gives a summary of the activities monitored during the study, the PPE worn by the operators and the results of the inhalation monitoring. **Table 4.9** gives a summary of the dermal exposure monitoring results. Again, the results for the cutting operations are used in the next scenario – cutting of foam. The results for the cutting operation are used in the next scenario – cutting of foam. There was no inhalation monitoring result available for the laboratory technician.

In addition, personal sampling data from the manufacture of foam using TDCP and V6 have also been used to determine RWC and typical exposures for both inhalation and dermal exposure. These data are presented in **Tables 4.6, 4.7, 4.8, 4.10, 4.11** and **4.12**.

Table 4.5 Summary of activities monitored, PPE worn and results of personal inhalation monitoring carried out on operators during production 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))
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

*The foam was slightly heated when sampled. On consulting industry about the possible temperature of the foam, they indicated that the centre of a 60m block of foam is at approximately 50^{0}C after 48hrs. The sample will cool somewhat during transport around to the cutting area and will have a temperature gradient down to ambient at the outer skins. As a reasonable estimate by industry, it is predicted that approximately 50% of the foam cut by sampler will be around 45^{o}C. Although this will theoretically increase the SVC of liquid TCPP slightly, it is unlikely to increase volatilisation significantly due to restricted diffusion through the foam bulk.

Results of static measurements taken in both plants around the mixing head area indicated a concentration of 7.0 μg/m^3 (plant 1) and 9.5 μg/m^3 (plant 2) TCPP. The static measurement was made on the platform, near the mixing head. In both plants, the measurements were made on the platforms at the height of the breathing zone of the worker. Operator 1 and operator 5

remained on the platform for the duration of the monitoring period. The sampling duration for the static measurement in plant 1 was 409 mins (6.8 hrs) and was 160 mins (2.7 hrs) in plant 2. The measured values are 8hr TWAs.

Table 4.6 Inhalation exposure to TDCP at Plant A during the production of PUR foam

Job title or work area	n	Inhalation TWA 8 h (µg/m^3)
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 4.7 Inhalation exposure to TDCP at Plant B during the production of PUR foam

Job title or work area	Inhalation TWA 8 h (µg/m^3)
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 4.8 Inhalation exposures to V6 at Plants X and Y during the production of 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

Summary of measured inhalation exposure data

Inhalation exposure monitoring was carried out at 2 flexible PUR foam production plants. 8 hr TWAs ranged from 3.6 µg/m^3 for an operator working in the mixing head area of plant 2 to 30.5 µg/m^3 for an operator working in the cut off area of plant 2. Overall, there were significant differences between exposure levels in both plants. In addition, personal inhalation sampling data from flexible foam manufacturing plants using TDCP 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.

Measured dermal exposure data

Dermal exposure of operators during flexible PUR foam manufacture was also measured in these studies. The results of personal dermal monitoring are given in **Table 4.9** below.

Table 4.9 Results of personal dermal exposure monitoring carried out on operators involved in production of PUR foam

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

The highest exposure level was found in operator 1 who was in the mixing head area. He often wore protective gloves, which appeared to be rather contaminated during the monitoring period. He also had slight contact with the blocks by touch to the cut face to assess the characteristics of the foam at the end of the tunnel. It was noted that during the study, he did this without protective gloves. Industry has indicated that it is not normal for an operator to routinely touch the face of the block for control of the foaming process, as there is a hand held airflow measurement device available which gives all the information required. However, as the operator did touch the face of the block during this study, while industry has indicate it is not normal practice, it will be taken as the reasonable worst case for this scenario

It can be noted that the operators in the second plant were monitored for 4 hours only, for both inhalation and dermal monitoring. This corresponds to the foaming time. Industry has indicated that during the remainder of their shift they would be involved in other tasks where they are not exposed to TCPP.

The tasks of the operator from the quality control laboratory included sample preparation, flammability testing, density and hardness measurements and tensile preparation and pulling. He did not wear PPE during these activities. Very low hand exposure to TCPP was detected for this operator (0.003 mg/kg bw).

Table 4.10 Dermal exposure to TDCP at Plant A during the production of PUR foam

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 4.11 Dermal exposure to TDCP at Plant B during the production of PUR foam

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 4.12 Dermal exposure to V6 at Plants X and Y during the production of 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

Summary of measured dermal exposure data

The highest dermal exposure level, 1.5 mg/kg bw, was observed with the operator from plant 1 who touched the face of the block. This is considered to be a worst-case exposure level as it not considered routine for the operators to do this. Other exposure levels ranged from very low in laboratory workers at 0.003 mg/kg bw to 0.68 mg/kg bw for an operator in plant 1. That operator worked in different areas of the line and also cleaned the tunnel. In addition, personal dermal sampling data from flexible foam manufacturing plants using TDCP 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^{-5} to 0.25 mg/cm^2/day assuming an exposure area of 420cm^2.

Values taken forward to risk characterisation

For inhalation exposure, the reasonable worst case taken forward to risk characterisation is 5.1 µg/m^3. This was the 90th 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 µg/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 420cm^2. 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.

4.1.1.1.3 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 saws. In the UK alone, there are hundreds of foam cutters. Therefore, the potential number of workers exposed is extensive.

Measured inhalation exposure data

There is some monitoring data available for cutting of foam containing TCPP. The data was produced from an industry study carried out in a plant that manufactures and cuts flexible polyurethane foam containing TCPP. Foam cutting during the study consisted of continuous deformation cutting of a foam sheet into 2 finished sheets with a convoluter. TCPP had been previously incorporated into the foam at the production stage. The % content of TCPP in the foam was 11.3%. During the monitoring period a total of 23 rolls of foam were cut. This amounted to 1161 kgs of foam; in total, 131 kgs of TCPP were handled.

The result from the personal inhalation monitoring (the method used for monitoring was the same as that described previously for monitoring for TCPP levels during TCPP production) indicated that the one operator monitored was exposed to an airborne concentration of TCPP of 5.4 μg/m^3. The operator was only monitored once, for a duration of 135 mins. For the rest of his shift he carried out activities (such as loop slitting of ester foam) during which he was not exposed to TCPP. His main tasks included putting on the roll of foam and guiding it to the convoluter. He removed and packed the finished foam rolls. During the task, the operator did not wear any RPE. His calculated 8 hr TWA for this is 1.5 μg/m^3.

Static measurements were also carried out near the convoluter and this monitoring indicated an airborne concentration of TCPP of 5.5 μg/m^3 in this area (monitoring period was for 143 mins). This data point was not used in the determination of the value taken forward to risk characterisation. **Table 4.13** below gives the results of this static monitoring.

Table 4.13 Results of static monitoring carried out near the convoluter during the cutting 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)
Operator at convoluter	Convoluter	None	135	5.4	1.5
Static sample at convoluter	Convoluter	Not applicable	143	5.5	Not applicable

In addition, data from cutting of foam containing TDCP and V6 have been used. 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 TCPP data. **Tables 4.14** and **4.15** give the results of this personal monitoring.

Table 4.14 Results of personal monitoring during the cutting of flexible foam containing TDCP

Plant identification	Job title or work area	n	InhalationTWA 8 h (μg/m^3)
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 4.15 Results of personal monitoring during the cutting of flexible foam containing V6

Plant identification	Operator	n	Inhalation TWA 8 h (µg /m^3)
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

Measured dermal exposure data

Dermal exposure monitoring was also carried out and the results obtained indicate that the two operators monitored (one of them being the operator monitored for inhalation exposure, as described above), were exposed dermally to concentrations of TCPP of 0.017 mg/kg bw and 0.28 mg/kg bw. They each were monitored once, for a period of 130 mins and 135 mins, respectively. The results are given in **Table 4.16** below. Both operators carried out the same tasks as described for the operator monitored for inhalation exposure above and neither of them wore any PPE while carrying out their tasks. The foam being cut was produced one week beforehand. It can be noted that the results obtained for both operators were quite different. They each performed the same tasks, were monitored for more or less the same time period and neither wore PPE. There appears to be no any obvious reason for the difference and when queried, industry could not offer any likely explanation.

Table 4.16 Results of dermal exposure monitored carried out during the cutting of flexible PUR foam

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

In addition, data from cutting of foam containing TDCP and V6 have been used. The activities are the same and there is the possibility of dermal exposure to dust from cutting foam containing flame retardant. It is therefore considered valid to utilise these data to supplement the TCPP data. **Tables 4.17** and **4.18** give the results of this personal monitoring.

Table 4.17 Results of dermal exposure monitoring carried out during the cutting of flexible foam containing TDCP

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 4.18 Results of dermal exposure monitoring carried out during the cutting of flexible foam containing V6

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

Values taken forward to risk characterisation

The RWC for inhalation exposure during machine cutting is 4.1 μg/m^3. This is the 90th percentile for the real data for TCPP, TDCP and V6 combined. The typical exposure value to be taken forward is 1.9 μg/m^3. This value is the median value for the real data for TCPP, TDCP and V6 combined.

For dermal exposure the RWC value to be taken forward for risk characterisation for machine cutting is 3 mg/day; the 90th percentile for the real data for TCPP, TDCP and V6 combined. This is equivalent to 7.1 x 10^{-3} mg/cm^2/day, assuming an exposure area of 420 cm^2. The typical exposure value to be taken forward is 0.41 mg/day or 9.8 x 10^{-4} mg/cm^2/day. This is the median value for the real data for TCPP, TDCP and V6.

4.1.1.1.4 Scenario 4: Occupational exposure during the production of foam granules and rebonded foam

TCPP is present in off-cuts of slabstock foam, which undergo rebonding. However, there may be foam containing other flame retardants (V6 or TDCP) as the scrap foam for recycling will come from many different sources. However, TCPP is the most common flame retardant in use. Scrap foam can be shredded and granulated for use as a loose crumb and used in deep-buttoned soft-cushions for garden furniture and some low grade furniture applications. The shredding and granulating processes do not introduce new TCPP.

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.

There is no real monitoring data available for this process, but monitoring was undertaken at two plants which manufactured flexible foam and it is thought that the results of monitoring of operators handling new foam as it comes out of the tunnel are relevant.

Measured inhalation data

There are two data points from manufacture of flexible foam that are considered relevant. They were 9.4 μg/m^3 and 17 μg/m^3, which translated to 4.6 μg/m^3 and 14.2 μg/m^3 8hr TWA respectively. These results were for operators handling foam as it came out of the tunnel and taking samples of the foam as it was cooling.

In addition, some data have been taken from relevant operations during the manufacture of foam containing TDCP and V6. These data are presented in **Tables 4.19** and **4.20**.

Table 4.19 Data from relevant operations during the manufacture of foam containing TDCP

Job title or work area	n	Inhalation TWA 8 h (µg/m³)
Cut-off area	2	<0.2, 1.7
Cut-off block operator	1	<0.20
Cut-off Start/End operator	1	<0.20

Table 4.20 Data from relevant operations during the manufacture of foam containing V6

Job title or work area	n	Inhalation TWA 8 h (µg/m³)
Block Cutter	2	<0.64,<0.59
Cut Off Block	1	<0.59
Cut Off Start/End	1	<0.58

Measured dermal exposure data

There are two data-points from manufacture of flexible foam that are considered relevant to this scenario. They were for the operators handling foam as it came out of the tunnel and taking samples of the foam as it was cooling. The results were 0.01 mg/kg bw and 0.003 mg/kg bw respectively. These results equate to 1.7 µg/cm^2/day, (or 0.714 mg/day), and 0.5 µg/cm^2/day, (or 0.21 mg/day), assuming 70 kg body weight and an exposed area of 420 cm^2.

In addition, some data have been taken from relevant operations during the manufacture of foam containing TDCP and V6. These data are presented in **Tables 4.21** and **4.22**.

Table 4.21 Data from relevant operations during the manufacture of foam containing TDCP

Job title or work area	n	mg TCPP / pair of gloves (mg/day)
Cut-off area	1	27.0
Cut-off block operator	1	0.173
Cut-off start/end operator	1	0.124

Table 4.22 Data from relevant operations during the manufacture of foam containing V6

Job title or work area	n	mg TDCP / pair of gloves (mg/day)
Block cutter	2	0.14, 0.28
Cut-off block	1	0.34
Cut-off start/end	1	0.23

Values taken forward to risk characterisation

The RWC taken forward for inhalation exposure is 4.6 µg/m^3 8 hr TWA. This is the 90th percentile value from all the data presented. The typical exposure value taken forward is 0.59 µg/m^3 8 hr TWA, which is the median value of all the results presented.

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.

4.1.1.1.5 Scenario 5: Occupational exposure during the formulation of systems and manufacture of spray foams

As outlined in section 2.2.2.1.1, some PUR producers purchase pre-mixed, ready to use systems. PUR systems consist of component A, the polyol component containing amongst other things, the flame retardant, and component B, the isocyanate component. TCPP is added to polyols in the formulation of PUR systems. In the year 2000, 16,600 tonnes of TCPP was used in the production of PUR systems. An estimated 75 to 80% of PUR systems are manufactured and supplied by the four major raw material manufacturers. There are at least 50 small to medium sized systems houses in the EU and an industry survey has shown that the processes and controls in place are similar to the big integrated systems houses operated by the major chemical producers.

Of the 16,600 tonnes of TCPP used in 2000, over 3,850 tonnes was used by the systems houses in the manufacture of spray foams. Industry has indicated that the process of the production of systems for spray foams is identical to that for other foams, so the occupational exposure during the manufacture of spray foams is considered in the same scenario.

Measured inhalation exposure data

Plant 1

In this first study carried out in a polyol formulating facility, a total of 5 personal measurements were taken. In this plant, production is discontinuous (batch-wise). The plant is equipped with ventilation equipment. TCPP is transferred from tankers in the open air and into the storage tanks suspended in gas. The operator performs follow-up and completion work (each procedure takes about 10 mins), monitors the transfer and takes a sample from the transfer line (less than 1 min), via outlet tap, into a container. The product is transferred via lines with connections having the minimum of dead spaces. Filters in the transfer lines are checked once or twice a year and any necessary maintenance to filters carried out (open air plant; time < 15 mins). The quantity-related metering of products used for production uses closed system piping. The operator monitors metering (adjust valves), plant (visual inspection via inspection window) and takes samples (from each receiver and section – time 5 min/sample). The receivers are emptied after analytical release. The operator connects the pipes (about 10 mins/receiver). The formulations are then decanted into tanks, drums and 1 m^3 IBCs. For the filling of containers (drums or IBCs), siphoning is carried out using nozzles fitted with local exhaust ventilation (LEV). The operator places the filling device in the container, monitors filling and changes and seals the container. On completion of filling, the transfer pipe is scraped and the scrapings disposed of.

The method employed for measuring TCPP was the same method as has been previously described. Briefly, air samples were collected at 1 L/min for up to 240 litres XAD-2 OVS

sampler tubes. The air concentration range studied was approximately 0.05 ppb v/v to 5 ppb v/v for 240 litre air samples. The filter and front sorbent section were desorbed together in toluene. The backup section was desorbed separately in toluene. Sample solution was analyzed by GC with a nitrogen phosphorous detector. Dupont constant flow sampler pumps were used and the sampling equipment was prepared by the plant's hygiene unit.

Three measurements were taken while the operator was monitoring the plant, including sampling. Two other measurements were taken while the operator was decanting TCPP-containing polyol formulations into drums and then scraping the tube. All measurements were taken on different days. The results of the monitoring are given in **Table 4.23** below.

Table 4.23 Exposure levels to TCPP during formulation of polyols containing TCPP

Activity Monitored	Monitoring Time (mins)	TCPP (µg/m³)
Plant operation, including sampling	510	<5
Plant operation, including sampling	435	<5
Plant operation, including sampling	458	<5
Decanting of TCPP containing formulations, including scraping of tube	320	<5
Decanting of TCPP containing formulations, including scraping of tube	450	<5

Plant 2

Monitoring was also carried out by industry to determine the exposure to TCPP from open handling of TCPP during the production of the polyol component for rigid foam systems. Details of the analytical method followed were provided to the plant by a European polyurethane company. Briefly, air was passed through a silica-gel tube at a constant flow rate. The adsorbed TCPP was desorbed with methanol, applying ultrasound (the internal standard, tributylphosphane oxide, was added to the methanol beforehand). The sample was analysed by GC, with a pulsed flame-photometric detector.

The work process monitored was the manufacture of a polyol component where TCPP is mixed into the polyol in an open 200L steel drum. During the production of the polyol components, TCPP is transferred from the storage tank via pipeline in the mixing vessel for the polyol component. The formulation of the polyol component is therefore a closed system. Nevertheless, for research and development, small quantities of special polyol components are occasionally required. In such cases the polyol component is mixed in the drum and open handling of TCPP occurs (this was the process that was monitored and as it is open handling, is considered a worst case scenario).

During the monitoring period, the sampling tubes were placed 20 cm above the liquid while TCPP was poured into the polyol and homogenised by a stirrer. Normally, an operator would be about 0.5-2 meters away from the point of pouring the TCPP, so this measurement could be taken as reasonable worst case for this scenario. Two runs were carried out. In the first run, TCPP was mixed into a polyol to a final concentration of 7%. Two drums of polyol were prepared and so 2 samples were taken. For the first drum, a higher volume of air was collected because after addition and homogenisation of TCPP, the stirring was monitored for several minutes. For drum 2 sampling was finalised after the TCPP was homogenised. In the second run, a system was formulated that contained 11% TCPP. The samples were taken in duplicate

and only a short time period for homogenisation was covered. As above, the sampling tubes were located about 20 cm above the surface level of the polyol.

Table 4.24 below gives the results obtained from this monitoring. As the monitoring period was very short, the results obtained can be taken as short-term exposure values.

Table 4.24 TCPP exposure from open handling of TCPP during formulation of polyol component

Run/Sample	Monitoring Time (mins)	TCPP (ppm)	TCPP (mg/m^3)
Run 1; Drum 1	20	0.02	0.27
Run 1;Drum 2	20	0.06	0.80
Run 2;Drum 1	10	0.192 and 0.098	2.57 and 1.31
Run 2;Drum 2	10	0.17 and 0.147	2.28 and 1.97

There was quite a difference in results obtained between the 2 runs. It is considered by industry that raising the TCPP concentration from 7% to 11% is not the reason for this difference. In the preparation of a polyol component the different components are manually poured into the drum and mixed by a drum-stirrer. The monitoring started when the operator opened the TCPP line (the components are supplied by lines, which are connected to storage tanks). After the TCPP is poured into the polyol (about 20 seconds), the polyol component is stirred for a maximum of 10 minutes. During this time, other components are added and homogenised. It is assumed that the main exposure occurs when TCPP is poured into the polyol, rather than while the polyol is being stirred. During run 1, the time of stirring was longer than that in run 2, thus perhaps explaining the difference in results.

Modelled dermal exposure data

No monitoring data is available for dermal exposure to TCPP during the formulation of polyols. Therefore, EASE modelling has been used to estimate this exposure. The appropriate EASE scenario is non-dispersive use with intermittent contact. EASE predicts dermal exposure to be in the range 0.1-1 mg/cm^2/day. The reported range of TCPP concentrations in the mixture was 7 to 11%. The estimated dermal exposure range can therefore be refined to 0.007 to 0.11 mg/cm^2/day. In practice, the dermal exposure will be reduced if the operators wear suitable gloves and change them regularly.

Values taken forward to risk characterisation

There were short-term and 8-hour measurements available from industry for inhalation exposure. As the short-term measurements were static and for "open top" mixing for Research and Development purposes, these have not been considered for risk characterisation. All the long-term samples were personal samples and taken during normal production activities. The value for RWC inhalation exposure to be taken forward for risk characterisation is 5 $\mu g/m^3$. All the values reported for this scenario were <5 $\mu g/m^3$, so this value is taken in the absence of any other meaningful data. The value taken forward for a typical inhalation exposure is half the RWC, at 2.5 $\mu g/m^3$, which is in line with TGD guidance.

The RWC value for dermal exposure to be taken forward for risk characterisation is 0.11 mg/cm^2/day, or 46.2 mg/day, assuming an exposure area of 420 cm^2. This is the highest value estimated using EASE and professional judgement, but in the absence of any other data, the precautionary approach has been adopted.

The figure taken forward for risk characterisation for typical exposure is 0.05 mg/cm^2/day, or 21 mg/day. This is half of the RWC and has been used in the absence of any other data. The area exposed is estimated to be 420 cm^2. This contact area was selected as the description of the process indicted that there was potential for contact, particularly during transfer and post-transfer activities such as scraping the transfer pipe.

4.1.1.1.6 Scenario 6: Occupational exposure during the use of spray foams

Spray foams are used in building construction and maintenance and repair and are not available for use by the general public. They are usually applied *in situ* to walls, roofs, tanks and pipes. The product from one of the key manufacturers of spray foam is a PUR rigid foam with up to 95% closed cell content used as a roof spray. It is produced through the mixing of two liquid components, the A-component (polyol) and the B-component (diphenylmethane diisocyanate – MDI). The mixing of the two components produces a reactive mixture, which forms under heat evolution. The temperature reached in the spray 'gun' is typically 49 to 60^{O}C. At the end of the reaction phase, the foam starts to solidify and cure. The foam is applied by a spray gun in several layers. Within a few minutes, the foam is cured and hard enough to walk on.

Workers from the specialist applicator companies that apply these spray foams may be occupationally exposed to TCPP within the A-component during their work. During this work, the operators wear RPE as they are working with diisocyanates and amine-based catalysts. In addition, the work is normally an outdoor operation. The operators could be engaged in this work for up to several hours a day. There is no measured personal monitoring data available for this process. However, there are data available from the manufacture of rigid foam, which may be used to estimate exposure for this scenario. The manufacture of rigid foam takes place with LEV in use and the foam is covered top and bottom with paper or metal facings. This is not the case with spraying foam onto walls and ceilings. **Table 4.25** below summarises the exposure values measured during rigid foam manufacture which have been used to derive the inhalation and dermal exposures for this scenario.

Table 4.25 Summary table of values used in rigid foam manufacture which have been used to derive RWC and typical exposures for this scenario

Measurement	TCPP	Calculated 8-hr TWA
Plant 1 - Operator – product feed side	< 5 μg/m^3	<5 μg/m^3
Plant 1 - Operator – removal of sheets	< 5 μg/m^3	<5 μg/m^3
Plant 1 - Lab Technician	< 5 μg/m^3	<0.27 μg/m^3
Plant 3 – Operator	<20 μg/m^3	<20 μg/m^3
Plant 5 - Laydown operator	< 0.3 mg/m^3	<0.3 mg/m^3
Plant 5 - Laydown operator	< 0.3 mg/m^3	<0.3 mg/m^3
Plant 5 - Laydown operator	< 0.2 mg/m^3	<0.2 mg/m^3

The values taken forward for risk characterisation for manufacture of rigid foam were 150 μg/m^3 for RWC and 20 μg/m^3 for typical exposure. For application of these foams, a RWC is considered to be 300 μg/m^3, with a typical exposure of 40 μg/m^3. These values are proposed taking into account the differences in the application of the foam and the controls in place. However, according to industry information provided, it is unlikely that sprayers would be

spraying foam all day. If a precautionary figure of 5 hours spraying per day is used, the exposure estimates are refined to 187.5 μg/m^3, 8-hour TWA and 25 μg/m^3, 8-hour TWA. EASE cannot be used to estimate exposure to low volatility liquids as EASE has limitations in estimating the inhalation exposure to such a substance.

Modelled dermal exposure data

In the absence of any dermal exposure data for this task, EASE was used to estimate a range of exposures. The parameters used were inclusion onto a matrix direct handling and intermittent contact, which gives an exposure range of 0.1 to 1 mg/cm^2/day. The reported range of TCPP concentration in rigid foams is 2 to 23 % (see Section 4.1.1.1.7). Thus the estimated range of dermal exposure can be refined to0.002 to 0.23 mg/cm^2/day. It is estimated that an area of 420 cm^2 could be exposed.

The parameter inclusion onto a matrix was used in this scenario rather than wide-dispersive use, to take into account the fact that the spraying is not conventional spraying of liquids (on which EASE is based), but of fast coagulating and solidifying foam, so the opportunity for dermal exposure would be lower.

Values taken forward to risk characterisation

For RWC inhalation exposure a value of 187.5 μg/m^3 is taken forward for risk characterisation, with a typical inhalation exposure value of 25 μg/m^3.

For dermal exposure a RWC value of 0.23 mg/cm^2/day, or 96.6 mg/day is taken forward for risk characterisation. A typical exposure value of 0.12 mg/cm^2/day, or 50.4 mg/day is taken forward for risk characterisation, which is half of the RWC and in line with TGD guidance. The area of skin exposed is estimated to be 420 cm^2.

4.1.1.1.7 Scenario 7: Occupational exposure during the manufacture of rigid PUR foam

26,650 tonnes of TCPP were used by rigid foamers in the production of construction products in the year 2000. ISOPA has indicated that there are about 190 rigid foam manufacturers in the EU (ISOPA survey, 2003). Rigid foams are mainly produced as blocks and panels and used for insulation purposes. For PUR insulation foams in general, 90% of the usage of additive flame retardants is currently accounted for by TCPP (Leisewitz A, Hermann K and Schram E, 2001).

Deliveries of TCPP are usually made via road tankers, although one foam producer also receives TCPP in IBCs. Deliveries are approximately weekly and take between 1.5 and 2 hours to offload the 10-20 tonnes. One producer takes a sample from an outlet valve on delivery and retains it for 3 months, but no analysis is carried out. The other producers do not carry out their own quality control sampling but work on certificates of analysis provided by the supplier. Producer 1 reported that the delivery is essentially a closed system, with no-spill pipe-work connectors and pipe-work for removal of displaced air. Producer 2 used flexible EPDM coupling to connect the tanker to the delivery pipe-work. Producer 3 uses a manual connection with a drip tray to collect any spillage on disconnection (approximately 200ml), which is returned to the polyol waste.

It is reported by the manufacturers that the TCPP content in rigid foams is usually in the range 2 – 9%, although in the sampling data sent, the foams have a range of 8 to 23% TCPP. The data set where the TCPP content was 23% was from a research pilot plant and does not reflect current practice on production plants. The range of TCPP content used in EASE calculations is therefore 2-13%.

For the production of PUR rigid foam, diphenylmethane-di-isocyanate is mixed with a polyol component in a mixing head. Driven by catalysts, the reaction starts within seconds while the mixture is poured on a transport belt, shielded by flexible or rigid facings, depending on the type of rigid foam required. The foam rises and cures and after several metres, the foam is sufficiently stable to be cut into blocks or panels.

The occurrence of any TCPP vapour during the production process will be limited and of short duration as the foam cells have to be closed to retain the blowing agent which also acts as the insulating gas. High temperatures (typically in the range 120-140^{0}C) are only reached when the foam cells are already closed and thus any TCPP will be kept within the foam. In the liquid phase, before the cells are formed, the temperature is up to 35^{O}C. Ventilation is provided in the production area as di-isocyanates (MDI) and, often, pentane, are used in the process.

There are some key products associated with PUR insulating foam. These are flexible-faced laminate, sandwich panels and discontinuous panels and full details of these are given in section 2.2.2.3.2.

There are two major differences between the production of flexible and that of rigid PUR foam. The first is the closed-cell nature of the rigid foam and the second is the point that almost all products are covered from the point of manufacture by impermeable or semi-permeable barriers.

The production process involved in the manufacture of flexible-faced laminate generally occurs in a closed system, with only a very short period (seconds) where the chemicals are in the open work environment. It involves the pouring of the foam chemicals onto the lower facing material which is carried by a conveyer belt, the chemicals react, the foam is formed and the upper facing is unrolled to meet the upper surface of the foam. The entire product is conveyed into a curing tunnel and at the end of the process the product is cut to size to be used in buildings. The potential for occupational exposure exists at the mixing head, when operators have to enter the tunnel and when the foam is cut, although the cutting, stacking and packing is all done automatically on the production line.

The production process for sandwich panels is similar to that for flexible-faced laminate except that the steel is supplied in rolls and fed through profiling rollers just before the polyurethane is applied; the product is then cut into lengths automatically (using saws). The potential for occupational exposure during the manufacture of these panels is the same as for flexible-faced laminate described above.

Discontinuous panels are produced by injecting the PUR foam chemicals in between pre-cut steel sheets. Again, the occupational exposure is considered to be similar as for the foams described above.

Measured inhalation exposure data

Production Plant 1. Pilot plant manufacturing PUR foam sheeting

Exposure to TCPP during the manufacture of TCPP-containing rigid polyurethane foam sheeting was measured by industry in a pilot plant of one processing facility (polyurethane foams are produced in the pilot plant for test purposes e.g. for determining applicational parameters). The plant used to produce the foams was located in a closed, ventilated area. Diisocyanate and the polyol preparation are pumped from storage containers via piping to the discharge point (mixing head); the components are then sprayed onto a substrate through the mixing head nozzles at about 30^0C. An extraction hood is installed above the discharge region. The final mixture in this case contained about 23% TCPP. After it is foamed, the PUR sheet is conveyed from the discharge point to the saw within about 5 min and sawn into blocks. The saw comprises chambers and has local exhaust ventilation (no workspace). After the sawing operation, the blocks are removed and stacked on a truck. There is local exhaust ventilation in place at the removal point. Operator tasks during this process include adjustment of the belt speed at the mixing head, monitoring application of the reactants, dismantling the nozzle head at the end of the test and placing the residual material produced in the process in a waste container. A laboratory technician determines various reaction parameters during the run. This person leans into the discharge area during sampling. On the take-off side, an operator removes the sawn-off PUR blocks and stacks them on a truck. During the monitoring period, 3 personal measurements were taken; one from an operator at the product-feed side, a second from an operator during the removal of final sheets and a third from the laboratory technician performing inspection at the laminator. The method used to measure TCPP was the same as that employed at the manufacturing facility (production plant 3 in scenario 1). Each operator was only monitored once. **Table 4.26** below gives the results of this monitoring. In calculating the 8 hr TWAs, it is assumed the operators could perform their tasks for the duration of their 8 hr shifts. The lab operator however performs his task for the time monitored and then would carry out other tasks where he would not be exposed to TCPP.

Table 4.26 Results of personal inhalation exposure monitoring carried out on operators involved in production of rigid PUR foam

Measurement	Monitoring period (mins)	TCPP (μg/m^3)	Calculated 8-hr TWA (μg/m^3)
Operator – product feed side	16	< 5	<5
Operator – removal of sheets	20	< 5	<5
Lab Technician	26	< 5	<0.27

Production plant 2. Plant manufacturing PU-covered polystyrene panels

Static monitoring was carried out by industry during the production of PU-covered polystyrene panels for floor-heating insulation. After pouring of the PU-system, the panels were covered by a glass-fibre textile. The polyol-isocyanate mix contained 12.5% TCPP and had a temperature of 22-24^{0}C. The delivering unit was swinging over the panels so that the distance to the silica-gel tube varied between 30 and 130 cm. For monitoring, the silica-gel tube was placed in such a way that the airflow going from delivery unit to the local exhaust ventilation (LEV) had to pass. In this area, operators put the polystyrene panels on the belt before the panels are covered by the reaction mixture. Industry has indicated that the operators

are 'up-wind' at this stage. In addition, during the process the holes from which the reaction pours can become blocked and the operator has to wipe them clean. This job takes about 10 seconds and would need to be done about 12 times an hour. Therefore, for an 8-hour shift, an operator would spend approx. 16 minutes in total located at this 'hot-spot' next to the mixing head where the static monitoring was carried out. Another sampling point was also located at the end of the tunnel, where the foam cured at a temperature of 44^0C. Operators generally do not spend time in this area, but they do monitor the belt from that area. Industry has indicated that operators will monitor the belt about 3-6 times a shift for a maximum of 30 seconds per event. This means an operator would spend at most 3 minutes in this area.

This product and the method of production have been reported by industry to be atypical. It is reported to be very unusual to have a situation where an operator would have to clear the holes in the mixing head. Modern plants have two mixing heads and switch from one to another if one gets blocked. Mixing heads get cleaned at the end of a run (normally one to two hours). At this point all the foam would be cured, so the possibility of dermal exposure would be very low

The methods employed to determine the TCPP concentrations were as described previously during TCPP production. There was only one measurement taken per monitoring point. **Table 4.27** below gives the results from this static monitoring.

Table 4.27 TCPP exposure during the manufacture of PU-covered polystyrene panels for floor-heating insulation

Monitoring point	TCPP (ppb)	TCPP (μg/m^3)
End of tunnel (1)	7.6	101
End of tunnel (2)	2.2	29
Airflow from delivery unit to LEV (1)	<4.8	< 64
Airflow from delivery unit to LEV (2)	<5	< 67
Airflow from delivery unit to LEV (3)	1.1	14

Production plant 3. Plant manufacturing rigid faced PU panels

Industry monitored one operator for TCPP exposure during the process of manufacturing steel faced PU rigid foam panels. TCPP levels were also measured at different static locations in the plant. The PU-system contained 8% TCPP and was at a temperature of 22^0C. The method employed for determining TCPP concentration was as per the method previously described during production of TCPP. The lay-down air was sampled in duplicate. The operator was only monitored once. As part of the work, the operator watches the lay-down area and controls the polyol to isocyanate ratio, the total amount of PU-system poured on the belt and the proper transport of the steel sheets via computer screens. An operator is generally 2-3 m away from the mixing-head.

The operator was monitored for 60 mins and results show he was exposed to <20 μg/m^3 TCPP. As he would work in this manufacturing area for his 8 hour shift and could be exposed to TCPP at various times through the shift, this can be taken as his 8hr TWA. Results from the static monitoring showed that the concentration of TCPP at the vent at lay down (monitored for 120 mins) was <20 μg/m^3 and TCPP concentration in the lay down area (monitored for 60 mins) was <21 μg/m^3.

Production plant 4. Plant manufacturing flexible faced PU rigid foam panels

Static monitoring was carried out by industry in a plant producing PU rigid foam panels. The PU system contained 13% TCPP. The first monitoring was carried out 10 cm above the mixing head (lay-down) for 120 minutes. The operator was standing about 2 meters away from the mixing head during the monitoring period. The results show that the concentration of TCPP at this point was <20 μg/m^3. Exhaust air at the extraction points of the LEV (at lay down and at the cutting area) was also monitored for the presence of TCPP. The extraction point at lay down was monitored for 130 minutes and results show that the concentration of TCPP was <20 μg/m^3. Static monitoring at the extraction point in the cutting area was carried out for 80 mins and the results show that the concentration of TCPP here was 28 μg/m^3.

Production plant 5

Personal monitoring was carried out on three laydown operators at this plant in 2005. The results are presented in **Table 4.28** below.

Table 4.28 Inhalation exposure results at production plant 5

Measurement	TCPP (mg/m^3)	Calculated 8-hr TWA (mg/m^3)
Laydown operator	< 0.3	<0.3
Laydown operator	< 0.3	<0.3
Laydown operator	< 0.2	<0.2

Summary of measured inhalation exposure data

Of the 5 production plants where monitoring for TCPP was carried out, only 3 of them performed personal monitoring on the operators. The results of the static monitoring are difficult to interpret as the relevancy to operator exposure in terms of location of the operator and overall time spent in the area is difficult to define. Therefore the results of the personal monitoring will be the ones used in this assessment. The calculated 8hr TWA for the operator in production plant 1 was <5 μg/m^3 and <20 μg/m^3 for the operator in production plant 3. In production plant 5 the results were <0.3 mg/m^3 and <0.2 mg/m^3. 0.15 mg/m^{3}will be taken as the reasonable worst-case exposure, which is half of the highest limit of detection in line with the guidance given in the TGD. The typical exposure level will be taken as the median value of the personal sampling results, i.e. 20 μg/m^3.

Dermal exposure data

There are no data for this scenario. According to information from industry, there is very little handling of the foam or the products, as most of the packing is carried out on the automated production lines. However, as there are some plants where handling will still take place, dermal exposure has been modelled to take this into account.

Modelled dermal exposure data

For stacking sheets of cut foam at the take off point, the parameters used were inclusion onto a matrix, direct handling, and intermittent contact. The exposure range predicted using EASE was 0.1 – 1 mg/cm^2/day. Taking into account the range of reported percentage TCPP content (2 –13%), the exposure range for this task is 0.002 – 0.065 mg/cm^2/day. The area of skin

exposed would be very small as most of the skin would be in contact with the sandwich panels rather than the foam within the facings. It is estimated that the area of skin exposed would not exceed 210 cm^2, equivalent to one quarter of each hand. The daily exposure range can therefore be estimated to be 0.42 mg/day to 13.65 mg/day.

Values taken forward to risk characterisation

For inhalation exposure the reasonable worst case to be taken forward to risk characterisation is 150 $\mu g/m^3$. This is because this was half the limit of detection for the highest personal samples from the data provided. The static sampling data did not seem to represent personal exposure given the locations at which the samples were collected relative to where the operators work. A value of 20 $\mu g/m^3$ will be taken forward as a typical exposure concentration, as this was the median value of the seven results considered.

For dermal exposure the reasonable worst case to be taken forward to risk characterisation is 13.65 mg/day or 0.065 mg/cm^2/day, assuming an exposure area of 210 cm^2. This is the highest value in the range for modelled data and is taken forward in the absence of any other data. The typical exposure taken forward to risk characterisation is 6.8 mg/day or 0.032 mg/cm^2/day, assuming an exposure area of 210 cm^2, which is half of the RWC and in line with TGD guidance.

4.1.1.1.8 Scenario 8: Occupational exposure during the use of rigid PUR foam

There is the potential for occupational exposure during the use of rigid PUR foam, by construction workers, especially if they cut the foam on site. Flexible-faced laminates are used in the insulation of the walls and roofs of buildings. This is the only rigid foam that may have to be cut on site by construction workers. While the number of construction workers potentially exposed is extensive, it is considered that the potential for worker exposure is low because the work will generally take place in the open air. In addition the closed cells of the foam would mean that only the first few 100 microns of foam interior is ruptured during cutting. It is also very unlikely that a worker would spend all day cutting foam, as only a small percentage of panels would need to be cut to enable them to fit corners and around obstructions etc. It is most likely that these panels would be cut using a handsaw or by scoring with a knife and snapping. The cutting of the product with a saw will generate some dust. It is unlikely that large amounts of foam would need to be cut on site, but if it were necessary the foam would be cut using a circular saw, probably fitted with extraction (information from construction firm).

The metal-faced panels (sandwich panels) are used to construct many types of buildings, including factories and stores. However, the steel facings on the panels fully protect the core. Therefore, occupational exposure of construction workers to TCPP contained within the rigid foam is considered negligible and will not be further investigated here. Industry has indicated that these panels are cut in the production facility and are not cut by the construction workers on site.

With discontinuous panels, the steel facings on these panels fully protect the core; therefore, the potential for occupational exposure of workers using these panels is negligible. Again, industry has indicated that these panels are cut in the production facility and are not cut by the construction workers on site.

Inhalation exposure data

There are no data available for the cutting of rigid foam. However there are data for cutting flexible foam containing TCPP (see section 4.1.1.1.3 - Scenario 3: Occupational exposure during cutting of flexible PUR foam for details) and these are re-presented in **Table 4.29**, below. It is considered valid to use these data to estimate exposure to TCPP during cutting of rigid foam.

In addition, data from cutting of foam containing TDCP and V6 have been used. 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 TCPP data. **Tables 4.30** and **4.31** give the results of this personal monitoring.

Table 4.29 Results of TCPP monitoring carried out near the convoluter during the cutting of flexible PUR 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
Static sample at convoluter	Convoluter	Not applicable	143	5.5	Not applicable

Table 4.30 Results of personal monitoring during the cutting of flexible foam containing TDCP

Plant identification	Job title or work area	n	InhalationTWA 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 4.31 Results of personal monitoring during the cutting of flexible foam containing V6

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

Modelled dermal exposure data

As no measurements have been made of the exposure of workers to TCPP during the use of rigid PUR foam, the EASE model has been used to estimate exposure. The only scenario where exposure is likely to occur is when construction workers have to cut flexible faced laminates on site. As the foam panels are faced on each side, the opportunity for skin contact is limited. For this reason a small exposure area has been assumed (210 cm^2). The EASE model has been used previously to estimate dermal exposure during the cutting of flexible PUR foam. The estimate for dermal exposure for that scenario was found to be within the range 0.1-1 $mg/cm^2/day$. However, in this scenario the likelihood is that very little cutting of foam will take place on site so for this scenario the parameters used are inclusion onto a matrix, direct handling with incidental contact giving a predicted exposure range of 0 to 0.1

mg/cm^2/day. Incidental contact was used to take account of the fact that most of the handling of the product will be by contact with the flexible facings on the foam rather than the foam itself. Taking into account that the range of TCPP concentrations within rigid PUR foam is reported as 2 to 13%, the predicted exposure range becomes 0 to 0.013 mg/cm^2/day, or 2.73 mg/day, assuming an exposure area of 210 cm^2. In practice, dermal exposure will be reduced if the workers wear suitable gloves correctly and change them regularly.

Values taken forward to risk characterisation

The RWC for inhalation exposure during machine cutting is 4.1 μg/m^3. This is the 90th percentile for the real data for flexible foam containing TCPP, TDCP or V6 combined. The typical exposure value to be taken forward is 1.9 μg/m^3. This value is the median value for the real data for flexible foam containing TCPP, TDCP or V6 combined.

For dermal exposure there was no actual data available for cutting rigid foam. The reasonable worst-case figure carried forward to risk characterisation is 0.013 mg/cm^2/day, or 2.73 mg/day. This figure is used as it takes into account a high percentage of TCPP in the foam and is precautionary in the absence of any real data. A figure for typical exposure taken forward for risk characterisation is 0.006 mg/cm^2/day, or 1.37 mg/day. This is half the RWC figure obtained when estimating using EASE and professional judgement. This figure is proposed in line with guidance in the TGD. It is estimated that the area of dermal exposure will be 210 cm^2. This dermal contact area was selected as although most of the foam is covered, the dust produced when cutting the foam would mean that a larger area of skin would be in contact than that in contact when just handling the foam.

4.1.1.1.9 Scenario 9: Occupational exposure during the manufacture of one-component (1-K) foams

1900 tonnes of TCPP is used in the EU in the production of 1-K foams. Most manufacturers use TCPP directly, but some use pre-formulated polyols. Large producers of 1-K foams receive TCPP in 10 or 20 tonne tanks and the TCPP is unloaded into dedicated storage tanks using dedicated lines. For the large producers, TCPP is pumped from the closed storage-tanks into a closed weighing tank where the product is mixed with polyols. This is done using a computerised batching system. From the weighing tank there is a direct connection with the filling heads of the aerosol machines. In general, ten seconds after filling the aerosol can with the polyol component containing TCPP, the can is closed air tight by the valve. Filling and valve crimpling is carried out in the same cabinet, which is ventilated to the outside. It is considered that the whole process is a closed loop system. (Rhee 2002 and ISOPA 2003).

There are 8 large manufacturers of 1-K foams in geographic Europe. It is estimated that the total workers in the unloading and mixing area is 2-5 per company.

Smaller producers generally buy TCPP in 10-20 tonne tanks and store it in dedicated storage tanks. Metering into the weighing tank is done by manual operating pumps or valves. Some smaller producers buy TCPP in drums or IBCs. The drums or IBCs are elevated above the weighing tank with a forklift and the valve of the tank is opened manually. After this, the steps of the procedure are the same as for the large manufacturers. Some of the very small fillers buy systems in IBCs and connect the IBC directly to the filling head of the filling machine. This system is relatively closed and there is little potential for worker exposure.

Industry has indicated that there are 15 remaining smaller producers in the whole of geographic Europe, of which 3 are located within the EU. Total workers working in the mixing area is estimated to be 1-3 per company.

There is no monitoring data available for occupational exposure to TCPP during the manufacture of 1-K foams.

Inhalation exposure

From descriptions of the process, the only point at which it is possible that there may be any exposure would be during the sampling and analysis of TCPP during delivery or during the addition of TCPP to mixing tanks from IBCs at the smaller manufacturers. There is potential for inhalation exposure during delivery. There is also potential for exposure during analysis of samples. There are some results for sampling and laboratory work in other scenarios and for different flame retardants which are considered appropriate for use here. There may be some differences in the type of work carried out or the frequency with which the work is carried out, but as EASE cannot be used for low volatility liquids, it was felt appropriate to use the small amount of real data available. These data are summarised in **Table 4.32** below.

Table 4.32 Result from laboratory testing work

Activity from which results taken	Exposure TWA 8h (μg/m³)
TCPP production – lab worker	<25, <25, <25
TDCP foam production – plant 1 lab worker	<0.2, <0.2, 1.3
TDCP foam production – plant 2 lab worker	<0.2
V6 foam production – plant 2	<0.6

In the absence of any other data it has been decided that the RWC will be taken as half of the <25 μg/m^3, at 12.5 μg/m^3, 8-hr TWA, with a typical exposure of half that figure at 6.7 μg/m^3.

Dermal exposure

Again, there are some real data available for sampling and laboratory work from other scenarios and for other flame retardants which are considered appropriate for use here. These data are summarised in **Table 4.33** below.

Table 4.33 Dermal exposure for laboratory work with foam containing TCPP, TDCP and V6

Activity from which results taken	Exposure μg/cm²/day
TCPP production – lab worker, sampling operator	1, 1
TDCP foam production – plant 1 lab worker	0.08, 5.2, 0.04
TDCP foam production – plant 2 lab worker	0.22
V6 foam production – plant 2	2.34

Values taken forward to risk characterisation

For RWC inhalation exposure a value of 12.5 μg/m^3, 8hr TWA will be taken forward for risk characterisation, with a typical exposure value of 6.7 μg/m^3.

For dermal exposure a RWC value of 5.2 μg/cm^2/day, or 1.1 mg/day and a typical exposure value of 1 μg/cm^2/day, or 0.21 mg/day will be taken forward for risk characterisation. The highest value has been taken for RWC value as it was not possible to calculate a 90th percentile. The median value of 1 μg/cm^2/day has been taken to represent the typical exposure. It is estimated that the area of skin exposed will not exceed 210 cm^2. This dermal contact area was selected as the description of the process did not indicate that there was any opportunity for large-scale dermal exposure.

4.1.1.1.10 Scenario 10: Occupational exposure during the use of one-component (1-K) foams

PUR 1-K foam is delivered in cans containing 500-1000 g of material. Some 1-K foams are used by construction workers on building sites while others are available to the general public for the DIY filling of cavities (see consumer exposure section 4.1.1.2.2). During application, the foam emerges through a plastic pipe and is injected into gaps for example, for installation of window- and door-frames. After one hour the foam is fully cured. After curing, the TCPP is embedded in the polycondensate structure of the PUR and has no tendency to migrate (Rhee 2002).

Measured inhalation exposure data during the use of 1-K foams

Monitoring was carried out by one of the producers of 1-K foam to determine the inhalation and dermal exposure to TCPP during the use of 1-K foams. In each test, the foam (containing 13% TCPP) was sprayed into a plastic (x1) or a paper sack (x2).

For potential inhalation exposure, a silica-gel tube was fixed at the right wrist of the operator and then the operator's hand and wrist were inside the sack while spraying. The volume inside the sack was 100L. It was considered that results obtained would be representative of a worst case scenario as the ventilation inside the sacks would be very poor and the silica tube was very close to the foam as it came out of the can. In practice, a professional user would generally be about 0.5-1 meter away from the outlet of the can while spraying. It is assumed that a user would empty a can in about 15 minutes and use up to 3 cans per day.

The method used for analysis was the same as that previously described (Akzo Nobel Method CG/6.089.3). Briefly, air was passed through the silica-gel tube at a constant flow rate. The adsorbed TCPP was desorbed with methanol applying ultrasound for 10 min (tributylphosphine oxide, the internal standard, had been added to the methanol beforehand). The sample was analysed by GC, with a pulsed flame-photometric detector.

Table 4.34 below gives the results from this monitoring. All 4 samples were taken from the same operator.

Table 4.34 Concentration of TCPP in air during use of 1-K foam

Sample	Monitoring time inside sack (mins)	Output (g foam/min)	TCPP (ppb)	TCPP (mg/m^3)	TCPP 8hr TWA (µg/m^3)
(a) 6 mins spraying	30	26.5	0.6	0.08	0.045
(b) 5 mins spraying	5	158	24	0.32	0.03
(c) 4 mins spraying	4	200	51	0.68	0.051
(d) 5.5 mins spraying	5.5	144	9.9	0.13	0.013

The concentration of TCPP appears to be linked to the output of foam per unit time, but the correlation is poor. Sample (c) was a nearly non-stop spraying process whereas in samples (b) and (d) small breaks of some seconds were taken. From industry experience, it is felt that these two samples may be more representative for workplaces. However, the positioning of the sampling media on the wrist and spraying into a bag, with the hand inside the bag is considered to over estimate the potential exposure of a worker using these cans. Although the worker may be using the foam in a relatively small space, his breathing zone is likely to be between 0.5 and 1m away from the can, so exposure would be expected to be less than that measured in the measurements described above. To take this into account, in the absence of any other valid data, the supplied data has been used to calculate an 8-hr TWA and this has been reduced by a factor of 10.

Measured dermal exposure

To estimate dermal exposure in the first test, the operator's right hand was covered with a cotton bandage and pieces were cut from the inside and outside for TCPP analysis after emptying 2 cans of 700 g of foam each. In the second and third tests, the operator wore a thin latex glove. After the tests, the glove was transferred into a glass bottle and methanol and internal standard were added followed by a 2-hour incubation period. After this, the methanol was filtered off, concentrated down to 2 ml and analysed.

Table 4.35 below gives a summary of the results obtained for the amount of TCPP extracted from cotton bandage/latex gloves following use of 1-K foams. Each sample was taken while one can was emptied, a process which took 4-6 minutes in each case. In sample 1, the cotton bandage was worn on the dorsal side of the hand and in sample 2, it was worn on the plantal side of the hand. Given that the spraying took place in a sack, there was much more opportunity for contact with the foam than would be the case during normal working operations. For this reason, the highest dermal exposure result has been discounted for workers, although it has been used for consumer exposure where it is more likely that contact with foam will occur. Also as it was reported that about 3 cans would be used per day, the results have been adjusted to reflect this as the sampling took place during the use of two cans.

Table 4.35 TCPP extracted from cotton bandage/latex glove following use of 1-K foam

Sample	TCPP (μg)	Foam (g)	TCPP (μg/cm²)
(a) Cotton bandage (49 cm²)	61	1582	1.24
(b) Cotton bandage (10 cm²)	1161*	1582	116.1
(c) Latex glove** (estimated 400 cm²)	113	802	0.28
(d) Latex glove (estimated 400 cm²)	375	794	0.94

* Direct contact with fresh foam.

** When questioned, industry has indicated that they do not know if TCPP penetrates latex. This work was carried out to get some indication of potential dermal exposure to TCPP. These latex gloves are not normally used as PPE.

Values taken forward to risk characterisation

The RWC inhalation value taken forward for risk characterisation is 0.005 μg/m^3, 8-hour time-weighted average, with a typical exposure value of 0.0025 μg/m^3, 8-hour TWA being taken forward.

For dermal exposure, a RWC exposure value of 1.9 x 10^{-3} mg/cm^2/day (1.24 μg/cm^2 x 3/2), or 0.78 mg/day which is taken forward for risk characterisation. This is derived from the measured data, adjusted for the use of 3 cans rather than 2. For typical exposure, a value of 9.3 x 10^{-4} mg/cm^2/day, or 0.39 mg/day will be taken forward for risk characterisation. This is half the RWC and also lies between the other two values supplied by industry (before adjustments are made for use of three cans). It is estimated that 420 cm^2 would be the area exposed particularly for inexperienced workers. In reality the use of suitable gloves would reduce exposure if changed regularly.

4.1.1.1.11 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 4.36**, below.

Table 4.36 Summary table of RWC and typical inhalation and dermal exposure values 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: Production of TCPP	25	12.5	1	0.1	210
2: Manufacture of flexible PUR foam	5.1	0.62	0.07	0.002	420
3: Cutting flexible foam	4.1	1.9	7.1×10^{-3}	9.8×10^{-4}	420
4: Production of foam granules & rebonded foam	4.6	0.59	1.7×10^{-3}	5.5×10^{-4}	420
5: Formulation of systems and manufacture of spray foams	5	2.5	0.11	0.05	420
6: Use of spray foams	187.5	25	0.23	0.12	420
7: Manufacture of rigid foam	150	20	6.5×10^{-2}	3.2×10^{-2}	210
8: Use of rigid foam	4.1	1.9	1.3×10^{-2}	6×10^{-3}	210
9: Manufacture of 1K foams	12.5	6.7	5.2×10^{-3}	1×10^{-3}	210
10 Use of 1K foams	5×10^{-3}	2.5×10^{-3}	1.9×10^{-3}	9.3×10^{-4}	420

4.1.1.2 Consumer exposure

There are currently only three uses of TCPP identified by industry that could result in consumer exposure. There is no requirement on manufacturers to use TCPP in any other consumer products and the manufacturers have reported that it is therefore not used.

4.1.1.2.1 Potential exposure from flexible polyurethane foam

The current use pattern provided by industry indicates that most of the TCPP produced in the EU in 2000 was used in the production of polyurethane foam in Europe. Most of the TCPP used in flexible foam is used in upholstery and bedding. 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 expected that consumer exposure to TCPP from these foams is very low

Measured consumer exposure data

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 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 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 μg/m^3. **Table 4.37** below gives the results of this study.

Table 4.37 Results of chamber tests with mattresses made of TCPP-containing flexible PUR foam

Mattress Type	Air Concentration (μg/m^3)				
	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/m^3, 1.5% TCPP

[2]CME = Combustion modified ether, 33 kg/m^3.

[3] CMHR = Combustion modified high resilience foam, 35 kg/m^3

The detection limit was 2 μg/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. The concentration of TCPP in the chamber remained relatively constant for the CM foam, so a value of 19 μg/m^3 will be used. This is the highest value seen with the CM foam, and was also measured at the 120 hr time point with the HR foam.

The assumptions are as follows:

TCPP concentration in chamber air:	19 μg/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

From this study, the concentration of TCPP in indoor air in rooms with a high load of flame retarded flexible PUR foam can be estimated to be 3.8 μg/m^3.

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 of 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 4.38** below gives a summary of the results obtained for this study.

Table 4.38 Results of BRMA long-term aging trial on flexible PUR foam

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 (see Appendix B for further details).

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.

As people, particularly the elderly, could spend a large proportion of their time indoors in a room with PU foam-containing furniture, as a RWC, 3.8 $\mu g/m^3$, 24 hour TWA could be taken forward for risk characterisation. Assuming that the majority of consumers would spend some time in areas without PU foam-containing furniture a typical exposure could be estimated as 2.8 $\mu g/m^3$ 24hr TWA (18 out of 24 hours spent in areas with PU foam-containing furniture or other items).

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.

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. This is considered a valid means of generating information for risk characterisation as the two substances have quite 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.

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.

4.1.1.2.2 Potential exposure to consumers from the use of 1-K foams

Some 1-K foams are available to the general public for the DIY filling of cavities. The data given above (section 4.1.1.1.10) for the study carried out to measure occupational exposure during the use of 1-K foams can be used to estimate consumer exposure. From that study, a RWC inhalation exposure for a consumer can be estimated as 0.005 mg/m3 and a typical exposure as 0.0025 mg/m^3. These are the same values as for workers, although it is probably unlikely that a consumer would use 3 cans in one day. Dermal exposure is estimated (as a worst case scenario, assuming direct contact with the foam) as being 174 µg/cm^2. However, most consumers would not regularly be spraying foam. It is very unlikely that they would spray foam more than once per year, and more probably would use spray once or twice in a lifetime, if at all. Exposure for consumers in this scenario is considered to be negligible over a lifetime, but could be significant in the short-term.

4.1.1.2.3 Potential exposure from closed-cell rigid foam used for insulation purposes

One rigid foam-producing company carried out a chamber test to check TCPP emerging from a closed-cell rigid foam intended for insulation purposes. This spray-foam has also been developed for potential indoor-air application, which was the driving force behind the chamber-test. The foam had a thickness of 10 cm (regarded to be the upper limit for indoor-application) and contained 9% TCPP. The surface to volume ratio of the test-specimen was 1.4 m^2/m^3, which is considered to represent a typical real-life scenario. For the test, a concrete plate was covered with a layer of the spray foam and then transferred into a test chamber in the test laboratory. The volume of the test chamber was 119 litres, temperature 23^0C and relative humidity 50%. The air exchange rate was 0.5 h^{-1}. The loading of the test chamber was 1.4 m^2 test specimen per m^3 air volume. Air sampling from the chamber outlet air was carried out after 3 and after 28 days onto Tenax TA, followed by thermal desorption, gas chromatography and mass spectroscopy. The method applied was based on published methods. No TCPP could be detected (detection limit was 1 µg/m^3).

From this work, it can be concluded that consumers are potentially exposed to negligible amounts of TCPP in rooms containing closed-cell rigid foam.

4.1.1.3 Humans exposed via the environment

Table 4.39, which is taken from section 3 of this report, gives the predicted environmental exposures to TCPP and the daily human doses arising from releases from production, processing, manufacture and use of TCPP. 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 2×10^{-4} mg/kg/day and the highest local exposure (use large systems house) is 0.104 mg/kg/day.

These figures will be taken forward to risk characterisation.

Table 4.39 Indirect exposure of humans to TCPP 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	5.09E-08	5.15E-05	4.55E-06	3.00E-04	6.65E-08	8.19E-08	5.88E-05	4.15E-04
Producer 2	1.89E-07	2.56E-04	3.97E-05	3.44E-04	9.58E-08	1.18E-07	5.89E-05	6.99E-04
Producer 3	5.01E-08	5.15E-05	2.29E-06	3.00E-04	6.65E-08	8.18E-08	5.88E-05	4.13E-04
Producer 4	5.65E-08	1.22E-04	4.24E-06	6.91E-04	1.02E-07	1.26E-07	1.39E-04	9.57E-04
A1a: Large systems houses	3.13E-04	6.23E-04	2.23E-06	0.102	2.06E-05	2.54E-05	7.12E-04	0.104
A2: Medium systems houses	6.81E-05	9.72E-04	1.51E-04	0.0239	4.71E-06	5.80E-06	4.86E-04	0.0256
A3: Small systems houses	1.71E-05	2.55E-04	3.95E-05	6.22E-03	1.23E-06	1.51E-06	1.66E-04	6.70E-03
A4: Systems houses using preformulated polyol	1.44E-06	6.27E-05	5.26E-06	8.03E-04	1.62E-07	2.00E-07	7.16E-05	9.44E-04
B1a: flexible foam (furniture) very large	6.77E-07	5.49E-05	3.58E-06	5.18E-04	1.09E-07	1.34E-07	6.28E-05	6.40E-04
B1b: flexible foam (furniture) large	1.62E-07	5.21E-05	2.46E-06	3.39E-04	7.40E-08	9.12E-08	5.95E-05	4.54E-04
B1c: flexible foam (furniture) small - not using systems	6.38E-08	5.77E-05	4.74E-06	3.39E-04	7.05E-08	8.68E-08	6.60E-05	4.68E-04
B1d: flexible foam (furniture) small - users of systems	7.46E-08	6.26E-05	6.73E-06	3.69E-04	7.36E-08	9.06E-08	7.16E-05	5.11E-04
B2: flexible foam cutting	1.89E-07	5.23E-05	2.52E-06	3.48E-04	7.58E-08	9.34E-08	5.97E-05	4.63E-04
C1: rigid foaming large sites	7.10E-08	5.16E-05	2.26E-06	3.07E-04	6.79E-08	8.36E-08	5.90E-05	4.20E-04
C2: rigid foaming small sites	5.44E-08	1.39E-04	2.16E-05	5.66E-04	9.46E-08	1.17E-07	1.14E-04	8.41E-04

	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]
E1: one-component foams	6.13E-05	1.22E-03	1.89E-04	0.0223	4.33E-06	5.33E-06	5.94E-04	0.0243
F1: confidential	1.03E-05	1.67E-04	2.60E-05	3.92E-03	7.67E-07	9.44E-07	1.41E-04	4.27E-03
G1: confidential	5.49E-06	1.23E-03	9.73E-05	8.50E-03	1.00E-06	1.24E-06	1.40E-03	0.0112
G2: confidential	2.95E-07	1.15E-03	5.57E-05	6.45E-03	6.32E-07	7.78E-07	1.32E-03	8.98E-03
H1: confidential	3.22E-07	2.25E-03	6.17E-05	0.0125	1.18E-06	1.46E-06	2.57E-03	0.0174
I1: confidential	5.49E-06	1.62E-04	1.41E-05	2.63E-03	4.74E-07	5.83E-07	1.85E-04	3.00E-03
J1: confidential	5.83E-05	8.41E-04	1.31E-04	0.0219	4.14E-06	5.10E-06	7.05E-04	0.0236
K1: confidential	2.04E-05	1.27E-04	1.11E-05	7.15E-03	1.42E-06	1.75E-06	1.45E-04	7.46E-03
K2: confidential	7.32E-08	4.91E-04	4.25E-05	2.73E-03	2.87E-07	3.54E-07	5.61E-04	3.83E-03
L1: confidential	5.18E-08	5.48E-05	2.51E-06	3.19E-04	6.82E-08	8.40E-08	6.26E-05	4.39E-04
M1: confidential	2.29E-06	6.43E-05	7.30E-06	1.08E-03	2.18E-07	2.69E-07	7.35E-05	1.23E-03
N1: confidential	5.02E-08	2.72E-04	1.41E-05	1.51E-03	1.76E-07	2.17E-07	3.10E-04	2.11E-03
O1: rebonding	4.35E-07	5.15E-05	2.21E-06	4.22E-04	9.14E-08	1.13E-07	5.89E-05	5.35E-04
P1: confidential	6.78E-06	1.09E-04	1.69E-05	2.64E-03	5.23E-07	6.44E-07	1.01E-04	2.87E-03
Q1: adhesive pressing	6.58E-06	1.62E-04	1.65E-05	2.98E-03	5.44E-07	6.70E-07	1.85E-04	3.35E-03
R1: loose crumb	2.15E-07	5.15E-05	2.21E-06	3.52E-04	7.71E-08	9.50E-08	5.89E-05	4.65E-04
Regional	5.01E-08	2.38E-05	2.21E-06	1.47E-04	3.24E-08	3.99E-08	2.72E-05	2E-4

4.1.1.4 Combined exposure

The combined exposure to TCPP 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 4.36** (see section 4.1.1.1.11). The highest occupational RWC inhalation and dermal exposures occur during the manufacture of TCPP (scenario 1). The occupational dermal exposure level is significantly higher than the estimated exposure to consumers or indirect exposure via the environment, and thus will dominate the combined exposure estimate. Therefore, it is not considered necessary to include occupational exposure in the combined exposure calculation.

Consumers may be exposed to TCPP indirectly from a) flexible foam used in upholstery and bedding and b) closed-cell rigid foam used for insulation. Consumer exposure may also result from the use of 1-K foams containing TCPP, which are used in DIY applications.

Exposure is also possible indirectly via environmental sources.

The RWC exposures used in calculating the combined exposure are presented in **Table 4.40** below.

Table 4.40 Exposures taken into account for combined TCPP 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
Use of 1-K foam	
Inhalation	0.005 mg/m^3
Dermal	174 µg/cm^2
Release of TCPP from closed cell right foam	Negligible
Man via the environment	
Local exposure	0.104 mg/kg/day*
Regional exposure	0.0002 mg/kg/day

*highest exposure scenario for local exposure (A1a: large systems houses)

4.1.2 Effects assessment: Hazard identification and dose (concentration) - response (effect) assessment

As outlined in chapter 1, TCPP is a reaction mixture containing four isomers. The ratio of isomers in the commercial product can vary from each supplier. The exact composition of TCPP, from each supplier, is provided in a confidential annex, which is available to Member States on request. There is no information available on the toxicological effects of the individual isomers.

The individual isomers of TCPP have never been produced separately. Industry has informed the Rapporteur that they never produce or market individual isomers, therefore they are not commercially available. It is also important to note that humans will never be exposed to individual isomers, as the commercial product is always a mixture of isomers.

It has proven difficult to obtain information on the exact composition of what was tested (i.e. isomer distribution of the tested material) from the older studies on TCPP. Industry has indicated that what was tested was representative of what was on the market at the time. Any testing carried out during the course of the risk assessment was carried out on equal mixtures of the four products from the four suppliers. Therefore, as what was tested was always a composite sample of the 4 products, the isomer content would be constant. Regarding the 4 products placed on the market, the two main isomers (Tris(1-chloro-2-propyl)phosphate and Bis(1-chloro-2-propyl)-2-chloropropylphosphate) make up more than 90% of the product from each producer. This means that overall, there is limited scope for variation in isomer content between the four commercially available products.

The four isomers of TCPP are very similar in many ways. Their phys-chem properties are similar and they exhibit similar chromatographic properties. The main two isomers outlined above are structurally very similar. The minor structural difference between these 2 isomers is not expected to create a different biological effect or lead to different toxicological properties. The second 2 isomers (Bis(2-chloropropyl)-1-chloro-2-propylphosphate and Tris(2-chloropropyl)phosphate) are also structurally very similar to each other, and relatively similar to the first 2 isomers. All 4 isomers have the same HILL formula. The isomers are not R and S isomers (enantiomers or diastereomers) of each other, which could influence their biological effects, but rather they are structural isomers of each other.

QSAR analysis of the four isomers of TCPP (DEREK, carried out by Bayer, Germany) showed no differences in toxicological characteristics. QSAR programmes look for structural identifiers in molecules and all four molecules carry the same structural alerts. This outcome is also in line with phys-chem model calculations using EPA models, also carried out by industry.

Industry has confirmed that it is not proposed to isolate and test the individual isomers of TCPP.

4.1.2.1 Toxicokinetics, metabolism and distribution

4.1.2.1.1 Studies in animals

In vivo studies

Inhalation

No studies are available. In line with the TGD, 100% absorption is taken forward to risk characterisation.

Dermal

No studies are available.

Oral

In a comparative study on absorption, distribution, and excretion of flame retardants halogenated alkyl phosphate in rats (Minegishi et al., 1988), 5 rats were orally administered a single dose of 50 µmol/kg (16.38 mg/kg) ^{14}C-TCPP (purity 99%; specific activity 0.213 mCi/mmol) in olive oil. Urine and faeces were collected every 24 hours for 7 days. Expired 14CO2 was determined after 72 and 96 hours. Bile was collected via cannulation every 2 hours for the first 30 hours following administration, from 30 – 46 hours and from 46 – 48 hours. Tissue samples were taken at 3, 6, 12, 24, 72 and 168 hours. Tissue radioactivity was analysed by oxidation followed by LSC and also by GC.

The recovery of radioactivity after 7 days was urine (67.2%), faeces (22.2%), expired air (7.7%) and carcass (0.7%) (total recovery was 97.8%). Seven days after oral administration of TCPP, the tissue distribution of radioactivity was, in order of decreasing concentration, liver, kidney, lung, fat, muscle, gonads, spleen, blood, heart and brain. Approximately 45% of administered radioactivity was excreted via the bile in 48 hours. This excretion was quite rapid, with approximately 30% being excreted after 3 hours. The average T_{max} value for TCPP radioactivity in tissues was 5.7 hours. Tissue/blood ratios calculated at various intervals over 7 days were > 1 for liver, kidney and lung and from 12 hours in adipose tissue indicating incorporation of radioactivity into these tissues. The decrease in radioactivity in all tissues was biphasic. The longest $t_{½}$ was recorded in adipose tissue in both phases of elimination (16.5 hours and 103.4 hours, respectively). However, the concentration of radioactivity was low implying no bioaccumulation. The biliary/faecal excretion ratio was 2.23 at 48 hours indicating enterohepatic re-circulation from the GI tract.

Oral and Intravenous

In a study conducted by the Stauffer Chemical Co. (unpublished report, 1984) two types of experiments were performed. A "recovery" study was carried out in which animals were dosed and urine, faeces and expired air were collected for 8 days (no serial blood samples were collected) and a "plasma" study was carried out in which blood samples, urine and faeces were collected. Radiolabelled-^{14}C-TCPP (40 µCi) was administered by oral gavage at a dose level of 200 mg TCPP/kg body weight to 5 male and 5 female Sprague-Dawley rat in both the plasma and recovery studies. A lower dose level of 20 mg/kg was also administered intravenously to 5 male rats and a further 5 male rats were dosed orally at this dose level. Tissues were isolated at sacrifice (at end of study period, 8 days post-dosing). Sample

radioactivity was determined by liquid scintillation counting (LSC). Metabolites were purified and identified by thin layer chromatography (TLC). Metabolite samples and standards were also identified via derivatisation and analysis by GC-RAM (radioactive monitor) or GC-MS.

C_{max} after intravenous injection of 20 mg/kg averaged 142 µg equivalents/ml and was reached in 0.15 hours while after oral administration of 20 mg/kg a Cmax of 7.68 µg equivalents/ml and was reached in 0.5 hours. Cmax after high dose oral administration was 84 µg equivalents/ml and was reached within 2 hours.

A significant difference ($p<0.05$) in the urinary excretion of radioactivity between rats given TCPP orally and intravenously was observed at the 20 mg/kg dose level. Approximately 63% of the dose was excreted in the urine of rats dosed intravenously, whereas only 49% of the dose was excreted in the urine of rats dosed orally. Significant dose-related changes in urinary excretion were also observed between rats at the 20 and 200 mg/kg dose levels. At the end of the eight-day study period, an average of 48% and 70% of the dose was recovered in the urine of male rats at the 20 and 200 mg/kg dose levels, respectively. This data indicates that while urinary excretion is the primary route of elimination for TCPP, the extent of excretion via this route is dependent on both the dose administered and the route of administration.

Faecal excretion also appeared dependent on the route of administration and dose level. Approximately 27% of the 20 mg/kg dose was excreted in the faeces following intravenous administration, whereas 40% of the dose was excreted via the faeces in male rats dosed orally. In contrast to the 40% excreted at the low dose level, 22% of the oral dose was excreted in the faeces of male rats at the 200 mg/kg level. Despite the differences in urinary and faecal excretion of total radioactivity at the different dose levels and with different routes of administration, the total elimination of radioactivity via the two routes was rapid and constant, averaging 89% of the dose at 72 hours.

Radioactivity was detected in many tissues. When expressed as ng equivalents/g tissue/mg TCPP, the overall distribution trend in decreasing order was liver, skin (especially at the high dose), fat, small intestine (especially at the high dose), lung, kidney, spleen, large intestine, heart (especially after i.v. administration), brain, stomach, gonads and muscle. However, while the substance was detected in many tissues, the actual amounts were extremely low, thus indicating little distribution.

Radioactivity remaining in the tissues of rats at the end of the 8 days was <1% of that administered. This indicates minimal bioaccumulation.

Highest concentrations of residual radioactivity at the end of the 8 day study period were found in the liver, small and large intestines, gonads, fat and skin of females at the 200 mg/kg dose level and in the liver, fat and skin of males at the high dose level. Normalisation of the individual concentrations of radioactivity with respect to dose revealed that the distribution of residual radioactivity in the tissues was dependent on both the route of administration and the dose level. At the 20 mg/kg dose level, the normalised concentrations of residual radioactivity in the spleen, stomach, heart, lungs and kidneys were significantly ($p<0.05$) higher in rats dosed intravenously than those administered the same dose by the oral route. This indicates less than 100% absorption. The normalised concentration of residual radioactivity was also significantly higher in the liver of males at the 200 mg/kg dose level, in comparison to those dosed orally at the 20 mg/kg level. In the kidney, for example, the normalised concentration was 40.37 ng equivalent/g of tissue/mg TCPP in the i.v. dosed group compared to 27.43 ng equivalent/g of tissue/mg TCPP in the orally dosed group. It is possible to get an estimate of absorption from the GI tract by comparing tissue levels after oral exposure versus intravenous

administration, at a similar time point. The percent absorption is the oral sample divided by the intravenous sample; basing this calculation on the values given above for the kidney, the oral absorption can be estimated to be 68%.

TCPP was extensively metabolised prior to excretion in the urine and faeces. Unchanged TCPP represented less than 2% of the administered dose at both dose levels. 0,0-[Bis(1-chloro-2-propyl)]-0-(2,proprionic acid)phosphate was identified as a major metabolite in both urine and faeces, accounting for over 50% of the dose in males at both doses. At the low dose, this metabolite was excreted approximately equally in the urine and faeces in males, whereas at the higher dose, it was excreted predominantly in the urine. In females, at the higher dose, it was again excreted predominantly in the urine. The dose-dependent excretory pattern of this metabolite in the urine and faeces corresponds well with the dose-dependent changes in excretion of total radioactivity observed at both dose levels, suggesting that this metabolite is responsible for the dose-dependent excretory pattern noted at these dose levels. Other metabolites isolated and identified were bis(1-chloro-2-propyl)monophosphoric acid and 1-chloro-2-propanol. The monophosphoric acid metabolite accounted for 12% of the total radiocarbon administered to male rats at both dose levels. The 1-chloro-2-propanol metabolite was not quantified.

In vitro studies

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]-TCPP to human skin for 8 hours. Three dose levels were tested, 0.002, 0.1 and 1.0 mg/cm^2, which corresponded approximately to the typical exposure during manufacture of 1K foams, a mid dose to enable a dose response extrapolation and the reasonable worst case exposure during manufacture of TCPP, 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 TCPP in the receptor fluid was determined to be *ca*. 270 µg/ml, which was considered sufficient. 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 2.5 x 10^{-3} cm/h were selected for the study.

The dose solutions were prepared on the day of application. [^{14}C]-TCPP was mixed with non-radiolabelled TCPP 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 TCPP was applied in a small volume of acetone (20µl) 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 TCPP 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 4.41** below gives a summary of the amount of TCPP found in each sample.

Table 4.41 Summary of percutaneous penetration of TCPP through human skin *in vitro*

	A		B		C	
Concentration measured [mg/ml]	0.066		3.199		31.914	
Dose [µg/cm²]	2.049		99.96		997.33	
n	6		6		6	
Penetration into the receptor fluid after 24 h	% of dose	µg/cm²	% of dose	µg/cm²	% of dose	µg/cm²
	18.81	0.39	9.65	9.64	1.78	17.75
Maximal flux [µg/cm²/h]	0.027		0.602		0.836	
Lag time [h]	2.7		4.1		2.8	
Mean total absorption [%]* (SD)	22.7 (5.8)		13.6 (3.6)		3.7 (1.3)	

* Total absorption is defined as the amount in the receptor fluid, the receptor compartment wash and skin membrane, excluding tape strips.

The mean recovery of TCPP in human skin was 99.7 ± 6.2%, 99.2 ± 5.7% and 93.5 ± 6.9%, for the high, mid and low doses, respectively.

The mean penetration of TCPP into the receptor fluid after 24 hours was 0.39, 9.64 and 17.75 µg/cm^2, for the low, mid and high dose, respectively. The mean maximal flux was 0.027, 0.602 and 0.836 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 0.002 mg/cm^2, the total absorption ranged from 17 % to 32.8%, with a mean total absorption of 22.7 %. At the mid dose of 0.1 mg/cm^2, the total absorption ranged from 9.8% to 18.2%, with the mean total absorption of 13.6%. At 1 mg/cm^2, the total absorption ranged from 2.3% to 5.2%, with a mean total absorption of 3.7%.

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/0750/03 Final, October 2003). Therefore, a worst case mean total absorption value of 23 % has been taken forward to risk characterisation for all scenarios where there is potential exposure to "neat" TCPP. This is considered to be a reasonable worst case value since 16 of 18 individual membrane measurements taken were found to be 23% or lower.

In a separate *in vitro* percutaneous absorption study (TNO Quality of Life, 2005) conducted to GLP guidelines and to OECD Guideline No. 428, the percentage of TCPP absorbed across the skin as a result of handling flexible PUR foam containing TCPP was determined. The study was performed using human skin membranes and the dermal absorption was determined by monitoring the compound-related radioactivity in the receptor fluid during 8 and 24 hour exposure periods.

In order to determine the target concentrations for the main study, a preliminary release test was performed. A stack of 15 filter papers wetted with artificial sweat was placed on top of two pieces of polyurethane foam (100 cm^2 surface area) containing 10 % w/w TCPP. One stack was pressed to ca. 70% of its original thickness to mimic squeezing during handling and

both stacks were incubated at 40°C for 2 hours. The amount of TCPP in each individual filter paper layer was determined by GC-FID analysis following extraction with methanol. The flux values (calculated from the sum of TCPP released into all filters over 2 hours) were 2.78 µg/cm²/h (no pressure applied) and 4.6 µg/cm²/h (pressure applied). In addition, the migration of TCPP was determined to establish the maximum quantity of TCPP potentially extractable from foam in artificial sweat by submerging pieces of foam completely in artificial sweat for two hours. The migration from the foam was determined to be 130 µg/cm²/h.

The main study was performed using human skin membranes in flow-through diffusion cells. As mentioned above, the target concentrations for the *in vitro* dermal absorption study were derived from the release test. The highest dose level was based on a total release of TCPP of 920 µg over 2 hours and a 100 cm² surface area. The release of TCPP from foam corresponds to 4.6 µg/cm²/h. The lowest dose level of 80 µg/ml was based on the amount of TCPP measured in the first filter paper (164 µg) mimicking the direct contact layer with the skin. The actual concentrations of TCPP tested in an artificial sweat solution over an 8 hour exposure period were 76 µg/ml and 506 µg/ml (reached during preparation and considered appropriately close to the target concentrations). A volume of 10 µl/cm² of each dose was applied to the skin surface.

Radiolabelled TCPP ([^{14}C]TCPP) was mixed with non-radiolabelled TCPP to obtain a target amount of radioactivity of ca. 1 x 10^6 dpm per skin membrane. The appropriate volume of artificial sweat was added to the TCPP prior to application to the skin. Each skin membrane was tape stripped using D-squame. After 8 hours of exposure, TCPP was removed from the surface of the skin using a mild soap solution and cotton swabs. Receptor fluid samples were collected at 2 hourly intervals until 24 hours after application. Exposure to the highest concentration was also performed during a 24 hour exposure period under occluded conditions to determine the maximal flux.

Radioactivity in all samples was determined by liquid scintillation counting. At 24 hours after application, the total mean absorption of TCPP into the receptor fluid, the receptor compartment wash and the skin (excluding tape strips) was 33.3% and 38.1% for the low and high doses respectively. The mean recovery of TCPP in human skin was 93.1% and 92.2% for the low and high doses respectively. The permeability constant (K_p) for TCPP in artificial sweat under infinite conditions (24 hour exposure) was 7.65 x 10^{-3}/cm/h. **Table 4.42** below gives a summary of the amount of TCPP found in each sample.

Table 4.42 Summary of percutaneous penetration of TCPP in artificial sweat through human skin *in vitro*

Concentration of TCPP	**76 µg/ml**	**506 µg/ml**
	Mean percentage of dose	
Receptor fluid + Receptor wash	31.64	35.63
Donor compartment	0.93	1.02
Tape strips	1.78	1.78
Cotton swabs	57.13	51.34
Skin	1.64	2.42
Total recovery	93.1	92.2
Total absorption*	33.3	38.1

* Sum of the amount in receptor fluid, the receptor compartment wash and the skin (excluding tape strips).

Therefore, the worst case mean total absorption value of 40 % has been taken forward to risk characterisation for scenarios where there is potential exposure due to handling of foam containing TCPP, i.e. Scenario 3 "Cutting of flexible PUR foam", Scenario 4 "Production of rebonded PUR foam" and Scenario 8 "Use of rigid PUR foam".

Metabolism

An *in vitro* comparative metabolism study was carried out with TCPP and the structurally similar substances, TDCP and TCEP using liver S9 fraction and liver slices from male Wistar-Han rats (BASF Aktiengesellschaft, 2007). In the first assay, ^{14}C-TCPP, TCEP and TDCP were incubated in rat liver S9 fraction for four hours. The suspensions were then centrifuged and the resultant supernatants used for Radio HPLC and LC/MS analysis. In the second assay, the radiolabelled substances were incubated with rat liver slices for 24 hours and following the incubation, the liver slices were removed and the mediums used directly for Radio HPLC and LC/MS analysis.

The recovery of radioactivity following incubation with liver S9 fraction was generally greater than 95% for ^{14}C-TCPP, TCEP and TDCP. For the incubations in liver slices, the recovery of radioactivity was greater than 80% for ^{14}C-TCPP and TCEP, and greater than 62% for ^{14}C-TDCP.

The metabolic turnover for ^{14}C-TCPP was 89% and 61% when incubated with liver S9 fraction and liver slices, respectively. The results indicate that TCPP was metabolised to a hydroxylated metabolite by chlorine substitution in liver S9 fraction and liver slices, followed by glucuronic acid conjugation in liver slices. 11% and 39% of unmetabolised TCPP were detected in S9 fraction and liver slices, respectively.

4.1.2.1.2 Studies in humans

No data are available.

4.1.2.1.3 Summary of toxicokinetics, metabolism and distribution

After oral administration, there were indications of <100% absorption, when oral and i.v. dosing were compared. It is quite difficult to estimate the percent oral absorption. However, it appears from the available information that oral absorption is at least 75%, and may be slightly higher (based on the Minegishi data, and supported by the Stauffer data). Therefore, 80% oral absorption will be taken forward to risk characterisation.

After oral administration, C_{max} in plasma was reached in 0.5 to 2 hours and 5.7 hours in tissues. Tissue radioactivity concentrations were dose and administration route-dependent (oral and intravenous). Although tissue/blood ratios over 7 days were > 1 for liver, kidney, lung and adipose tissue, absolute concentrations were low and the bioaccumulation potential was considered minimal. TCPP is extensively metabolised and accounted for <2% of urinary or faecal radioactivity after oral administration. Metabolites identified in urine and faeces, in order of abundance, were 0,0-[Bis(1-chloro-2-propyl)]-0-(2-propionic acid)phosphate, bis(1-chloro-2-propyl)monophosphoric acid and 1-chloro-2-propanol. Elimination of TCPP from plasma and tissues was biphasic. The average terminal plasma t½ was 48.7 hours. The longest tissue t½ was recorded in adipose tissue (up to 103.4 hours). Urinary and faecal excretion of radioactivity was dose and administration route-dependent (oral and intravenous), and occurred quite rapidly. The observed biliary/faecal excretion ratio is indicative of

enterohepatic recirculation. In a separate *in vitro* comparative metabolism study with ^{14}C-TCPP, TCEP and TDCP, TCPP was metabolised to TCPP was metabolised to 89 and 61% respectively in rat liver S9 mix and liver slices. An *in vitro* percutaneous absorption study using human skin membranes was conducted to determine the absorption following topical application of [^{14}C]-TCPP. The skin membranes were exposed to TCPP for 8 hours, mimicking a normal working day. The mean total absorption was 22.7 %, 13.6 % and 3.7 %, for doses 0.002, 0.1 and 1 mg/cm^2, respectively. The total absorption value of 23% is taken forward to risk characterisation for scenarios where there is exposure to "neat" TCPP. A second *in vitro* study was conducted to determine the percentage of TCPP absorbed across the skin resulting from manual handling of flexible PUR foam containing TCPP. The skin membranes were exposed to the target concentrations of TCPP in artificial sweat for a period of 8 hours, mimicking a normal working day. It was determined that the total mean absorptions were 33.3% and 38.1% for the low and high doses of TCPP respectively. Therefore, with respect to risk characterisation, 40% dermal absorption will be taken forward for those scenarios where there is exposure due to handling of foam containing TCPP, i.e. Scenario 3 "Cutting of flexible PUR foam", Scenario 4 "Production of rebounded PUR foam" and Scenario 8 "Use of rigid PUR foam".

No toxicokinetic data is available for the inhalation routes at present. For this route, and in line with the TGD, 100% absorption is assumed.

4.1.2.2 Acute toxicity

4.1.2.2.1 Studies in animals

In vivo studies

Inhalation

In a study carried out by Inveresk Research International (1990a), 2 groups of 2 male and 2 female Sprague-Dawley rats were exposed to a measured gravimetric concentration of TCPP of up to 3.80 mg/L by snout only inhalation, in a dose range finding study, for a period of 4 hours (no other doses were given in the report). A further group of 5 female and 5 male Sprague-Dawley rats were exposed to a concentration of TCPP of 7.19 mg/L, also for 4 hours. This was based on the results obtained with the concentration used in the range-finding study. All animals were observed continuously for clinical signs throughout the exposure period, for the first 1-2 hours post-dosing and thereafter once daily during the subsequent 3 day (dose range finding study) or 14 day (main study) observation period. All rats were weighed immediately before dosing and on days 1 and 3 post exposure for the dose ranging finding study and on days 2, 3, 4, 7, 10 and 14 post exposure for the main study. At the end of the study, all animals were subjected to a macroscopic post mortem exam.

There were no deaths during the study. No unusual clinical observations were recorded during the exposure period. All animals appeared slightly unkempt and had red staining around the snout and eyes immediately after dosing. No abnormalities were observed during the subsequent 14-day observation period. There was no effect on body weight. No abnormalities were observed at post mortem. The acute inhalation LC_{50} is taken to be greater than 7 mg/L from this study.

In a limit test performed by Stauffer Chemical Co., Sprague-Dawley rats (10/sex) were exposed to a fine-particle aerosol (mean particle size 2.9 MMAD) at a mean concentration of 4.6 mg/L/4hr (unpublished report, Stauffer Chemical Co., 1979a). Toxic signs observed included mild lethargy and matted fur in males and females during exposure. All rats appeared normal by day 1 post exposure. No mortalities occurred after 14 days and gross necropsy revealed no lesions in males and reddened lungs in 3/10 females. The acute inhalation LC_{50} was deemed to be >4.6 mg/L/4hr. The study was performed to GLP and in accordance with USEPA Guidelines.

A one-page report of an acute inhalation toxicity study using TCPP was provided by Environmental Affairs and Toxicology Dept (1981a). In this study 10 rats (Sprague-Dawley) (5/sex) were exposed to a single-dose fine-particle aerosol concentration of 5.05 mg/L for 4 hours. All rats displayed decreased activity, partially closed eyes, wet coats and watery salivation during exposure. During the subsequent 14 days, the rats exhibited slight to severe lethargy, reddish lacrimation, acute bodyweight depression (magnitude not specified in report), brown discharge around oral cavity, slight alopecia, convulsions and dyspnea. Death occurred in 3/5 females and 0/5 males. By day 14, all observed effects had disappeared in surviving animals. The acute inhalation LC_{50} for rats was considered to be >5.05 mg/L/4hr for males and approximately 5 mg/L/4hr for females.

In a one-page report submitted from Environmental Affairs and Toxicology Department (1981b), 10 rats (sex and strain unspecified) were exposed to an aerosol of TCPP at a nominal concentration of 17.8 mg/L. The rats were exposed to the test substance for 1 hour. No details of the actual chamber concentrations were provided. No mortalities occurred. All rats exhibited decreased activity, partially closed eyes, swollen eyelids and lacrimation during exposure. Nine of 10 rats had oily and/or matted fur upon removal from the chamber, which persisted through day 10 and most rats (number not specified) exhibited dry rales during the first 4 days post exposure. Three female rats experienced small transient weight losses. Although no raw data were provided, the author stated that the dry rales, decreased body weight and necropsy findings did not demonstrate any persistent toxicity. However, given the limited details, no conclusions can be drawn from this study.

Oral

In a series of studies conducted to OECD guidelines (Safepharm Laboratories Ltd., 1994a, 1996a and b, 1997a and b), the acute oral toxicity of TCPP was determined. LD_{50}s through the 5 studies ranged from 930 mg/kg to 1550 mg/kg.

In the 1994a study, male rats received 707, 841 and 1000 mg/kg and female rats received 707 mg/kg only. One mid-dose male died at 24 hours and 4 high dose males died after 1 hour. One female died after 4 hours. The LD_{50} (for males) was estimated to be 931 mg/kg.

In the 1996(a) study, male rats were dosed with 800, 1183 and 1750 mg/kg while female rats received 800 mg/kg. 3 mid-dose males died at 1 hour, 4 high-dose males died at 4 hours while 1 female died at 5 hours. The LD_{50} for this study was estimated to be 1310 mg/kg (males).

Similar results were obtained from the 1996(b) study where 5 male rats received 1000, 1414 and 2000 mg/kg TCPP and 5 female rats received one dose of 1000 mg/kg TCPP. One low-dose and one mid-dose male died at 4 hours. All 5 males dosed with the highest dose died after 1 hour. Four out of the 5 females died after 1 hour. The LD_{50} for this study was estimated at 1363 mg/kg for males and <1000 mg/kg for females.

In the 1997a study, female rats received 1000, 1414 and 2000 mg/kg while male rats received 1000 mg/kg. Three mid-dose females died at 6-7 hours and 4 high-dose females died at 4 hours. No male rats died in this study. The LD_{50} was estimated at 1548 mg/kg (females).

The 1997b study dosed female rats with doses up to 1500 mg/kg and male rats with one dose of 817 mg/kg. Three females dosed with 1000 mg/kg died after 2 hours. All females dosed with the top two doses, 1225 and 1500 mg/kg, died at 4 hours and 2 hours, respectively. Two males dosed with 817 mg/kg died after 2 hours. The LD_{50} from this study was 980 mg/kg (females).

Common signs of systemic toxicity noted in all five studies included ataxia, hunched posture, lethargy, laboured respiration, increased salivation, partially closed eyelids, body tremors, clonic/tonic convulsions, pilo-erection, ptosis, loss of righting reflex, red-brown staining around the mouth. Common abnormalities noted at necropsy included haemorrhagic lungs, dark liver and/or kidneys and sloughing of the gastric mucosa. These specified effects were common across all 8 studies and occurred to varying degrees across the various doses. In the 1997b study, males were considered to be more sensitive to the test material than females but this was the only study where significant differences in sensitivity between sexes were observed.

The acute oral toxicity of TCPP in Sprague-Dawley rats was also tested in two other studies (Huntingdon Life Sciences, 1997a and b). Both studies were performed to GLP and to current regulatory standards. In both studies 5 female rats were dosed with 500, 1260 (1000 in the second study) and 2000 mg/kg, while 5 males received 2000 mg/kg. In the first study, 1 female died at 500 mg/kg after 1 hour. Two died at the mid-dose after 1 hour while all 5 treated with the top dose died after 1.6 hours. Four of the five treated male rats died after 1.6 hours. In the second study, all females treated with the mid- and highest dose died after 1 and 1.6 hours, respectively, while all males treated died after 1.6 hours. The LD_{50} for females was estimated to be 1011 and 707 mg/kg in the first and second study respectively. The LD_{50} for males was estimated to be <2000 mg/kg. Clinical signs of toxicity were similar to those described for the five studies above. No macroscopic abnormalities were observed at necropsy and all surviving animals achieved satisfactory weight gain throughout the study.

In another study (Stauffer Chemical Co., 1979b) male rats were dosed at 6 different dose levels (10 animals per dose level). No deaths occurred at the two lowest dose levels (1259 and 2000 mg/kg). Single oral doses of 3162 and 4000 mg/kg produced 3/10 and 4/10 deaths, respectively. Six animals (out of 10) died at both of the highest dose levels (4487 and 5000 mg/kg). From these results, the LD_{50} for male rats was taken as 4200 mg/kg. Ten females were administered single oral doses of 794 and 1259 mg/kg – these produced 0/10 deaths in both cases. Further single oral doses of 2000, 3162, 3565, 4000 and 5000 mg/kg produced 1/10, 3/10, 8/10, 9/10 and 10/10 deaths, respectively. The report concluded that the LD_{50} for females was 2800 mg/kg; however, based on the results outlined, the LD_{50} is better estimated at 3300 mg/kg. Major toxic signs in both sexes included depression, tremors, stained fur, lacrimation and salivation. In addition, females exhibited convulsions and hyperactivity. No abnormalities were observed in males at necropsy. Females were found to have bloated caecums and/or stomachs. This study was carried out in compliance with GLP and in accordance with USEPA Guidelines, however, the lowest dose in the male study was quite high (1259 mg/kg). The substance was administered in a corn-oil vehicle.

The acute oral toxicity of TCPP (in corn oil) in Wistar rats was determined in a GLP compliant study (Stropp, 1996). Doses of 200 and 500 mg/kg were administered by gavage to groups of 10 rats (5/sex) and an additional dose of 2000 mg/kg was given to a group of 5

females only. Mortalities only occurred at the top dose where all 5 females died. Macroscopic investigation of this test group revealed mottled reddened lungs. Clinical signs of toxicity in this high dose group included apathy, palmospasm and blood-crusted snout. There were no clinical signs of toxicity present in the 200 mg/kg and 500 mg/kg treatment groups. The acute oral LD_{50} was calculated as 632 mg/kg for female Wistar rats and >500 mg/kg for males.

In a screening study (Safepharm Laboratories Ltd., 1995), mortalities were produced in male and female rats by 3 samples of TCPP. Each sample contained varying ratios of the four TCPP isomers (individual isomers were not isolated or tested). All samples were tested at the 1000 mg/kg bw dose level. For the first TCPP sample, 2 males and 4 females out of 5 died; for the second sample, 2 males and all 5 females died and for the third sample, 4 out of the 5 males and 1 of the females died. Clinical observations were similar among all animals dosed with all three TCPP samples. In general, clinical observations such as hunched posture, salivation, laboured respiration, decreased respiratory rate, red/brown staining around the mouth, clonic convulsions, were observed within the first 4 hours of dosing. The samples tested were 'commercial' samples generally available on the market at the time.

In a second screening study (Safepharm Laboratories Ltd., 1994b), seven different samples of TCPP, again containing varying ratios of the 4 TCPP isomers, were tested on both male and female rats at three different dose levels (750, 1000, 1500 mg/kg). All 7 samples produced mortalities at the highest dose level. Six of these produced a 50% mortality rate at the 1000 mg/kg dose level. A 50% mortality rate also occurred following treatment with 3 of the isomers at a dose level of 750 mg/kg. Of the 7 samples tested, the LD_{50} was 1500 mg/kg for 1 of them, it was 1000 mg/kg for a further 3 of them and for the remaining 3 samples, the LD_{50} was 750 mg/kg. As above, the samples tested were 'commercial' samples generally available on the market at the time and the individual isomers were never isolated or tested on their own.

Two other studies conducted by Safepharm in 1979 (Safepharm Laboratories Ltd., 1979a and b) were not GLP compliant but the procedures described appear to be in accordance with current OECD guidelines. The acute oral LD_{50} of TCPP when tested on male and female Sprague-Dawley rats (5/sex/dose level) at 4 different dose levels (0.25, 0.68, 1.84, 5.0 ml/kg) was calculated to be 1.12 ml/kg for both sexes combined (approx LD_{50} 1440 mg/kg - specific gravity of TCPP = 1.287).

A study conducted by Stauffer Chemical Company in 1970 gave an LD_{50} of 2000 mg/kg for males and 1260 mg/kg for females. Groups of rats (5/dose) were dosed with TCPP at concentrations of 464, 1000, 2150 and 4640 mg/kg. One male and 1 female died after 4 hours and 5 hours, respectively, when dosed with 1000 mg/kg. Two males dosed with 2150 mg/kg died after 2-4 hours. All males dosed with the top dose and all females dosed with the two highest doses died after 2-12 hours. Signs of toxicity included depressions and intermittent muscle spasms in animals dosed with 464 mg/kg. Higher dose levels produced spasms, salivation, ataxia and spasmodic jumping. Male survivors of the 1000 mg/kg dose appeared normal when necropsied 14 days after treatment. At necropsy, females dosed with 2150 mg/kg exhibited congested kidneys, adrenals and liver. It appears from the report that these were the only animals necropsied. In another poorly reported study by Stauffer Chemical Co. in 1972, the acute oral LD_{50} in male rats following gavage at 4 dose levels (464, 100, 2150 and 4640 mg/kg) was determined to be 2710 mg/kg. Female rats were not tested.

Brief summary reports (actual studies not provided) for two acute oral toxicity studies in rats using TCPP (Mobil Environmental and Health Safety Laboratory, 1980a and 1981a) gave

calculated LD_{50} values as 1546 mg/kg for males and 1017 mg/kg for females (1980 study) and as 1824 mg/kg for males, 1101 mg/kg for females (1981 study).

A GLP-compliant acute oral toxicity study on Sprague-Dawley rats, (Inveresk Research International, 1989a) concluded an LD_{50} >2000 mg/kg for TCPP (Tolgard TMCP).

The following observations were made during an *in vitro/ in vivo* UDS assay (Bayer Healthcare 2005). In the pilot study, Wistar rats were treated with a single oral administration of 2000 mg/kg (3 males), 1500 mg/kg (2 males and 2 females) and 1000 mg/kg (2 females) TCPP. There were no mortalities at any dose level. One animal in 2000 mg/kg group showed narrowed palpebral fissures on Day 1. The males at 1500 mg/kg and the females treated at 1000 mg/kg were without findings, while the females treated at 1500 mg/kg showed piloerection on Days 1 and 2 and in addition twitches and narrowed palpebral fissures on Day 1. In the main study, TCPP was administered by oral gavage at 750 and 1500 mg/kg in corn oil to 4 females per dose group per sacrifice time (4 and 16 hours). Piloerection, narrowed palpebral fissures, apathy and accelerated breathing were noted in animals in 1500 mg/kg group prior to necropsy.

Acute delayed neurotoxicity

A delayed neurotoxicity study in hens was conducted using TCPP (Stauffer Chemical Co., 1979c). The test substance was administered as 10 ml/kg undiluted (equivalent to 12.9 g/kg) to 18 hens in two doses, 21 days apart. 5 hens were dosed with TOCP as a positive control. Food consumption, body weight and clinical signs (behavioural or postural abnormality, laying, locomotor ataxia, paralysis, feather loss, comb droop or discolouration, diarrhoea, morbidity) were observed. A specified behavioural neurotoxicity assessment was conducted weekly. Full histological examination of the brain, spinal cord and distal portions of the right and left sciatic nerves were carried out. In addition, four hens were dosed with corn oil, TOCP or TCPP for the biochemical assessment of plasma acetylcholinesterase and brain neurotoxic esterase (NTE) activities. The NTE activity was measured 18-24 hours later in an assay based on paraoxon and mipafox. Plasma ChE activity was measured in blood samples obtained after 24 hours using a colorimetric method.

Clinical signs of toxicity included feather loss, non-vocal behavioural change and diarrhoea. Feather loss was moderate to severe by study termination. A single mortality occurred on day 4. Mean body weights were significantly reduced compared to base line controls on days –7 and –14. The reduction was observed from treatment day 22 right through to day 43. When compared to the base line values taken at days –7 and –14, the % reductions ranged from 85-73% and 84-72% respectively. Food consumption was also significantly reduced in treated hens when noted on various days over the study and in comparison to baseline values taken on days –6 and –13. The % reductions ranged from 63-22% compared to values on day -6 and 83-29% when compared to values from day –13. Egg production ceased during the second week of the study. This was in comparison to control hens and to baseline values taken 7 days and 14 days before the hens were treated. Walking behaviour was impaired with a significant ($p<0.01$) increase in mean walking behaviour in treated hens on day 29 and 36 when compared to baseline scores taken on days –7 and –14. This was due to increased incidences of leaning back and questionable leg weakness and/or coordination. One hen stumbled when pivoting or side-stepping on day 29 and another swayed when walking (also on day 29). Findings on microscopic examination of treated hens were comparable between negative controls and TCPP treated hens. – Two treated hens showed minimal axonal degeneration in dorsal funiculi of the cervical, ventro-lateral funiculi of thoracic or ventro-medial funiculi of sacro-lumber spinal cord, tracts known to be sensitive to organophosphate-

induced degeneration. One of these hens was the hen that swayed when walking on day 29. Plasma, brain esterase and brain neurotoxic esterase were uninhibited by TCPP. There is no indication however, that TCPP actually reached the brain. The TOCP control gave an appropriate response in both the neurobehavioural and biochemical tests.

In conclusion, the principal effects of TCPP treatment of hens were reduced mean body weight and food consumption, feather loss and cessation of laying. There was no evidence of inhibited plasma acetylcholinesterase or brain neurotoxic esterase enzyme levels at a dose producing marked toxicity. Walking behaviour was impaired on days 29 and 36 of the study in TCPP-treated hens. The mean scores were significantly ($p<0.01$) greater than the base-line score on day –7. One hen stumbled on day 29, and this hen then showed minimal axonal degeneration at more than one level of the spinal cord. Overall, this is considered to be an isolated incident, and so there is no concern for this end-point.

Dermal

In one study, (Inveresk Research International, 1989b), no deaths occurred in either the dose-range finding study or the main study following dermal application of the test substance (purity unspecified) to Sprague-Dawley rats up to a maximum concentration of 2000 mg/kg. No clinical signs or post-mortem observations were noted. The acute dermal LD_{50} was therefore deemed to be >2000 mg/kg.

A second study (non-GLP) (unpublished report, Safepharm Laboratories Ltd., 1979c), determined that the acute percutaneous LD_{50} of TCPP after a 24 hour exposure in Sprague-Dawley rats was >10 ml /kg (equivalent to >12.9 g/kg).

In a report by the Stauffer Chemical Co. (1979b), TCPP was applied to the skin of male and female New Zealand albino rabbits at doses of 2000 and 5000 mg/kg (4 animals per sex and per dose level). The skin was abraded on half the animals and left intact on the others. The test sites of all animals were wrapped with a gauze binder. No deaths occurred over the 14-day observation period. Local effects included slight/mild erythema (observed at both dose levels). There were no gross abnormalities at necropsy with the exception of one rabbit (exposed to 2000 mg/kg), which showed discolouration of the ventral surface of the liver. The LD_{50} was estimated to be > 5000 mg/kg.

In a poorly reported study (Stauffer Chemical Co., 1970), 4 rabbits (sex and strain unspecified) received a dose of 5000 mg/kg TCPP and were observed for 14 days. No details of methods and materials used were provided in the study. No skin irritation or apparent signs of toxicity were observed. The LD_{50} was assumed to be > 5000 mg/kg.

In a study reported by Mobil Environmental and Health Science Laboratory (1981b), TCPP was applied to the clipped skin of 3 male and 3 female New Zealand albino rabbits at a dose level of 2000 mg/kg for 24 hours. The skin was abraded on half the animals (2 male; 1 female) and left intact on the others. Gauze and occlusive dressings were applied to the test sites and the animals were observed for signs of irritation/toxicity on the day of treatment and daily thereafter for 14 days. No deaths occurred. Transient clinical signs noted in four animals were decreased activity and/or decreased food consumption. No necropsy details were provided. The LD_{50} was estimated to be > 2000 mg/kg.

In a second, poorly reported study by Mobil Environmental and Health Science Laboratory (1980b), six New Zealand white rabbits (sex unspecified) received a dermal dose of 2000 mg/kg TCPP (no details of methods were provided). All six rabbits showed some erythema and oedema (grade of irritation not specified) at 24 hours, which had disappeared at 72 hours.

When necropsied, the skin on the test site was scaling or scarred in two animals. The LD_{50} was estimated to be > 2000 mg/kg.

4.1.2.2.2 Studies in humans

No data are available.

4.1.2.2.3 Summary of acute toxicity

The inhalation exposure studies in animals were somewhat equivocal and in general lacking in detailed information. One study yielded an LC_{50} of > 7 mg/L/4hr. A limit test yielded an acute LC_{50} value of >4.6 mg/L/4hr. No deaths occurred at this concentration. Toxic signs observed in this study, and in 2 further poorly reported studies, included mild lethargy, matted fur, acute bodyweight depression and convulsions. From the studies, it appears that TCPP is more toxic when administered whole body as aerosol than by nose-only exposure. This suggests that some of the systemic toxicity observed when TCPP is administered whole body may result from dermal or oral uptake, rather than inhalation. Therefore, it is concluded that TCPP is of low toxicity via the inhalation route.

Studies in rats indicated that TCPP is of moderate toxicity via the oral route of exposure, with LD_{50} values from the better quality studies ranging from 632 mg/kg up to 4200 mg/kg, with the majority of values determined to be <2000 mg/kg. Common clinical and macroscopic signs of toxicity observed on nearly all studies included depression, ataxia, hunched posture, lethargy, laboured respiration, increased salivation, partially closed eyelids, body tremors, pilo-erection, ptosis, haemorrhagic lungs and dark liver and/or kidneys. A NOAEL of 200 mg/kg can be identified for acute oral toxicity. This is taken from the Stropp 1996 study, in which no clinical signs of toxicity were observed in animals dosed with 200 mg/kg TCPP. Based on the results of the acute oral studies, TCPP should be classified with R22, harmful if swallowed.

In a delayed neurotoxicity study conducted in hens, TCPP showed moderate toxicity. The principle effects were reduced mean body weight and food consumption, feather loss and cessation of laying. There was no evidence of inhibited plasma acetylcholinesterase or brain neurotoxic esterase enzyme levels. Therefore, there is no concern for acute delayed neurotoxicity for TCPP.

Studies in rats and rabbits indicated that TCPP is of low toxicity via the dermal route of exposure with LD_{50} values of >2000mg/kg.

4.1.2.3 Irritation

4.1.2.3.1 Skin

Studies in animals

The irritant/corrosive effects of TCPP were tested on the skin of albino HC:New Zealand white rabbits (Bayer, 1991b). This study was in accordance with OECD guideline 404 and in compliance with GLP. A volume of 0.5 ml of the test material was applied via a patch to the shaved skin (6 cm^2) of each of three rabbits. A further patch, moistened with water was also

applied on the opposite shaved dorso-lateral area of the trunk. After an exposure period of 4 hours, patches and dressing were removed and the treated sites were carefully washed with water. Dermal irritation was scored (following the OECD recommended scoring system) and recorded at termination of exposure as well as 1 hr, 24 hrs, 48 hrs, 72 hrs, 7 days and 14 days after exposure.

The test substance did not cause oedema formation in any of the animals (primary irritation score 0). Well-defined erythema (score 2) was evident at the test site of two rabbits 1 hour after termination of exposure but by 24 hrs only slight erythema was evident (score 1) and all evidence of erythema formation had disappeared by 48 hrs. In the other rabbit, slight erythema was observed at 1 hr but had disappeared by 24 hrs. The mean skin irritation index for each of the two most sensitive rabbits was 0.3. TCPP therefore does not have a local irritant potential in the rabbit skin.

A GLP-compliant test was carried out according to OECD guidelines (Inveresk Research International, 1989c) to examine the acute dermal irritation of TCPP in New Zealand White rabbits. 0.5 ml of test material was applied to the intact, clipped skin of three rabbits under a 2.5 cm x 2.5 cm patch of gauze. The patch was appropriately covered and held in position for a period of 4 hours. At the end of the 4 hour exposure period the patches were removed, the test sites were wiped carefully with water-moistened tissues and the skin reactions were assessed 1, 24, 48 and 72 hours after patch removal using the OECD recommended scoring system.

Oedema formation was not recorded in any of the animals. Well-defined erythema was recorded at two treated sites, with very slight erythema at one site 1 hour after patch removal. By 24 hours, all treated sites showed very slight erythema and all sites were normal 48 hours after patch removal. TCPP produced mild transient skin irritation, which was fully reversible by 48 hrs.

TCPP was examined for primary skin irritation properties using a patch test technique on the intact and abraded skin of albino rabbits (SafePharm Laboratories Ltd., 1979d). The method followed was broadly equivalent to OECD guidelines. 0.5 ml of test substance was introduced under a composite patch to the intact and abraded skin of 6 rabbits for 24 hours. The irritation was scored according to the Draize criteria 24 and 72 hrs after removal of the test material. No oedema formation was observed in any of the rabbits. Very slight erythema formation (score 1) was observed at the intact site on 2 animals and at the abraded site of one rabbit 24 hrs after patch removal. All effects had disappeared at 72 hours. The test material produced a very low primary cutaneous irritation score of 0.1.

Two studies were carried out to test the primary skin irritation potential of TCPP on rabbits. Neither of these studies were GLP-compliant. The guidelines followed were reported to be the Code of Federal Regulations (FDA Proposed Revision of Test for Primary Skin Irritants) (Draize Dermal Procedure) and were broadly in line with OECD guidelines.

In the most recent study (Stauffer Chemical Co., 1979b), 0.5 ml of test substance was introduced under a one-inch square gauze patch to the abraded and intact skin of 6 New Zealand Albino rabbits. The patches were secured appropriately for a period of 24 hours. Scoring and evaluation followed the Draize criteria.

TCPP caused very slight erythema in all animals (intact and abraded sites) after 24 hours. This slight erythema (score 1) persisted to 72 hours in one animal (intact skin site). Oedema formation did not occur. The substance resulted in a mean primary irritation score of 0.42.

From a poorly reported earlier study (Stauffer Chemical Co., 1972), it would appear that 0.5 ml of test substance was applied to the abraded and intact skin of 6 rabbits (strain not specified). The test substance did not cause erythema or oedema formation in any of the animals (primary irritation score = 0).

A brief summary report for two primary skin irritation studies (Mobil Environmental and Health Science Laboratory, 1980c and 1981c) were also provided. In both studies, 0.5 ml of TCPP was applied to the abraded and intact skin of 6 New Zealand White rabbits for 24 hours. The test sites were scored for irritancy using the Draize scale at approx. 30 minutes and 72 hours following removal of the test patches (1981 study) or at 24hrs and 72 hrs (1980 study). The primary irritation indices were reported to be 0.5/8.0 (1980 study) and 1.0/8.0 (1981 study), which, according to Federal Regulations means the substance is not a primary skin irritant (i.e. the primary irritation index was less than 5.0/8.0).

In section 4.1.2.2.3, acute toxicity, 2 studies were reported in which erythema and oedema were observed. In the Stauffer Chemical Co, (1979b) study, TCPP was applied to the skin of male and female New Zealand albino rabbits at doses of 2000 and 5000 mg/kg (4 animals per sex and per dose level). The skin was abraded on half the animals and left intact on the others. The test sites of all animals were wrapped with a gauze binder. No deaths occurred over the 14-day observation period. Local effects included slight/mild erythema (observed at both dose levels). In the second study by Mobil Environmental and Health Science Laboratory (1980b), six New Zealand white rabbits (sex unspecified) received a dermal dose of 2000 mg/kg TCPP (no details of methods were provided). All six rabbits showed some erythema and oedema (grade of irritation not specified) at 24 hours, which had disappeared at 72 hours. When necropsied, the skin on the test site was scaling or scarred in two animals.

Studies in humans

No data are available.

4.1.2.3.2 Eye

Studies in animals

The irritant/corrosive effects of TCPP were tested on the eyes of albino HC:New Zealand white rabbits in accordance with OECD Guideline No. 405 and in compliance with GLP by Bayer (Bayer, 1991b). A volume of 0.1 ml of the test material was instilled into the conjunctival sac of one eye of each of three rabbits. The other eye remained untreated and served as control. The treated eyes were rinsed with saline 24 hours post-treatment. There were no signs of irritation in any of the treated eyes at any of the observation times of 24, 48 and 72 hours. One animal had evidence of aqueous humour discharge at 48 hours, but this did not persist.

The eye irritation potential of TCPP was tested on the eyes of New Zealand white rabbits in accordance with OECD/EC guidelines and in compliance with GLP (Inveresk Research International, 1990b). A volume of 0.1 ml of the test material was instilled into the conjunctival sac of one eye of each of three rabbits. The other eye remained untreated and served as control. No corneal or iridial responses were noted. Slight conjunctival redness was noted in 1 animal at 1 hour post-instillation with all 3 treated eyes showing a slight discharge. By 24 hours, all treated eyes were normal.

TCPP was assessed for primary ocular irritation in the albino rabbit (Safepharm Laboratories Ltd., 1979e). A volume of 0.1ml test material was instilled into the right eye of each of 6 male New Zealand rabbits. The other eye remained untreated and was used as a control in each case. The test material produced no evidence of ocular irritation over the 7-day observation period.

TCPP was evaluated for its eye irritancy potential in New Zealand white albino rabbits in a further three different studies.

The most recent study (Stauffer Chemical Co, 1979b) was carried out in accordance with EPA guidelines. A volume of 0.1 ml of the test material was instilled into the left eye of 9 New Zealand white albino rabbits, the right eye acting as a control in each case. In the case of 3 animals, the eye was washed out with water after 20-30 seconds for 1 minute, the remaining 6 were unwashed. There were no signs of any irritation in any of the 9 treated eyes up to 7 days post-administration.

In a very limited reported 1970 study, no irritation was observed over a 72-hour period. However, no details were given e.g. the volume of test material instilled into the eye or the number of rabbits used.

TCPP was also evaluated for its eye irritancy potential in a study by Stauffer Chemical Co. in 1972. A volume of 0.1 ml of the test material was instilled into one eye in each of 6 New Zealand white albino rabbits, the other eye acting as a control in each case. There was no evidence of any irritation in any of the treated eyes up to 72 hours of observation. TCPP was also non-irritant to the rabbit eye under the conditions of the above test.

The eye irritation potential of TCPP was assessed in New Zealand white albino rabbits (Mobil Environmental and Health Science Laboratory, 1980c and 1981d). In the 1980 study, a volume of 0.1 ml of test material was instilled into one eye of each of 6 rabbits. The test eyes remained unwashed throughout the 72 hours of observation. There was no evidence of any irritation in any of the treated eyes over the 72 hours. However, no specification was given for the test material and no individual animal data was provided in the report. A very short summary report was provided for the 1981 study. A volume of 0.1 ml of TCPP was instilled into one eye of each of 6 rabbits. The eyes remained unwashed and were scored for irritancy over 72 hours. No potential for irritation was seen in any of the treated eyes. It should be noted that no test material specification and no individual animal data were given.

Studies in humans

No data are available.

4.1.2.3.3 Respiratory tract

No studies are available. In one of the acute inhalation studies (see section 4.1.2.2.1), (Stauffer Chemical Co., 1979a), reddened lungs were observed in 3 out of 10 females dosed with 4.6 mg/L for 4 hours. In a second acute study, (Environment Affairs and Toxicology Department, 1981a), dyspnea was observed in animals dosed with 5.05 mg/L for 4 hours. These may be indicators of some respiratory irritation, but in the absence of other effects, it is felt that there is not concern for respiratory irritation.

4.1.2.3.4 Summary of irritation

No human data are available, however, there is an extensive database in animals, indicating that TCPP is non-irritant in the rabbit eye and skin. The lack of any substantial skin or eye irritation and the lack of irritation observed in the acute inhalation studies suggest that TCPP would be unlikely to produce significant respiratory tract irritation.

Based on the available studies, TCPP needs no classification for irritation according to EU guidelines.

4.1.2.4 Corrosivity

Results from animal skin and eye irritation studies indicate that TCPP is not corrosive.

4.1.2.5 Sensitisation

4.1.2.5.1 Studies in animals

Skin

No evidence of skin sensitisation was found in a 1979 study (SafePharm Laboratories Ltd., 1979f). Following a range-finding test, 0.1 ml of a 5% solution of TCPP was selected for intradermal induction followed, after 24 hours, by application of undiluted test substance for 48 hours, as the topical induction. 10% sodium lauryl sulphate was applied 24 hours prior to the topical induction. 10 guinea pigs were treated with TCPP and 4 remained untreated as controls. Two weeks after topical induction, the neat test substance was applied for 24 hours under occlusive dressing. There was no significant response after challenge. While this study is not GLP compliant (performed in 1979), its result is considered to be acceptable as a negative result.

In a GLP-compliant Local Lymph Node Assay (LLNA) conducted in accordance with OECD Guideline No. 429 (2002) and EC Method B42 (2004), TCPP was considered to be a non-sensitiser (SafePharm Laboratories Ltd., 2005). In a preliminary test, 25 µl of undiluted TCPP was applied topically to the dorsal surface of the ears of one CBA/Ca mouse for three consecutive days and observations were made up to day 6. No clinical signs were noted. In the main test, groups of four CBA/Ca mice were treated with 25 µl undiluted TCPP or concentrations of 50% or 25% v/v in acetone: olive oil 4:1 for three days. A further group of four mice received the vehicle alone. Five days following the first topical application, all mice were injected via the tail vein with 250 µl of phosphate buffered saline (PBS) containing a total of 20 µCi ^{3}H-methyl thymidine (specific activity 2.0 Ci/mmol). All mice were terminated five hours after injection.

Stimulation indices of 1.55, 1.97 and 1.56 were recorded for concentrations of 25, 50 and 100% v/v, respectively. There were no mortalities or clinical observations and all bodyweights were comparable to those of the control animals.

Respiratory tract

No data are available.

4.1.2.5.2 Studies in humans

No data are available.

4.1.2.5.3 Summary of sensitisation

Evidence from a guinea pig study as well as from a local lymph node assay, indicates that TCPP does not possess significant skin sensitisation potential. No information is available on the respiratory sensitisation potential of TCPP.

4.1.2.6 Repeated dose toxicity

4.1.2.6.1 Studies in animals

In vivo studies

Inhalation

No studies are available.

Oral

Groups of 20 male and 20 female Sprague Dawley rats were fed diets containing 0, 800, 2,500, 7,500 and 20,000 ppm of TCPP for a period of thirteen weeks (Stauffer Chemical Co., 1981). This corresponds to mean substance intake values of 0, 52, 160, 481, and 1349 mg/kg/day for males and 0, 62, 171, 570, and 1745 mg/kg/day for females. Animals were observed for clinical signs and food consumption and weight gain was measured. Blood samples were taken for clinical chemistry (including plasma and erythrocyte acetylcholinesterase concentration) and haematological measurements at initiation of the study, at the midpoint and at termination. Urine samples were taken for urinalysis at six weeks and at termination. Complete necropsy was carried out after terminal sacrifice. Liver, kidney, heart, thyroid and all significant gross lesions from low and mid-dose animals were examined microscopically.

There were no treatment-related mortalities. No clinical observations were considered to be related to treatment. A slight, but statistically significant ($p<0.05$) reduction in mean body weight was apparent from day 22 of the study until termination in the high dose males (7.75% less than controls at day 80) and from day 35 in high dose females (11.8% less than controls on day 80). The mean absolute and relative liver weights were statistically significantly ($p<0.05$) increased in all male groups given TCPP and in females given 7,500 ppm and 20,000 ppm. In males given 800 ppm the group mean relative hepatic weight exceeded the control group mean by 16%. The absolute liver weight in this low dose group was also 16% greater than control. Relative liver weight of males given 20,000 ppm exceeded the control mean by 41% (absolute liver weight was 31% greater than controls for this group). In females given 7500 and 20,000 ppm, the mean relative liver weight exceeded that of controls by 20% and 30% respectively. The only histopathological finding related to this was periportal hepatocyte swelling (hypertrophy) in the high dose groups (7/20 males and 8/20 females). 0/20 male and 5/20 female control animals showed liver periportal swelling. Relative kidney weights were statistically significantly ($p<0.05$) increased in males at the two highest doses

(13% and 16% greater than control). There was some evidence of histopathological change in the renal cortical tubule with the finding of mild degenerative change (hyaline droplet formation) in the two highest dose groups in males (12 animals and 7 animals, respectively) and vacuolation in females dosed with the highest dose (4 animals, compared to 1 control animal). The hyaline droplet formation is a male rat specific nephropathy and is not relevant for humans. Mild thyroid follicular cell hyperplasia was recorded in males at all doses (0/20, 2/20, 2/20, 5/20, and 8/20 at 0, 800, 2,500, 7,500 or 20,000 ppm respectively). This was seen in 5/20 females of the 20,000 ppm group, compared to 0/20 in the control group. There were no significant alterations in clinical chemistry, haematology or urinalysis parameters and no treatment-related changes in plasma, erythrocyte or brain cholinesterase activity. A slightly excessive fatty infiltration indicative of mild bone marrow hypoplasia was seen in three high dose females.

Based on the increase in absolute and relative liver weights, accompanied by mild thyroid follicular cell hyperplasia, observed in males of all dose groups a LOAEL of 800 ppm (equivalent to 52 mg/kg/day) is derived from this study for males. A NOAEL of 2,500 ppm (equivalent to 171 mg/kg/day) is derived for females, based on increased liver weights observed in females dosed at 7,500 ppm and above. The effects on the thyroid in the male animals at all doses and the females at the highest dose could be secondary to altered liver metabolic activity. The LOAEL of 52 mg/kg/day for males will be taken forward to risk characterisation, as males appear to be more sensitive.

In a 28-day study conducted to EC guidelines (Bayer, 1991c), groups of 6 male and female Wistar rats were dosed daily by gavage with 0, 10, 100 or 1000 mg/kg body weight TCPP formulated in DAB 9 peanut oil. The doses were selected based on a preliminary 7-day study in which male Wistar rats were dosed with 0, 10, 100 or 1000 mg/kg body weight. In that preliminary study, the test animals exhibited no reaction to the treatment at any of the doses.

In the 28-day study, animals were checked twice daily for morbidity, mortality and general clinical signs. A detailed individual animal clinical examination was made weekly. Ophthalmic examinations of all animals in the control and highest-dose groups were performed three days before the first treatment and in the fourth week of treatment. Body weights were recorded prior to the first treatment and at weekly intervals thereafter. Laboratory tests of blood and urine from all animals of every group were carried out at study termination. The organs and tissues of necropsied animals at the end of the study were subjected to detailed gross pathological inspection.

One male and two females in the 1000 mg/kg treatment group died, the male at the end of the first week of treatment and the two females at the beginning of the second week. The male was in poor general condition, was emaciated and exhibited a bloody muzzle. The authors of the study report have indicated that the presumed cause of death was treatment error. Since no male died in the preliminary study at levels up to and including 1000 mg/kg, the mortality of the male animal is not considered to be test substance related. The two females exhibited no clinical signs prior to or on the day they died. These two mortalities could be treatment related.

Regarding clinical signs during the study, all females in the 1000 mg/kg group exhibited a squatting position at 10 mins after dosing for the first 3 days of dosing. This persisted for about three to five hours on the first two days and for approximately one hour on the third day. No further clinical signs were observed. The ophthalmic examinations of the control and highest-dose group animals prior to study initiation and in the fourth week of the study indicated no unusual findings.

The body weight determinations of TCPP-treated males and females indicated slightly higher results than those of control animals, but the differences were minor and did not reach statistical significance. There was no dose-response effect and so the effect is not considered treatment-related. The mean daily food intakes in all groups were comparable to that of controls.

There were no changes in white and red blood cell populations in any treatment group when compared to controls. In male rats in the two highest treatment groups, the monocyte count was statistically significantly ($p<0.05$) lower when compared to the control group (1.7% for the 100 mg/kg treatment group and 1.6% for the 1000 mg/kg group, compared to a control value of 4.1%). The count was also reduced in the 10 mg/kg treatment group (2.3%), but this did not reach statistical significance. There were no changes in alkaline phosphatase or aspartate aminotransferase enzyme activities in any of the treated animals. A statistically significant ($p<0.01$) depression of alanine aminotransferase activity was seen in high-dose male (by 46%) and female (by 34%) animals. The low dose female group saw a statistically significant ($p<0.01$) increase in this enzyme's activity (increased by 28%). There was also an increase in glucose observed in the treated male animals. This reached statistical significance ($p<0.05$) in the low (4 mmol/L) and high dose ($p<0.01$) (4.05 mmol/L) groups when compared to a control value of 3.57 mmol/L. There were some changes observed in the clinical chemistry investigations, but these appeared to be isolated deviations from the controls and were relatively minor and not dose-dependent. For these reasons, they are not considered to be of toxicological significance. Potassium was statistically significantly ($p<0.05$) increased in the low dose male animals (5.5 mmol/L) and inorganic phosphorous was statistically significantly ($p<0.05$) decreased in the mid-dose females. The semi-quantitative determinations of the pH, bilirubin, glucose, urobininogen and ketonic bodies levels indicated no changes among any treatment group when compared to levels in control animals. Quantitative determination of creatinine levels indicated a statistical significant ($p<0.05$) increase in creatinine in the high-dose males when compared to controls (78 mcmol compared to 63 mcmol). Protein was also statistically significantly ($p<0.01$) increased in this treatment group (12.3 mg compared to 9.2 mg).

No macroscopic findings attributable to the test substance were reported at scheduled necropsy. The animals that died during the study exhibited dark red dis-colourations in the lungs. The absolute liver weights were statistically significantly ($p<0.01$) increased by 30% and 42% in male and female high dose animals respectively, when compared to controls. The relative liver weights were statistically significantly ($p<0.01$) increased by 27% and 34% in high dose males and females respectively, when compared to controls. In addition, relative liver weights were statistically significantly ($p<0.05$) increased in the low-dose male animals (increased by 6%) and in the mid-dose female animals (increased by 7%). The increase in liver weights was accompanied by slight hypertrophy of the periacinary hepatocytes in one of the mid-dose males (remainder of mid dose males exhibited minimal periacinary hepatocyte hypertrophy) and in all of the high-dose males. No hepatic alterations were noted in treated females.

Based on the increased liver weight changes in the high dose groups, accompanied by hepatocyte hypertrophy in all high–dose males and one mid-dose male and in addition to the changes in ALAT activity observed in high-dose animals, a NOAEL of 100 mg/kg/day can be identified from this study.

Groups of 10 male and 10 female Sprague Dawley rats were fed diets containing 0, 4,200, 6,600, 10,600 and 16,600 ppm of TCPP for a period of two weeks (Stauffer Chemical Co.,

1980a). This corresponds to mean substance intake values of 417, 648, 1015, 1636 mg/kg/day for males and 382, 575, 904, 1517 mg/kg/day for females.

Animals were observed for clinical signs, food consumption and weight gain was measured and blood samples taken for clinical chemistry (BUN, serum glutamic pyruvic transaminase, glutamic oxaloacetic transaminase, alkaline phosphatase, glucose, total and direct bilirubin and plasma and erythrocyte acetylcholinesterase concentration) and haematological measurements (packed cells volume, total erythrocytes, total leucocytes, haemoglobin concentration and platelet count). Necropsy was carried out after terminal sacrifice.

There were no clinical signs of toxicity other than variable incidence of alopecia and ulcerative dermatitis in the shoulders and head regions of all rats. There was a significant ($p<0.05$) reduction in weight gain in high dose males during week one. Compared to starting weights, the weights of males given the test substance at a dietary concentration of 16,600 ppm were statistically significantly reduced by an average of 22% on study day 7. The males in the group that received 10,600 ppm showed a weight gain of 9.5%. This was in comparison to control animals that showed a weight gain during the first week of 17.8% (the other 2 treatment groups, 4,200 and 6,600 ppm, showed weight gains comparable to controls at 16.2 and 17.1%, respectively). Females in all treatment groups showed comparable weight gains to controls during the first week. Weight gain was not different from controls in either sex during week two. Male rats in the two highest treatment groups ate statistically significantly less in the first 3 days of the study. They ate on average 15.67 g and 12.89 g respectively, compared to an average in the control group at this time point of 21.7 g. For the remainder of the study food consumption of all treated groups was similar to control groups. Clinical pathology parameters including haematology, serum chemistry and plasma and erythrocyte acetylcholinesterase concentrations were unaltered by treatment when compared to controls. There were no treatment-related findings at necropsy. A NOAEL of 10,600 ppm (equivalent to 1015 mg/kg in males) can be derived from this study, based on an adverse effect on body weight gain in male animals treated with the highest dose.

In the dose range finding study of a developmental study, (Kawasaki *et al*., 1982), groups of 5 female rats were dosed (forcibly by mouth) each day for 7 days with 8, 40, 200 or 1000 mg/kg TCPP suspended in olive oil. Body weight gain was unaffected and no abnormal behaviour or adverse symptoms were recorded. One animal dosed at 1000 mg/kg died on day 2. Kidney weights were significantly increased at 40 mg/kg (10% increase when compared to controls), 200 (increased by 20%) and 1000 mg/kg (increased by 10%). No dose-response effect was observed. Liver weight was also significantly increased at 1000 mg/kg (increased by 10% when compared to control values).

In an oral two-generation reproductive toxicity study in rats (TNO Quality of Life, 2007), 28 animals/sex/group received TCPP in the diet, corresponding approximately to 0, 85, 293 and 925 mg/kg bw/day for males and 0, 98.6, 329.9 and 988.2 mg/kg bw/day for females, over two successive generations. The animals were fed diets containing the test substance from the start of the study, during the premating period of at least 10 weeks, throughout gestation and lactation until sacrifice. Dams were allowed to raise one litter per generation. On PN21, litters were weaned and 28 male and 28 females were selected for the next generation. Animals were observed for clinical signs, and food consumption and body weight gain were recorded. Females were sacrificed at or shortly after weaning and males after at least 11 weeks of exposure.

There were no treatment related clinical signs. During premating, an F1 male of the mid dose group was found dead on Day 41 and a female of the same group was killed in moribund on

Day 50. The cause of death or the cause of the moribund condition could not be detected at necropsy. There were no other mortalities. A treatment related decrease in body weights was observed in F0 and F1 males of mid and high dose groups. During premating, body weights of females in F1 generations were decreased in the mid and high dose groups. During gestation, the mean body weights were decreased in high dose females in F0 females and in mid and high dose F1 females. Body weights were decreased in mid and high dose F1 females during lactation. Mean food consumption was decreased in F0 and F1 males and females of the high dose group and in F0 males and females and F1 females of the mid dose group.

Terminal body weights were decreased in mid and high dose males of both generations and in females of the F1 generation. Organ weight changes in both males and females are presented in **Tables 4.43** and **4.44**, respectively.

Table 4.43 Summary of absolute and relative organ weight changes in males in the 2-generation reproductive toxicity study

	F0-generation						F1-generation					
	Low		Mid		High		Low		Mid		High	
Organ weight	**A**	**R**	**A**	**R**	**A**	**R**	**A**	**R**	**A**	**R**	**A**	**R**
Terminal body weight	-		↓		↓		-		↓		↓	
Adrenal	-	-	-	-	-	-	-	-	↓	-	-	↑
Brain	-	-	-	-	↓	↑	-	-	↓	-	↓	↑
Epididymides	-	-	-	-	↓	-	-	-	-	-	-	↑
Kidneys	-	-	-	-	↓	-	↓	-	↓	-	↓	↑
Liver	-	↑	-	↑	-	↑	-	-	-	-	-	↑
Pituitary	-	-	-	-	-	-	-	-	-	-	↓	-
Prostate	-	↑	-	-	-	-	-	-	-	-	↓	-
Seminal vesicles	-	-	↓	-	↓	-	-	-	↓	-	↓	-
Spleen	-	-	-	-	↓	-	-	-	↓	-	↓	-
Testes	-	-	-	-	↓	-	-	-	-	↑	-	↑
Thyroid	-	-	-	-	-	↑	-	-	-	-	-	↑

A: absolute weight; R: relative weight

Table 4.44 Summary of absolute and relative organ weight changes in females in the 2-generation reproductive toxicity study

	F0-generation						F1-generation					
	Low		Mid		High		Low		Mid		High	
Organ weight	**A**	**R**	**A**	**R**	**A**	**R**	**A**	**R**	**A**	**R**	**A**	**R**
Terminal body weight	-		-		-		-		↓		↓	
Adrenal	-	-	-	-	-	-	-	-	-	-	-	-
Brain	-	-	-	-	-	-	-	-	-	-	↓	↑
Kidneys	-	-	-	-	-	-	-	-	-	-	↓	-
Liver	-	-	-	-	↑	↑	-	-	-	-	-	↑
Ovaries	-	-	-	-	↓	-	-	-	-	-	-	-
Pituitary	-	-	-	-	↓	↓	↓	↓	↓	-	↓	↓
Spleen	-	-	↓	↓	↓	↓	-	-	-	-	↓	↓
Thyroid	-	-	-	-	-	-	-	-	-	-	-	-
Uterus	↓	↓	↓	↓	↓	↓	-	-	-	-	↓	↓

A: absolute weight; R: relative weight

It is noted that only the relative liver weight was increased in low dose males and was not accompanied by any increase in absolute organ weight or clinical chemical effects. Therefore, this can be considered an adaptive effect and therefore not adverse.

There were no treatment related macro-or microscopical changes observed in the F0 or F1 parental animals. The incidence of mineralisation in the kidneys of the high dose F1-females was higher than in the controls (5/28 in control versus 11/28 in the high dose group). However, kidney mineralisation is a common finding in female rats and therefore not thought to be treatment related.

The low-dose of approximately 99 mg/kg for females is considered to be the LOAEL for parental toxicity. This is based on decreased body weight and food consumption seen in mid and high dose parental animals and the effects on uterus weight seen in all dosed F0 animals. For males, a NOAEL of approximately 85 mg/kg is derived for parental toxicity, based on decreased body weights, food consumption and organ weight changes observed at mid and high dose groups.

Dermal

No studies are available.

4.1.2.6.2 Studies in humans

No studies are available.

4.1.2.6.3 Summary of repeated dose toxicity

A study is available in which male and female rats were fed diets containing TCPP for 13 weeks at concentrations corresponding to mean substance intake values of up to 1349 mg/kg/day and 1745 mg/kg/day for males and females respectively. This study indicated the liver and thyroid to be the main target organs affected by TCPP. Effects observed included statistically significant increases in absolute and relative liver weights in males at all doses and females at the two highest doses, periportal hepatocyte swelling in high dose groups and mild thyroid follicular cell hyperplasia in males at all doses and females at the highest dose. Based on the increase in both absolute and relative liver weights, accompanied by mild thyroid follicular cell hyperplasia observed in males of all dose groups, a LOAEL of 52 mg/kg/day is derived and taken forward to risk characterisation. This LOAEL is taken forward in preference to the NOAEL which was identified in a 4-week study in which rats were dosed with TCPP at concentrations of 0, 10, 100 and 1000 mg/kg/day, as it was derived from a study of longer duration. The 4-week study also showed the liver as the target organ, with increased liver weight changes observed in the high dose groups, accompanied by hepatocyte hypertrophy in all high–dose males and one mid-dose male and changes in ALAT activity in high-dose animals.

A two-week study in which rats were fed diets of TCPP at concentrations corresponding to mean substance intake values of up to 1636 mg/kg/day for males and 1517 mg/kg/day for females showed no major clinical signs of toxicity. There was a significant reduction in weight gain and food consumption in high dose males during week 2, but there were no other significant findings.

In a 2-generation reproductive toxicity study in which rats were fed TCPP in the diet over two successive generations, the low-dose of 99 mg/kg for females is considered to be the LOAEL for parental toxicity. This is based on decreased body weight and food consumption seen in mid and high dose parental animals and the effects on uterus weight seen in all dosed animals. For males, a NOAEL of approximately 85 mg/kg is derived for parental toxicity, based on decreased body weights, food consumption and organ weight changes observed at mid and high dose groups.

No data are available on inhalation and dermal repeated dose toxicity.

4.1.2.7 Mutagenicity

4.1.2.7.1 Studies *in vitro*

Studies in bacteria

In a plate incorporation mutagenicity test, TCPP did not produce any increase in the number of revertants (Zeiger *et al.,* 1992). *Salmonella typhimurium* strains TA-1535, 1537, 97, 98 and 100 were tested with doses of 3.3, 10, 33, 100, 333, 666 and 1000 μg/plate both in the presence and absence of Aroclor-induced rat liver S9. Each dose was tested in triplicate. A second independent experiment did not appear to be carried out. The solvent used was DMSO. Positive controls without metabolic activation include sodium azide (TA-1535 & 100), 4-nitro-o-phenylenediamine (TA-1538 & 98), mitomycin C (TA-102), methyl methanesulfonate (TA-104) and 9-aminoacridine (TA-97 & 1537). The positive control with

metabolic activation for all strains was 2-aminoanthracene. All positive controls gave the expected response.

Negative results were obtained in a plate incorporation assay using *Salmonella typhimurium* strains TA-1535, 1537, 1538, 98 & 100 (Stauffer Chemical Co., 1978a). Test compound was added to each strain to give a final dose of 1, 10, 100, 1000 and 5000 nl/plate both in the presence and absence of Aroclor or phenobarbital-induced rat liver S9. Positive controls without metabolic activation included ethyl methanesulfonate (TA-1535, 100 & D4), quinacrine mustard (TA-1537) and 2-nitrofluorene (TA-1538 & 98). The positive control with metabolic activation for all strains was 2-aminoanthracene. The top dose was toxic in all *S. typhimurium* strains in the presence of phenobarbital-induced rat liver S9 fraction with a significant reduction in the number of revertant colonies per plate. The number of revertants due to positive controls was significantly increased compared with solvent controls.

TCPP was shown to be non-mutagenic when tested at concentrations of 30, 100, 330 and 1000 nl/plate in *Salmonella typhimurium* strains TA-1535, 1537, 1538, 98 & 100 both in the presence and absence of Aroclor-induced rat liver S9 (Mobil Environmental and Health Safety Laboratory, 1980e). Positive controls without metabolic activation include methylnitrosogaunidine (TA-1535 & 100), 2-nitrofluorene (TA-1538 & 98) and 9-aminoacridine (TA-1537). The positive control with metabolic activation for all strains was 2-aminoanthracene. The top dose was toxic in all strains in the presence or absence of S9 fraction. The number of revertants due to positive controls was significantly different from that due to solvent controls.

In a non-GLP study, the mutagenicity of TCPP was investigated in *Salmonella typhimurium* strains TA-1535, 1537, 1538, 98 & 100 (Stauffer Chemical Co., 1976). Each strain was tested with doses of 1, 10, 100 and 1000 nl/plate both in the presence and absence of Aroclor-induced rat liver S9. The highest dose produced some evidence of toxicity. Positive controls without metabolic activation include methylnitrosoguanidine (TA-1535, 100), 2-nitrofluorene (TA-1538 & 98) and quinacrine mustard (TA-1537). Positive controls with metabolic activation include 2-anthramine (TA-1535 & 100), 2-acetylaminofluorene (TA-1538 & 98) and 8-aminoquinoline (TA-1537). All positive controls gave the expected response. TCPP was not mutagenic in any of the tester strains in the presence or absence of S9 fraction.

In a GLP study conducted to OECD and EC guidelines, TCPP was shown to be non-mutagenic in *Salmonella typhimurium* and *Escherichia coli* strains (SafePharm Laboratories Ltd., 1992). *S. typh*. strains TA 1535, TA 1537, TA 98 and TA 100 and *E. coli* strain WP2uvrA- were treated with TCPP by the Ames plate incorporation method in triplicate at dose levels of 8, 40, 200, 1000 and 5000 µg/plate both in the presence and absence of Aroclor 1254-induced rat liver S9. The positive controls without metabolic activation, N-ethyl-N'-nitro-N-nitrosoguanidine (TA 100, TA 1535 and WP2uvrA-), 9-aminoacridine (TA 1537) and 4-nitroquinoline-1-oxide (TA 98), and the positive controls with metabolic activation, 2-aminoacridine (TA 1535 and WP2uvrA-) and benzo[a]pyrene (TA 100, TA 1537 and TA 98), produced marked increases in the numbers of revertant colonies. The solvent control, dimethyl sulphoxide, gave revertant colony counts within the normal range.

TCPP was shown to be non-mutagenic at doses of 0.3, 1, 3, and 10 µmole/plate (in the presence and absence of Kanechlor 500-induced liver S9 fractions) in *Salmonella typhimurium* strains TA-1535, 1537, 1538, 98 & 100 in a non-GLP study (Nakamura *et al.,* 1979). The purity of the test compound was 67%; major contaminants include bis(1-chloromethyl)(2-chloropropyl) phosphate (28%) and bis(2-chloropropyl)(1-

chloromethylethyl) phosphate (5%). There was no data provided regarding positive controls. The experimental methods were those of Ames *et al.,* 1975.

The mutagenicity of TCPP was investigated in a plate incorporation assay in *Salmonella typhimurium* strains TA-1535, 1537, 1538, 98 & 100. (Tenneco Chemicals Inc., 1977a). In a preliminary study, the ED_{50} of the test agent was chosen as the highest dose for the assay. TCPP at doses of 1, 5, 10, 25 and 50 µl/plate was added both in the presence and absence of Aroclor-induced S9. Positive controls without metabolic activation include methylnitrosoguanidine (TA-1535 & 100), 2-nitrofluorene (TA-1538 & 98) and 9-aminoacridine (TA-1537). Positive controls with metabolic activation include 2-aminoanthracene (TA-1535), 6-aminochrysene (TA-1537), 2-aminofluorene (TA-1538) and aflatoxin B_1 (TA-98 & 100). In the assay without S9 fraction, 10 µl TCPP/plate gave a revertant count more than twice the negative control value for TA-1535. However, this was an isolated finding and there was no dose-response. The revertant colony count for strain TA-98 was also more than twice the negative control value for all doses tested. Again there was no dose-response relationship. The experiment was not repeated in the cases of an increased frequency of mutation and so there was no independent confirmation of the findings. The number of revertants due to positive controls was significantly different from that due to solvent controls. However, in light of the fact that results from all other *in vitro* studies in bacteria were negative and the fact that there was no dose-response effect observed, it is concluded the results of this study are not reliable evidence.

The mutagenicity of TCPP and its possible metabolites, 1,3-dichloro-2-propanone, 1,3-dichloro-2-propanol and 3-chloro1,2-propanediol, was evaluated in a standard Ames test and in a modified quantitative suspension assay using the *Salmonella typhimurium* strain, TA 100 in the presence of Aroclor 1254-induced rat liver S9 and phenobarbital-induced mouse liver S9 (Majeska & Matheson, 1983). In the standard plate assay, TCPP and 1,3-dichloro-2-propanol showed dose-related responses at 500 µg/plate and lower. 1,3-dichloro-2-propanone showed increases in revertants at less than 50 µg/plate. In the quantitative assay, TCPP showed responses at doses resulting in ≤3% survival whereas 1,3-dichloro-2-propanol induced responses at ≤80% survival and 1,3-dichloro-2-propanone at ≤30% survival. 3-chloro1,2-propanediol was non-mutagenic in both assays. There was no record of positive or negative controls used.

In a study by Föllmann & Wober (2006), TCPP was evaluated in Ames test using the pre-incubation procedure, in the presence and absence of rat S9-mix, with *Salmonella typhimurium* strains TA97a, 98, 100, 102, 104, 1535, 1537 and 1538. There was no mutagenic effect observed in any strain, either in the presence or absence of metabolic activation.

The mutagenicity of TCPP was investigated in the E. coli repair test using strains W3110/po1A+ and p3478/polA- at doses of 2, 10 and 20 µl/plate (Tenneco Chemicals Inc, 1977b). p3478/polA- is more sensitive to agents that covalently bind DNA. Methyl methane sulfonate was used as a positive control and chloramphenicol was used as a negative control. TCPP did not cause zones of inhibition of either strain of E. coli in the presence or absence of S9 fraction. Large differences between the strains in the extent of the inhibition zone were recorded for the positive control while the difference recorded for chloramphenicol was insignificant. Therefore, TCPP does not elicit a differential cell mortality between repair-deficient and repair-competent E. coli strains.

Studies in Fungi

In a plate incorporation mutagenicity test for the detection of induced gene conversion with *Saccharomyces cerevisiae* strain D4, doses of TCPP of 1-5000 nl/plate did not produce any increase in the number of revertants, either in the presence or absence of Aroclor or phenobarbitol-induced liver S9 fraction (Stauffer Chemical Co., 1978a). TCPP was tested over a series of doses such that there was evidence of a toxic effect. The low dose was below a dose that demonstrated any toxic effect. The positive controls used gave the expected responses.

In a second gene conversion test with Saccharomyces cerevisiae strain D4, negative results were also obtained with doses of TCPP from 0.001 µl to 1 µl/plate both in the presence and absence of Aroclor-induced liver S9 fraction (Stauffer Chemical Co., 1976). TCPP was tested over a series of doses such that there was evidence of a toxic effect. The low dose was below a dose that demonstrated any toxic effect. The positive control without metabolic activation was methylnitrosoguanidine and the positive control with metabolic activation was dimethylnitrosamine. Both of these positive controls gave the expected responses.

Studies in mammalian cells

TCPP did not induce forward mutation at the TK locus in L5178Y mouse lymphoma cells (Stauffer Chemical Co., 1978b) at doses of 80, 160, 240, 320 and 480 nl/ml in the presence or absence of S9 fraction and an exposure time of 4 hours. This corresponds to 103, 206, 310, 412 and 619 µg/ml, respectively. These doses were based on preliminary studies in which doses of ≥ 640 nl/ml resulted in precipitates and were very toxic to the cells. The positive control without metabolic activation was ethyl methylsulfonate while the positive control with metabolic activation was dimethyl nitrosamine. The positive controls elicited a significantly greater number of total mutant clones compared to solvent controls.

In a briefly reported study, TCPP showed evidence of mutagenicity in L5178Y mouse lymphoma cells in the presence of rat liver S9 fraction when tested to a dose of 580 nl/ml (corresponding to 748 µg/ml) (Environmental Affairs & Toxicology Department, 1981c). However, this dose produced almost total cell death. Therefore, the maximum dose tested was 366 nl/ml (corresponding to 472 µg/ml), which gave an acceptable growth rate of 18%. The assay was performed once in the absence of liver S9 fraction and twice in its presence. In the absence of liver S9 fraction, cell cultures exposed to TCPP did not show an increase in the mutagenic frequency at the highest acceptable dose. In the presence of liver S9 fraction, evidence of mutagenicity at the maximum dose tested dose was obtained in the first assay but no dose-response was observed. There was no indication in the report if this effect was statistically significant and no data were supplied, so the effect could not be quantified. In the second assay, no dose-related toxicity was observed, but a dose-related mutagenic response was obtained at all doses. The highest dose showing an acceptable growth of 42%, exhibited an induced mutation frequency 18 times that of the negative controls. This is the only information supplied in the report. Therefore, it is not possible to give an indication of the dose-response relationship i.e. mutation frequency. There was no indication of a positive control used in the study and what response this gave. It is therefore felt that the positive result obtained in this mouse lymphoma study is questionable and therefore considered to be equivocal. In the WHO Environmental Health Criteria (EHC) series (no. 209), this study is evaluated and is reported to be equivocal.

In a confirmatory mouse lymphoma study carried out to GLP and in accordance with OECD Guideline No. 476, TCPP was shown to be mutagenic in the presence of metabolic activation

(Covance Laboratories Ltd., 2005). The cytotoxicity of a mixture of four samples of TCPP, mixed in equal measure, was initially examined both in the absence and presence of Aroclor 1254 induced rat liver S9 fraction at doses of 62.5, 125, 250, 500, 1000 and 2000 μg/ml. Complete toxicity was observed at 500 μg/ml in the absence of S9 and at 250 μg/ml in the presence of S9. Therefore, for the first experiment doses of 150, 200, 250, 300, 400 and 450 μg/ml without S9 and 80, 100, 112.5, 125, 137.5, 150 and 200 μg/ml with S9 were tested for viability and trifluorothymidine (TFT) resistance. A 3-hour treatment incubation period was employed for all treatments in the presence and absence of S9 mix. The highest dose of 500 μg/ml without S9 was considered too toxic and was excluded from viability and (TFT) plating. For the second experiment the following doses were plated for viability and TFT resistance: 100, 150, 200, 250, 300, 350, 400 and 450 μg/ml without S9 and 25, 50, 75, 100, 125, 150, 175 and 200 μg/ml with S9. The highest doses of 475 and 500 μg/ml without S9 and 250 and 300 μg/ml with S9 were excluded from plating due to excessive toxicity. DMSO was used as the negative control and the positive controls were 4-nitroquinoline without S9 mix and benzo(a)pyrene with S9.

In the absence of metabolic activation, there was no significant increase in mutation frequency in either the first or second experiment up to toxic doses. In the presence of S9, statistically significant increases in mutation frequency were observed at the highest doses in both the first (137.5, 150 and 200 μg/ml) and second (150, 175 and 200 μg/ml) experiment. The relative total growth (RTG) at these doses were 52, 58 and 41% in the first experiment and 28, 18 and 10% in the second experiment, respectively. Large and small colonies were scored for the doses at which statistically significant increases in mutation frequency were observed. Increases in both small and large colony mutant frequencies were noted as well as a clear increase in the proportion of small colony mutants, indicating potential clastogenic activity.

Three unscheduled *in vitro* DNA synthesis (UDS) assays have been carried out with TCPP. In the first study, conducted in accordance with GLP and OECD guidelines, TCPP did not induce unscheduled DNA synthesis in adult male rat liver primary cell cultures (Bayer AG, 1991d). Doses of 12.5, 25, 50, 100, 150 and 200 μg/ml TCPP and 10 μCi ^{3}HTdR (16.3 Ci/mmole) were applied to cells for 18-24 hours. Cell viability before treatment was 82.2%. The minimal survival at 200 μg/ml was 47.3%. This dose was omitted from any calculations. Minimum survival in the other doses was 70%. DMSO was used as a solvent and negative control. The positive control was 2-acetyl aminofluorene. The criteria specified for a positive response included an average net nuclear grain count of 5 and ≥ 20% of the cells in repair. On this basis there was no statistically significant increase in DNA repair in any of the dose groups. The positive control was moderately toxic and the increased nuclear grain count was biologically and statistically significant.

In a second assay, TCPP was not genotoxic when investigated in a rat hepatocyte/DNA repair assay (Williams *et al*., 1989). Hepatocytes were isolated from adult male F344 rats with preparation viabilities of ≥ 90%. Monolayer cultures were simultaneously exposed to test material (5 x 10-3M) and 10 μCi [3H]thymidine/ml (60-80 Ci/mmole) for 18-20 hours. 2-Aminofluorene was used as a positive control.

In the third UDS assay, human embryonic lung WI-38 cells were treated with a dose range of 5 to 100 nl/ml TCPP (corresponding to 6.45 to 129 μg/ml) (based on toxicity observed in a preliminary test above 0.1 μl/ml) ± Aroclor-induced liver S9 fraction (Stauffer Chemical Co., 1978c). Exposure time was 1.5 hours. This is a much shorter exposure time than in the previous 2 assays. The positive control without metabolic activation was N-methyl

nitrosoguanidine while the positive control with metabolic activation was benzo(a)pyrene. These positive controls gave expected results. Results were presented as DPM/μg DNA. In the absence of metabolic activation, the results for doses of 5, 10, 50 and 100 nl/ml TCPP (corresponding to 6.45, 12.9, 64.5 and 129 μg/ml) were 124%, 153%, 141% and 100% respectively, when compared to the control, taken as 100%. In the presence of metabolic activation, the results were 100%, 148%, 95% and 81%, respectively when compared to the control value. Although the results obtained suggest a possible effect, especially at the 10nl/ml dose, the data, both in the presence and absence of metabolic activation, do not show a clear dose-response relationship in the absence of any toxicity to the cells. Given this, combined with the clear negative result obtained in Bayer 1991d study above and the fact that the study is older, performed in a non-standard cell-line, this result appears questionable. When WHO evaluated this study in EHC 209, these results were reported as equivocal.

TCPP was shown to induce transformed foci in BALB/3T3 cells when tested at doses of 39, 78, 156, and 312 nl/ml (corresponding to 50, 100, 200 and 400 μg/ml) (625 nl/ml [800 μg/ml] was toxic to the cells) (Stauffer Chemical Co., 1978d). Exposure was for 72 hours. The positive control was 3-methyl cholanthrene and DMSO was the negative control. No metabolic activation system was incorporated in this study. The mean number of foci per plate was significantly increased in the positive control (270%) and at each dose level tested (ranging from 230-244% of negative control at doses of 39-312 nl/ml [50-400 μg/ml]). No dose-response relationship was observed.

In another study, TCPP did not induce significant numbers of transformed foci in BALB/3T3 cells when tested at doses of 2.5, 5, 10, 20 and 40 nl/ml (corresponding to 3.22, 6.45, 12.9, 25.8 and 51.6 μg/ml, respectively). (Stauffer Chemical Co., 1980b). Eight to ten replicates per dose level were prepared. Exposure was for 72 hours. After dosing, the plates were washed and replenished with fresh medium. The plates were then incubated for an additional 3-4 weeks with twice weekly medium changes. Plates were monitored daily for cell integrity. After incubation, cells were washed and stained with Giemsa and all potential foci were examined microscopically. Results were presented as the number of foci per set of replicate plates. The dose levels tested were based on preliminary tests and gave a survival rate of ≥ 75%. The positive control was 3-methyl cholanthrene (5 μg/ml) and the negative control, DMSO. No metabolic activation system was incorporated in this study. The positive control gave results in the expected range.

The potential for TCPP to induce DNA strand breaks was investigated in an *in vitro* Comet assay in V79 cells (Föllmann & Wober, 2006). V79 cells were incubated for 24 hours with 1μM or 1mM of TCPP in the presence or absence of rat S-9 mix. Following incubation, cells were washed and a single cell suspension prepared, with the cell density adjusted to 8 x 10^6 cells per ml medium. An aliquot of the suspension (25 μl) was added to 75 μl agarose, which was then transferred to an agarose-covered slide to solidify. The slides were placed in cold lysis buffer overnight after which they were electrophoresed (25 V and 300 mA for 30 minutes). The slides were neutralised and stained with ethidium bromide and 100 cells of each concentration were analysed by measuring tail lengths using Comet Assay II software, with observation made at 400x magnification using an epifluorescent microscope DMRB, equipped with an excitation filter of 515-535 nm, a 100 W mercury lamp and a barrier filter at 590 nm.

No significant difference in tail length was observed between TCPP treated cells and vehicle control (DMSO). It was concluded that TCPP did not induce DNA strand breaks either in the presence or absence of S-9 mix.

4.1.2.7.2 Studies *in vivo*

An *in vivo* Comet assay in the rat liver (Covance Laboratories Ltd., 2006) was carried out to GLP and, as there is currently no official test guideline, in accordance with most recent methodology available (recommendations of IWGTP workshop and current literature). The Comet assay is a technique for investigating DNA breakage and damage in individual mammalian cells by using electrophoresis of DNA which has been unwound under alkaline conditions (> pH 13). Electrophoresis results in the charged DNA being drawn away from the nucleus, with relaxed and broken DNA fragments migrating further than undamaged DNA complexes. The use of alkaline conditions enables single stranded and alkaline labile sites as well as double stranded DNA breaks to be expressed during electrophoresis.

In this study, groups of six male rats were administered TCPP in corn oil as a single dose via oral gavage at either 750 or 1500 mg/kg. The choice of dose levels was based on previous toxicity studies on TCPP, which identified 1500 mg/kg as the maximum tolerated dose. In the absence of any gender differences in the acute toxicity studies with rats, only male animals were tested. The negative (vehicle) control group received corn oil only. The positive control group received a single gavage dose of ethyl methansulfonate (EMS) at 250 mg/kg three hours prior to necropsy. The liver was chosen for comet analysis as TCPP caused an increased mutation frequency in the mouse lymphoma assay in the presence of S9 and induced liver enlargement in repeated dose toxicity studies.

The TCPP or the vehicle control treated rats were killed 3 or 24 hours after dosing. At necropsy, TCPP animals were examined internally for signs of cytotoxicity. For each animal, a section of the liver was removed, cut into small pieces and pushed through bolting cloth of pore size 150 μm to produce single cell suspensions. An aliquot of the cell suspension was then added to agarose, plated onto four slides and allowed to gel. Three slides were placed in lysis buffer for 1 hour, then transferred to electrophoresis buffer (pH > 13) to allow DNA to unwind for 30 minutes, after which the slides were electrophoresed at 0.7 V/cm for 40 minutes. At the end of the electrophoresis period slides were neutralised, dried and stained with ethidium bromide for comet analysis. The fourth slide was neutralised and used to determine the degree of highly damaged cells in the cell suspensions (diffusion analysis). Scoring of slides was made using fluorescence microscopy at x 200 magnification and Comet scoring was performed using Perceptive Instruments 'Comet Assay III' image analysis system. Measurements of tail moment and tail intensity (% DNA in tail) were obtained from 100 cells per animal. The tail moment is defined as [tail profile centre of gravity – head profile centre of gravity] x tail % DNA, and therefore gives a measure incorporating both tail length and tail content. Each slide was also examined for possible indications of cytotoxicity, with cells with 'clouds', which is a morphology indicative of highly damaged cells often associated with severe cytotoxicity, necrosis or apoptosis, were not included in Comet scoring. Diffusion slides were scored by assessing 100 cells per slide.

Lethargy was observed in one animal at 1500 mg/kg, with no other clinical signs noted. At necropsy, the livers of animals dosed at 1500 mg/kg were noted to be darker in appearance than those of the 750 mg/kg or vehicle control groups. Cloud assessment and analysis of diffusion slides of TCPP and vehicle control treated animals demonstrated low levels of cells (less than 15%) with significantly fragmented DNA, indicating little cytotoxicity, necrosis or apoptosis in the cell preparations. Comet analysis of livers treated with TCPP, sampled at either 3 or 24 hours post dosing, had slightly elevated group mean tail moments and intensities compared with the concurrent control. However, there was no dose response, the increases were within the degree of variation frequently seen with this assay and also fell within the historical control range. The positive control induced a clear increase in tail

moment and tail intensity. **Table 4.45** below summarises the group mean data, including tail moment and tail intensity values.

Table 4.45 Summary of group mean data for *in vivo* Comet assay with TCPP

Treatment group (mg/kg/day)	Sample time (hrs after dosing)	Group mean % clouds	Group mean % diffused cells	Tail Moment ± SEM	Tail Intensity ± SEM
Vehicle (0)	3	2.17	6.33	0.29 ± 0.04	2.20 ± 0.20
TCPP (750)	3	3.08	4.83	0.48 ± 0.04	2.94 ± 0.12
TCPP (1500)	3	2.50	8.83	0.51 ± 0.05	3.46 ± 0.25
Positive control EMS	3	2.17	11.33	1.40 ± 0.05	6.77 ± 0.31
Vehicle (0)	24	2.17	5.50	0.41± 0.04	2.91 ± 0.20
TCPP (750)	24	1.42	6.67	0.41 ± 0.02	2.90 ± 0.14
TCPP (1500)	24	1.33	7.50	0.49 ± 0.05	3.29 ± 0.32

It was concluded that TCPP did not induce DNA damage in the liver or rats treated up to 1500 mg/kg.

An *in vitro*/*in vivo* UDS assay was carried out to GLP and in accordance with OECD Guideline No. 486 and EC Method B.39 (Bayer Healthcare 2005). A preliminary range finding study was performed by initially dosing three male rats by oral gavage at a dose of 2000 mg/kg. Subsequently two male and two female rats were treated at 1500 mg/kg and two females at 1000 mg/kg. One male dosed at 2000 mg/kg showed narrowed palpebral fissures one day post administration. The females dosed at 1500 mg/kg showed signs of pilo-erection, twitches and narrowed palpebral fissures on days one and two post administration. The males treated at 1500 mg/kg and the females treated at 1000 mg/kg were without findings.

In the main study, TCPP was administered by oral gavage to four female Wistar rats per dose group per sacrifice time at doses of 750 and 1500 mg/kg in corn oil. Two sacrifice time points were employed: 4 hours and 16 hours following dosing. Vehicle control animals and positive control (2-acetylaminofluorene and N,N'-dimethylhydrazine) animals were treated concurrently by oral gavage. During animal observations, pilo-erection, narrowed palpebral fissures, apathy and accelerated breathing were noted in animals at 1500 mg/kg prior to necropsy.

Hepatocytes were harvested following perfusion of the livers of each rat from each group at the two selected time points. Hepatocyte cultures were established and, following an attachment period of 90 minutes, parallel 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.

A statistically significant increase of mean net nuclear grain (NNG) count above the control count was noted for both doses and at both sacrifice times. At the 16-hour time point only, the increase was dose related. The NNG counts for TCPP were above the laboratory historical control threshold for both time points. However, the NNG counts for each dose did not exceed zero and therefore it is not possible to indicate that the result is a positive one. In interpreting the results of this assay, both statistical significance and biological significance should be taken into account. As the NNG counts did not exceed zero, the biological significance of the result is questionable. Results are usually considered to be biologically significant if the NNG count is greater than zero. The vehicle and positive controls yielded acceptable results.

Owing to the difficulty in interpreting the results of this study, Industry had it reviewed independently by a leading expert in this field. His conclusions (Kirkland, 2005) were that the findings in the study do not support a positive conclusion. He noted that all animals treated with TCPP had NNG counts less than zero and made the point that NNG values less than zero are not biologically significant. Additionally, he pointed out that the generally acceptable range for NNG counts in control animals is –8 to 0, and thus all NNG values in TCPP-treated animals fell in the control range. Overall, he concluded that although statistically significant increases in NNG counts were obtained, none of the findings were biologically significant. As a clear result was not obtained in the study, the results are considered to be equivocal.

TCPP was not clastogenic in a mouse micronucleus test (Bayer AG, 1991e) in a GLP study reported as adhering to OECD guidelines. Young adult male and female NMRI mice received a single intraperitoneal injection of 350 mg/kg test compound in peanut oil. This was based on the outcome of a preliminary toxicity study. In this pilot study groups of five animals including males and females were administered TCPP intraperitoneally at doses of 250, 300, 325, 350, 375 and 500 mg/kg. Apathy, staggering gait, lateral position, spasm, extension spasm, leaping spasm, twitching, shivering and difficulty in breathing were noted in animals up to 48 hours. 2/5 animals died in the 375 mg/kg dose group and 3/5 animals died in the 500 mg/kg group.

In the main study, the dose of 350 mg/kg was administered to four groups of 5 male and 5 female mice – a replacement group and a 16, 24 and 48-hour sacrifice group. The negative control was peanut oil and the positive control was cyclophosphamide. 1000 polychromatic erythrocytes (PCEs) were counted. Treated animals showed signs of toxicity for up to 16 hours after dosing including apathy, staggering gait, spasm, twitching shivering, difficulty breathing and salivation. After 16 hours, their appearance and activity appeared normal. Feeding behaviour throughout the study was normal. 2/40 animals died during the test period and this was related to the acute toxicity of TCPP. The ratio of PCEs to NCEs was not significantly altered by TCPP treatment compared to the negative control. The incidence of micronucleated PCEs or NCEs was not significantly increased in the treatment groups compared to the negative control. Cyclophosphamide did not induce a significant alteration in the ratio of PCEs to NCEs but did significantly increase the incidence of micronucleated PCEs.

There were a number of shortcomings in this study. According to the OECD guideline, three dose levels are used for the first sampling time if toxicity is noted in the preliminary study covering a range from the maximum to little or no toxicity. Only the highest dose needs to be tested at the later sampling time. However, in this study only a single dose was tested despite the toxicity noted in the preliminary study. The use of a single dose level is acceptable when toxicity is not observed. In addition only 1000 polychromatic erythrocytes per dose were scored for the incidence of micronucleated immature erythrocytes whereas the OECD and EC guidelines recommend that at least 2000 PCEs are evaluated per animal for the incidence of micronucleated immature erythrocytes.

TCPP did not induce an increase in chromosomal aberrations when investigated in a rat bone marrow cytogenetic assay (Stauffer Chemical Co., 1978e). Based on the results of a range-finding study, the doses tested were 0.011, 0.04 and 0.11 ml/kg, corresponding to 14.2, 51.6 and 142 mg/kg, respectively. DMSO was used as a solvent and negative control. The route of exposure was the oral route for TCPP and i.p. for the positive control. In the acute phase of the study, 8 adult male Sprague-Dawley rats were used in each dose group and at each of three sacrifice times, 6, 24 and 48 hours. In the sub-chronic phase of the study, 8 rats per dose group were dosed orally 5 times, 24 hours apart and were sacrificed 6 hours after the final

dose. Two hours prior to sacrificing, the animals were administered colchicine intraperitoneally. Fifty metaphase spreads were analysed from each animal. The positive control was triethylenemelamine. The % of cells with one or more aberrations in the positive control was 14.5% compared to 1.3 % in the negative control at the same kill time. Based on the frequency of total breaks per cell and the frequency of cells with ≥ 1 aberration, none of the test animal frequencies differed significantly from control frequencies ($p < 0.05$). Results were consistent with historical control data. There were no reports of toxicity observed during the study.

However, the confidence in this result is low due to a number of reasons. The choice of the top dose was based on preliminary study results which were not reported in the final test report and, therefore, it is not possible to establish whether the top dose tested in the main study is the maximum tolerated dose or the maximum feasible dose. According to the current OECD Guideline No. 475, at least 100 cells should be analysed per animal. Only 50 cells/animal were analysed in this study. In addition, no justification is provided in the test report for the sampling times used for the acute phase of the study, namely 6, 24 and 48 hours. According to the OECD guideline, the first sampling interval is 1.5 times the normal cell cycle length (the latter being normally 12-18 hours) following treatment.

4.1.2.7.3 Summary of mutagenicity

The results of all mutagenicity studies are summarised **Table 4.46** below.

Table 4.46 Summary of mutagenicity tests for TCPP

Study	Endpoint	Result	Comments	Reference
In vitro plate incorporation assay, bacteria	Gene mutation	Negative		Zeiger *et al*., 1992
In vitro plate incorporation assay, bacteria	Gene mutation	Negative		Stauffer Chemical Co., 1978a
In vitro plate incorporation assay, bacteria	Gene mutation	Negative		Mobil Environmental and Health Safety Laboratory, 1980e
In vitro plate incorporation assay, bacteria	Gene mutation	Negative		Stauffer Chemical Co., 1976
In vitro plate incorporation assay, bacteria	Gene mutation	Negative		SafePharm Laboratories Ltd., 1992
In vitro plate incorporation assay, bacteria	Gene mutation	Negative		Nakamura *et al*., 1979
In vitro plate incorporation assay, bacteria	Gene mutation	Positive without metabolic activation	No dose-response	Tenneco Chemicals Inc., 1977a
In vitro plate incorporation assay, bacteria	Gene mutation	Positive in the std. assay	Response only at ≤3% survival in quantitative assay	Majeska & Matheson, 1983
In vitro plate incorporation assay, bacteria	Gene mutation	Negative		Föllmann & Wober, 2006

Study	Endpoint	Result	Comments	Reference
In vitro E. coli repair test, bacteria	Gene mutation	Negative		Tenneco Chemicals Inc., 1977b
In vitro plate incorporation assay, fungi	Gene mutation	Negative		Stauffer Chemical Co., 1978a
In vitro plate incorporation assay, fungi	Gene mutation	Negative		Stauffer Chemical Co., 1976
In vitro mouse lymphoma assay	Gene mutation	Negative		Stauffer Chemical Co., 1978b
In vitro mouse lymphoma assay	Gene mutation	Positive, presence of metabolic activation	Result considered equivocal	Environmental Affairs & Toxicology Department, 1981c
In vitro mouse lymphoma assay	Gene Mutation	Positive, presence of metabolic activation	Increase in small colony mutants indicating possible clastogenic activity.	Covance Laboratories Ltd., 2005
In vitro UDS assay	DNA repair	Negative		Bayer AG, 1991d
In vitro UDS assay	DNA repair	Negative		Williams *et al.,* 1989
In vitro UDS assay	DNA repair	Equivocal	Result considered equivocal	Stauffer Chemical Co., 1978c
In vitro transformed foci in BALB/3T3 cells	Cell transformation	Positive	No dose-response	Stauffer Chemical Co., 1978d
In vitro transformed foci in BALB/3T3 cells	Cell transformation	Negative		Stauffer Chemical Co., 1980b
In vitro Comet assay in V79 cells	DNA damage	Negative		Föllmann & Wober, 2006
In vivo Comet assay in rat liver	DNA damage	Negative		Covance Laboratories Ltd., 2006
In vivo UDS assay	DNA damage & repair	Could be considered equivocal	Increase in NNG counts statistically significant however did not exceed 0.	Bayer Healthcare 2005
In vivo mouse micronucleus assay	Chromosome aberration	Negative	Not in full compliance with current guidelines	Bayer AG, 1991e
In vivo rat bone marrow cytogenetic assay	Chromosome aberration	Negative	Not in full compliance with current guidelines	Stauffer Chemical Co., 1978e

The mutagenic potential of TCPP has been well investigated *in vitro*. Evidence from several bacterial mutagenicity studies shows that TCPP is not a bacterial cell mutagen. TCPP was also shown to be non-mutagenic in fungi. In mammalian cell studies, TCPP did not induce forward mutations at the TK locus in L5178Y mouse lymphoma cells in one study, but in a second study, the result was considered equivocal (in the presence of rat liver S9 fraction). A confirmatory mouse lymphoma was conducted in accordance with the relevant regulatory guidelines. The results of the assay indicate that TCPP shows clastogenic activity *in vitro* in the presence of metabolic activation.

In one GLP study, TCPP did not induce unscheduled DNA synthesis *in vitro*. Two other *in vitro* UDS studies are reported. In one, TCPP gave a negative result; in the second, the result is considered equivocal. In an *in vitro* transformation assay, TCPP was seen to induce transformed foci in BALB/3T3 cells, whereas in another similar study, it did not.

As indicated above, the results of the most recent *in vitro* mouse lymphoma assay were positive. In particular in this study, there was a clear increase in the proportion of small colony mutants, giving rise to concern for a possible clastogenic effect of TCPP. Due to this positive study, industry proceeded to carry out the above-mentioned *in vitro/in vivo* UDS assay to further investigate the mutagenic potential of TCPP *in vivo*. In this study, statistically significant increases in NNG and a dose response effect at one time point were observed. However, as the counts did not exceed zero at either of the doses tested, the biological significance of the effect is doubtful and thus the result is considered equivocal.

The main concern for TCPP is clastogenicity, owing to the clearly positive *in vitro* mouse lymphoma study. The UDS assay is not considered to be the most appropriate test for investigating a potential clastogen, as clastogenic substances are not expected to be efficient at inducing unscheduled DNA synthesis.

In vivo, TCPP was not clastogenic in a mouse bone marrow micronucleus test. TCPP did not induce an increase in chromosomal aberrations in a rat bone marrow cytogenetics assay. However, there were some shortcomings in these studies and it is considered that they do not fulfil all current regulatory guidelines as described in the study summaries in 4.1.2.7.2.

Therefore, in order to investigate the potential for TCPP to induce DNA damage, an *in vivo* Comet assay in the rat liver was conducted. The liver was chosen for comet analysis as TCPP caused an increased mutation frequency in the mouse lymphoma assay in the presence of S9 and also induced liver enlargement in repeat dose studies. Under the conditions of this study, TCPP did not induce DNA damage in the liver of rats treated with either 750 or 1500 mg/kg TCPP.

Overall, it is considered that TCPP is not genotoxic *in vivo*.

4.1.2.8 Carcinogenicity

4.1.2.8.1 Studies in animals

There are no carcinogenicity data for TCPP.

As described in section 4.1.2.6 of this report, the study of longest duration for TCPP is a 90-day dietary study in rats. Increased liver weights were observed in males at 52 mg/kg and above and periportal hepatocyte swelling was noted at the highest dose (1349 mg/kg in males and 1745 mg/kg in females). In addition, mild thyroid follicular cell hyperplasia was noted in females at 1745 mg/kg and in all dosed males. In the kidney, vacuolation in females at the highest dose was also observed. A slightly excessive fatty infiltration indicative of mild bone marrow hypoplasia was noted in three high dose females. The selected LOAEL of 52 mg/kg/day is based on increased liver weights observed in males. In the absence of carcinogenicity data, it cannot be excluded that the effects observed in the 90-day study may progress to cancer. Therefore, as a reasonable worst case approach, this data will be used in a quantitative way to carry out a risk characterisation for carcinogenicity.

This initial concern for carcinogenicity is further supported by the fact that TCPP is structurally similar to two other chlorinated alkyl phosphate esters, TDCP and TCEP. TDCP and TCEP are considered to be non-genotoxic carcinogens and have agreed classifications of Carc. Cat 3 R40[20]. The acceptability of a read-across from TCEP and TDCP to address the potential carcinogenicity of TCPP is presented in Appendix D to this report. As described in that Appendix, it is considered that there is sufficient information from the structures, physical-chemical properties, toxicokinetics and mutagenic profiles of TCEP, TDCP and TCPP to support a qualitative read-across for carcinogenicity. However, based on the available data, there are some differences in the metabolism, target organs, the severity of the effects observed and the potency of the three substances, which indicate that a quantative read-across for carcinogenicity from either TDCP or TCEP to TCPP may not be appropriate, including a quantitative read across for the purpose of classification and labelling. There are no insights in the available data on TDCP and TCEP regarding an underlying mode of action for these substances which would make a prediction on a relative potency of TCPP possible.

The qualitative read across approach is used for hazard and risk assessment only. Overall, this approach is preferred as it enables a risk characterisation to be carried out and thus the situation in which a data gap would trigger the need for a cancer bioassay is avoided

4.1.2.8.2 Studies in humans

No studies are available.

4.1.2.8.3 Summary of carcinogenicity

As discussed in section 4.1.2.7, TCPP, like TDCP and TCEP is not genotoxic *in vivo*. Based on the available repeat dose toxicity data for TCPP, supported by a qualitative read-across from TDCP and TCEP, there is a potential concern for carcinogenicity for TCPP by a non-genotoxic mechanism. No quantitative read-across can be performed since there are no insights into an underlying mode of action for TCEP and TDCP which would make a prediction on a relatively potency of TCPP possible. Therefore, as a reasonable worst case approach, a risk characterisation will be carried out for this end-point.

It is proposed that the effects observed in the 90-day study for TCPP are taken as a starting point for risk characterisation. If these effects were to progress to cancer, they would do so by a non-genotoxic mechanism. Therefore, it is proposed that the LOAEL of 52 mg/kg/day, identified from the 90-day study with TCPP, should be used as a basis for risk characterisation of the carcinogenicity endpoint.

[20] 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

4.1.2.9 Toxicity for reproduction

4.1.2.9.1 Effects on fertility

Studies in animals

An oral two-generation reproduction toxicity study in rats was carried out in accordance with OECD Guideline No. 416 and to GLP (TNO Quality of Life, 2007). The main study was preceded by a preliminary range finding study (one-generation reproductive toxicity study), in which 10 animals/sex/dose were administered TCPP in the diet at 0, 1500, 5000 and 15000 mg/kg diet (corresponding to approximately: 0, 95, 325 and 1000 mg/kg bw/day for males and for females 0, 108, 370 and 1176 mg/kg bw/day during premating, 0, 100, 314 and 963 mg/kg bw/day during gestation and 0, 193, 680 and 1930 mg/kg bw/day during lactation). Males and females were treated for 5 weeks prior to mating and during mating, and then during gestation and lactation to post-natal day (PN) 21 for females. Dams were allowed to raise one litter. On PN4 litter sizes were adjusted to 4 males and 4 females per litter, where possible. Animals were observed for clinical signs and food consumption, and body weight gain was measured. Fertility and reproductive performance were recorded. Dams were sacrificed for necropsy at PN21. Males were euthanized after at least 42 days of exposure for sperm analysis and necropsy.

One female (C47) of the mid dose group, who did not eat after gestation day (GD) 14 and showed piloerection on GD21, was killed in moribund on GD21. At necropsy the remnants of 1 pup were found in the stomach, 1 pup in the vagina and 9 dead pups in the uterus. There were no other clinical signs in the treated animals. Parental mean body weights were statistically significantly decreased in males of high dose group during premating and mating, in the mid dose females during premating and in the mid and high dose females during gestation and lactation. In females, mean food consumption, expressed as g/kg body weight/day, was statistically significantly increased in high dose group during premating, decreased in high dose group during gestation and increased at the high dose during days 1-4 and then decreased for the remainder of the lactation period.

All TCPP treated females were found sperm positive within 4 days and all mated females were found to be pregnant. There was no difference in pre-coital time, mating index, and male and female fertility index between the control and TCPP treated groups. The post-implantation loss was higher (not statistically significant) in the low and mid-dose groups: 4.43%, 11.19%, 18.05% and 8.41% for control, low, mid and high dose groups, respectively.

The number of pups delivered and sex ratio was comparable in all groups. Pup mortality was statistically significantly increased in the high dose group; all 8 pups of one dam (D71) died or were missing on PN5. Litter and pup data are presented in section 4.1.2.9.2.

In parental males, there was no treatment related effect on motility or count of epididymal sperm, or on sperm morphology. Parental terminal body weights were statistically significantly decreased in the high dose male and females. In males, there was a statistically significant decrease absolute prostate weight of the low and high dose group, with a non-significant decrease in mid dose animals. Relative liver weights of mid and high dose males and high dose females were statistically significantly increased. In females, the absolute adrenal weight was statistically significantly decreased in the high dose group. There was a statistically significant increase in relative brain weight in the high dose group, which was most probably related to the decreased terminal body weight. Absolute and relative uterus

weights were statistically significantly decreased in low, mid and high dose groups when compared to the control group. The mean relative and absolute uterus weight in the control group was relatively high due to elevated uterus weights in three of the control animals. **Table 4.47** below summarises the significant body weight and organ weight changes.

Table 4.47 Mean terminal body weights and organ weights for males and females

		Dose (mg TCPP/kg diet)			
Organ	**Sex**	**0**	**1500**	**5000**	**15000**
Mean terminal body weight	M	388.5	370.3	381.7	359.1**
	F	274.1	270.0	260.6	246.2***
Mean absolute organ weight (g)					
Liver	M	14.276	13.293	15.002	15.411
	F	14.283	14.065	14.623	14.575
Brain	M	1.900	1.880	1.897	1.865
	F	1.768	1.755	1.753	1.701
Adrenal	M	0.055	0.053	0.055	0.054
	F	0.075	0.071	0.074	0.067*
Uterus	M	1.212	1.030*	1.092	0.982**
Prostate	F	0.548	0.303*	0.280**	0.286**
Mean organ weights relative to terminal body weight (g/kg bw)					
Liver	M	36.733	35.891	39.291**	42.906***
	F	52.154	52.078	56.088	59.037*
Brain	M	4.898	5.090	4.975	5.207
	F	6.459	6.503	6.736	6.924**
Adrenal	M	0.143	0.143	0.144	0.152
	F	0.274	0.264	0.284	0.272
Uterus	F	2.004	1.121*	1.070*	1.171*
Prostate	M	3.131	2.795	2.858	2.735

*/**/*** statistically significantly different to the control group p< 0.05/ 0.01/ 0.001

No treatment related gross or histopathological changes were observed in any of the treated animals. The female of the mid dose group killed in moribund on GD21 did not reveal any treatment related histopathological changes.

Based on the results of the preliminary study, 28 Wistar rats (Crl:WI(WU)/sex/group received TCPP in the diet over two successive generations. In each dose group, the concentration of the test substance was adjusted over the course of the study to maintain target concentrations of 0, 100, 333 and 1000 mg TCPP/kg bw/day The animals were fed diets containing the test substance from the start of the study, during the premating period of at least 10 weeks, throughout gestation and lactation until sacrifice. Vaginal smears were made three weeks prior to mating to evaluate the length and normality of the oestrus cycle and daily during the mating period to determine if sperm was present. Upon evidence of copulation, the females were caged individually for the birth and rearing of pups until PN21 or shortly thereafter when they were sacrificed. Dams were allowed to raise one litter per generation. On PN4, litters of more than 8 pups were adjusted to 4 males and 4 females per litter, where possible.

On PN21, the litters were weaned and 28 males and 28 females were selected at random from as many litters as possible in each group to rear the next generation. Animals were observed for clinical signs, and food consumption and body weight gain was recorded. Fertility and reproductive performance were measured. F0 and F1 dams were sacrificed at or shortly after weaning. F0 and F1 males were sacrificed after at least 11 weeks of exposure. At scheduled necropsy, epididymal sperm was assessed for motility, count and morphology and a testicular sperm count was also made.

The overall intake of TCPP was 0, 85, 293 and 925 mg TCPP/kg bw/day for males and 0, 99, 330 and 988 mg TCPP/kg bw/day for females, for the control, low, mid and high dose groups, respectively.

There were no treatment related clinical signs in parental animals in either generation. During premating, an F1 male of the mid dose group was found dead on Day 41 and female of the same group was killed in moribund on Day 50. The cause of death or cause of the moribund condition could not be detected at necropsy. There were no other mortalities. A treatment related decrease in body weights was observed in F0 and F1 males of mid and high dose groups, with a larger decrease observed in the F1 generation. During premating, there was no effect on body weight in females of F0 generation but body weights of females in F1 generations were decreased in the mid and high dose groups. During gestation, the mean body weights were decreased in high dose females in F0 females and in mid and high dose F1 females. Body weights were decreased in mid and high dose F1 females during lactation. Mean food consumption was decreased in F0 and F1 males and females of the high dose group and in F0 males and females and F1 females of the mid dose group.

The mean length of the longest oestrus cycle was statistically significantly increased in all dosed F0 females and in high dose F1 females. The number of cycles per animal was significantly decreased in the high dose groups of both the F0 and F1 generations, and the number of acyclic animals was increased in high dose F0 animals only.

This effect on the oestrus cycle appears only to be toxicologically significant at the highest dose as the effect on cycle length was only consistently seen in both the F0 and F1 generations at the highest dose, and is only outside the historical control range at this top dose and the number of acyclic animals and mean number of cycles was only affected in the high dose group. **Table 4.48** below summarises the oestrus cycle data.

Table 4.48 Effect of TCPP on oestrus cyclicity

		Dose Group				
Effect	**Generation**	**0**	**Low**	**Mid**	**High**	**Historical control range$**
No. of acyclic females	F0	1	0	0	6**	-
	F1	1	0	1	3	-
Length of longest oestrus cycle (days):						
4	F0	18	11	6	1	-
	F1	11	10	11	2	-
5	F0	7	13	16	13	-
	F1	12	12	7	10	-
6	F0	2	3	5	3	-
	F1	4	5	5	8	-
≥7	F0	0	1	1	5	-
	F1	0	1	3	5	-
Mean	F0	4.4	4.8*	5.1**	5.6***	4.1 – 5.2 (n=15)
	F1	4.7	4.9	5.0	5.8***	
Mean no. cycles per animal	F0	3.9	3.7	3.6	3.0*	-
	F1	3.8	3.6	3.7	3.1*	-

*/**/*** statistically significantly different to the control group p< 0.05/ 0.01/ 0.001. $ Historical control data taken from one-and two-generation oral reproductive toxicity studies and 90-day studies in Wistar rats conducted at TNO between 2003 and 2007

All females, except one of the high dose group in F0, were found sperm positive. One female in low dose group in F0 and one of high dose group in F1 showed only implantations. In both generations, no treatment related differences were observed in pre-coital time, mating index, female fecundity index, male and female fertility index, duration of gestation and post-implantation loss. All dams survived the delivery and there were no dams with stillborn pups in any of the groups.

The mean number of pups delivered was decreased in the mid-dose group of the F1-generation and in the high dose groups in both generations. This resulted for both high dose groups, in a lower mean number of live pups on PN1 and 4. The effect seen in the high dose group of the F1 generation was mainly due to one litter (10 pups) of dam D597 which was lost entirely on PN4. The study report states that due to a deviation from the study plan, the corpora lutea were not counted at scheduled sacrifice. It is not clear whether the effect on the number of pups per litter on PN1 is due to decreased fertility of the parental animals or a developmental effect on the pups. Additionally, it is noted that the effect on the mean number of pups delivered corrolates with a decrease in maternal body weight observed during the gestation period in these dose groups and therefore may be possibly due to maternal toxicity.

Overall, the effect on the number of pups delivered is observed mainly in the F1 generation, at both the mid and high doses, although the interpretation of the effect at the high dose is hampered by the fact that 10 pups of one single litter died at the high dose. The numbers of pups delivered at the mid and high doses in the F1 generation are outside the historical control ranges. It is noted that there is an increase in post implantation loss in the F1 generation (although this does not reach statistical significance), which could point more towards the

observed effect on the number of pups on PN1 being a developmental rather than a fertility effect. Litter data is presented in full in section 4.1.2.9.2

Pup mortality (PN1-4) was statistically significantly increased in the low dose group of F0 and in the high dose groups of F0 and F1. This effect was only observed when the pup was used as the statistical unit. There was no statistically significant difference in the mean number of pups on PN4.

No treatment related effect on epididymal sperm motility or sperm count, sperm morphology or mean testicular sperm count was observed in either generation at necropsy. Terminal body weights were decreased in mid and high dose males of both generations and in females of the F1 generation.

In males, absolute brain weight was decreased in high dose F0 and mid and high dose F1 animals, and relative brain weight was increased in high dose F0 and F1 animals. Relative adrenal weight was increased in high F1 males. Absolute kidney weights were decreased in high dose F0 males and in all dosed F1 males, with relative weights increased in high dose F1 males. Relative liver weights were increased in all dosed F0 males and mid and high dose F1 males. Absolute spleen weight was decreased in high dose F0 males and mid and high dose F1 males. Relative thyroid weights were increased in high dose F0 & F1 males. Decrease in absolute pituitary weight in high F1 males. There was a decrease in absolute epididymal weight in high dose F0 males and an increased in relative weight in high dose F1 males. Absolute seminal vesicle weights were decreased in mid and high dose F0 and F1 animals. Absolute testes weights were decreased in high dose F0 males. Relative testes weight increased in mid and high dosed F1 males. Decrease in absolute prostate weight in high F1 males.

Overall, with respect to effects on organ weights in males, the effect on the kidney at the highest dose group is considered to be the main effect.

In females, absolute and relative liver weights were increased in high dose F0 females and relative liver weight increased in high F1 females. Absolute and relative pituitary weight was decreased in high dose F0 females, in low and high dose F1 females; absolute weight was decreased in mid dose F1 animals. Absolute ovary weight was decreased in high dose F0 females. Absolute and relative spleen weight was decreased in mid and high dose F0 females and high dose F1 females. Absolute brain weight was decreased and relative brain weight increased in high dose F1 females. Absolute kidney weight was decreased in high F1 females.

Absolute and relative uterus weights were decreased in all dosed F0 females and high dose F1 females.

Overall, as regards effect on organ weights in females, there are clear effects on the spleen and the pituitary at the highest dose. The most significant observed in females was a decrease in uterus weight, which was noted at all dose levels of F0 and in the high dose group of F1: 82%, 68% and 68% of the control values for low, mid and high dose groups of F0 generation and 81%, 80% and 65% of the control for the low, mid and high dose groups of F1 generation, respectively. The decrease at the low and mid doses of F1 did not reach statistical significance. It is noted that a decrease in uterus weight was also observed in all dose groups in the preliminary study.

It is noted that the decrease in uterus weights, while significant was not accompanied by any histopathological changes. The oestrus cycle stage was not recorded at necropsy. It is accepted that uterine weight can fluctuate during the oestrus cycle and therefore, there is a

possibility that the effects observed may be due to normal variation in uterus weight in cycling females. However, as a reasonable precautionary approach it cannot be excluded that the effects on uterus weight are treatment related. **Tables 4.49** and **4.50** below summarises the significant organ weight effects.

Table 4.49 Mean terminal body weights and significant organ weights for males of F0 and F1 generations

		Dose Group			
Organ	**Generation**	**0**	**Low**	**Mid**	**High**
Mean terminal body weight	F0	416.5	400	394.9*	374.1#
	F1	397.8	390.8	367.3**	336.1#
Mean absolute organ weight (g)					
Kidney	F0	2.406	2.333	2.326	2.252**
	F1	2.313	2.200*	2.113#	2.061#
Spleen	F0	0.742	0.730	0.703	0.629#
	F1	0.751	0.736	0.672#	0.596#
Pituitary	F0	0.014	0.014	0.013	0.013
	F1	0.015	0.015	0.014	0.013#
Seminal vesicles	F0	1.595	1.518	1.419*	1.388*
	F1	1.475	1.392	1.211#	1.191#
Mean organ weights relative to terminal body weight (g/kg bw)					
Kidney	F0	5.788	5.850	5.901	6.026
	F1	5.843	5.645	5.761	6.164*
Spleen	F0	1.781	1.823	1.782	1.683
	F1	1.894	1.886	1.834	1.784
Pituitary	F0	0.033	0.035	0.032	0.036
	F1	0.039	0.038	0.038	0.038
Seminal vesicles	F0	3.841	3.808	3.591	3.723
	F1	3.712	3.585	3.310	3.511

*/**/# statistically significantly different to the control group p< 0.05/ 0.01/ 0.001

Table 4.50 Mean terminal body weights and significant organ weights for females of F0 and F1 generations

		Dose Group			
Organ	**Generation**	**0**	**Low**	**Mid**	**High**
Mean terminal body weight	F0	267	268	263	258
	F1	264	265	251*	246**
Mean absolute organ weight (g)					
Liver	F0	13.608	13.580	13.702	14.890**
	F1	13.629	13.673	13.389	13.872
Spleen	F0	0.508	0.490	0.466**	0.443***
	F1	0.507	0.505	0.483	0.438***
Pituitary	F0	0.016	0.016	0.016	0.015***
	F1	0.017	0.015**	0.016*	0.014***
Uterus	F0	0.46	0.375*	0.313***	0.311***
	F1	0.455	0.369	0.367	0.295***
Ovary	F0	0.082	0.081	0.077	0.073**
	F1	0.084	0.080	0.083	0.076
Mean organ weights relative to terminal body weight (g/kg bw)					
Liver	F0	50.918	50.791	52.031	57.611***
	F1	51.590	51.601	53.394	56.202**
Spleen	F0	1.9	1.833	1.770**	1.711***
	F1	1.922	1.908	1.928	1.779*
Pituitary	F0	0.062	0.060	0.061	0.057*
	F1	0.065	0.057**	0.062	0.059*
Uterus	F0	1.723	1.408*	1.192***	1.202***
	F1	1.732	1.399	1.465	1.202**
Ovary	F0	0.309	0.304	0.293	0.285
	F1	0.317	0.302	0.331	0.307

*/**/*** statistically significantly different to the control group p< 0.05/ 0.01/ 0.001

There were no treatment related macro-or microscopical changes were observed in the F0 or F1 parental animals. The incidence of mineralisation in the kidneys of the high dose F1-females was higher than in the controls (5/28 in control versus 11/28 in the high dose group). However, kidney mineralisation is a common finding in female rats and therefore not thought to be treatment related. Only the relative liver weight was increased in low dose males and was not accompanied by any increase in absolute organ weight or clinical chemical effects. Therefore, this can be considered an adaptive effect and therefore not adverse.

In deriving a N(L)OAEL for effects on fertility, consideration is given to the significant effects observed uterus weight in all dosed females in F0 generation and in high dose animals of F1 generation.

With respect to the decrease in the number of oestrus cycles, this was significant only in the high dose animals and so the effect on the cycle length observed at low and mid doses may be due to normal variation rather than a specific fertility effect.

The study director concluded in the study report that the effects observed on uterus weights in the low and mid dose females of the F0 generation were not adverse since they were not accompanied by any change in the number of oestrus cycles or histopathological findings in the uterus, and that there was no corresponding decrease in uterus weight in the low or mid dose F1 animals.

While the effects on the uterus weight and oestrus cycle may be due to normal variation or weight loss, overall, based on a weight of evidence approach, it cannot be excluded that TCPP has an effect on uterus weight. This effect on the uterus was also observed in all dosed females in the preliminary study. Although the effects on the uterus occurred in the absence of histopathological changes, the magnitude of the decrease in uterus weight in the dosed animals is sufficient to be considered as significant. In addition, the mean number of cycles per animal are decreased and the length of the longest oestrus cycle are statistically increased in high dose animals of both generations, indicating a possible treatment related effect on the oestrus cycle. Therefore, a LOAEL of 99 mg/kg, based on effects on uterus weight, is derived for effects on fertility and this figure is taken forward to risk characterisation for this endpoint.

The low-dose of approximately 99 mg/kg for females is considered to be the LOAEL for parental toxicity. This is based on decreased body weight and food consumption seen in mid and high dose parental animals and the effects on uterus weights seen in all dosed F0 animals. For males, a NOAEL of approximately 85 mg/kg is derived for parental toxicity, based on decreased body weights, food consumption and organ weight changes observed at mid and high dose groups.

In the 90-day study, in which 20 male and 20 female Sprague Dawley rats were fed diets containing 0, 800, 2,500, 7,500 and 20,000 ppm of TCPP, there were no effects observed in the testes or ovaries of treated animals when examined at necropsy. On histopathological examination, one male in the lowest treatment group had a reddened/swollen prostate. In the 7,500 ppm male group, one animal displayed a red focus and also a cleft-like cyst in the testis and in the highest treatment female group, one female showed a nodule on an ovary. These were isolated incidences. Mucometra was observed in treated females, but not in controls (2/20, 3/20, 4/19 and 1/20 in the 800, 2500, 7500 and 20000 ppm treatment groups, respectively). The female mammary glands were not examined.

On detailed microscopic examination in males, the testis, epididymis and seminal vesicles of all 20 animals in the high dose group were examined and all were unremarkable. 3/20 control males showed prostatitis, while 5/20 of the highest treatment group demonstrated it. In females, detailed microscopic examination of the ovaries of the 20 high-dose animals showed that 16 of them were unremarkable. Of the remaining 4, 3 had luteal cysts and 1 had follicular dystrophy. The follicular dystrophy was also observed in one of the control animals.

In the 28 day study (Bayer, 1991c), histopathology results indicated that one male animal treated with the highest dose (1000 mg/kg) out of the 6 treated animals had germinal epithelial acute degeneration in the testis. There was also abnormal sperm formation in the epididymis of this animal. The control animals did not demonstrate this. The male animals treated with the other two doses were not examined. There were no changes observed in the ovaries of treated female animals.

Studies in humans

No studies are available.

4.1.2.9.2 Developmental toxicity

Studies in animals

Developmental toxicity of TCPP to rats was investigated as part of the two-generation reproduction toxicity study described in section 4.1.2.9.1 above (TNO Quality of Life, 2007). In the preliminary range finding study (one-generation reproductive toxicity study), 10 animals/sex/dose group were administered TCPP in the diet at 0, 1500, 5000 and 15000 mg/kg diet. Males and females were treated for 5 weeks prior to mating and during mating, and then during gestation and lactation to post-natal day (PN) 21 for females. Dams were allowed to raise one litter. On PN4 litter sizes were adjusted to 4 males and 4 females per litter, where possible. At birth, litter size, and sex and weight of pups was reported. At weaning (PN21) all pups were thoroughly examined, abnormalities noted and thereafter sacrificed by CO_2/O_2.

Maternal body weights were statistically significantly decreased in the mid dose group during premating and in the mid and high dose groups during gestation and lactation. Mean food consumption, expressed as g/kg body weight/day, was statistically significantly increased in high dose group during premating, decreased in high dose group during gestation and increased at the high dose during days 1-4 and then decreased for the remainder of the lactation period.Mean pup weights were statistically significantly decreased in high dose group on PN14 and 21 and the mid dose on PN21. Pup mortality was statistically significantly increased in the high dose group; all 8 pups of one dam (D71) died or were missing on PN5. **Table 4.51** below summarises the pup and litter data.

Table 4.51 Pup and Litter data from the preliminary study

Effect	Dose (mg TCPP/kg diet)			
	0	**1500**	**5000**	**15000**
Total no. of pups delivered	98	96	92	98
Live birth index (%)	100	100	100	100
No. of pups lost (dying, missing and/ or cannibalized) on:				
Days 1-4	0	0	2	0
Days 5-7	0	0	0	[1]8**
Days 8-14	0	0	0	1
Days 15-21	0	0	0	0
No. pups alive Day 21	72	77	72	68**
Sex ratio on PN1 (M/F)	52/46	57/39	50/42	51/47
Mean no. of live pups per litter on PN1	10.89	9.60	10.22	9.80
Post implantation loss (%)	4.43	11.19	18.05	8.41

[1] All 8 pups of one dam (D71)

**Statistically significantly different to the control group ($p < 0.01$)

On PN4, the number of cold pups and pups with no milk in the stomach was statistically significantly increased in the high dose group (attributed to the litter of dam D71). The number of runts was statistically significantly increased in the high dose group from PN4 to PN21 and in the low and mid dose group on PN21. **Table 4.52** below summarises pup clinical observations.

Table 4.52 Clinical observations in pups on Days 1-21 of lactation

Dose (mg TCPP/kg diet)	0	1500	5000	15000
Runts				
Day 1	0	4(1)	3(2)	0
Day 4	0	3(1)	1	20***(2)
Day 7	2(2)	4(1)	3(3)	26***(6)
Day 14	2(2)	3(2)	9(5)	50***(7)
Day 21	1	18***(4)	52***(8)	68***(9) ***
Cold pups (Day 4)	0	0	0	8**(1)
No milk in stomach (Day 4)	0	0	0	8**(1)

/ * statistically significantly different to the control group p< 0.01/ 0.001

Figures in brackets represent the number of litters with pups showing the observation

Pups that were found dead showed no abnormalities.

Based on the results of the preliminary study, 28 Wistar rats/sex/group received TCPP in the diets at maximum dose levels of 0, 100, 333 and 1000 mg TCPP/kg bw/day over two successive generations. The animals were fed diets containing the test substance from the start of the study, during the premating period of at least 10 weeks, during gestation and lactation until sacrifice. Dams were allowed to raise one litter per generation. Pup body weights, clinical signs and malformations were recorded on days 1, 4, 7, 14 and 21 of lactation. On PN4, litter sizes were adjusted to 4 males and 4 females per litter, where possible. On PN21, the litters were weaned and 28 males and 28 females were selected at random from as many litters as possible in each group to rear the next generation. After selection of pups for next generation, 1 male and 1 female F1 pup of each litter were subjected to a thorough necropsy. After necropsy, the thoracic part of the skeletons was stained and the ribs and sternum of these pups were examined for skeletal abnormalities. For F2 pups, the anogenital distance was measured in all pups on PN1. 1 male and 1 female F2 pup per litter was selected for assessment of vaginal opening and preputial separation.

The overall intake of TCPP was 0, 85, 293 and 925 mg TCPP/kg bw/day for males and 0, 99, 330 and 988 mg TCPP/kg bw/day for females, for the control, low, mid and high dose groups, respectively.

Maternal body weights were decreased in mid and high dose animals in F1 generation during premating, in high dose F0 and F1 animals and mid dose F1 animals during gestation and in mid and high dose F1 animals during lactation. Mean food consumption was decreased in F0 and F1 females of mid and high dose groups.

The mean number of pups delivered and the mean number of live pups per litter were decreased in the mid dose group of the F1 generation and in the high dose groups of both generations. These effects corrolate with a decrease in maternal body weight observed during

gestation period in these dose groups and therefore could possibly be due to maternal toxicity. Pup mortality (PN1-4) was statistically significantly increased in the low and high dose groups of F0 and in the high dose group of the F1 generation. This effect was only observed when the pup was used as the statistical unit. The effect observed in the F1 generation was mainly due to the loss of one litter (10 pups) of a single dam on PN4. There was no statistically significant difference in the mean number of pups on PN4. Thereafter (up to PN21), all pups of all groups remained alive. **Table 4.53** summarises the delivery, pup and litter data.

Table 4.53 Delivery, pup and litter data for F0 and F1 generations

	Dose Group				
Effect	**0**	**Low**	**Mid**	**High**	**Historical control range[$]**
F0:					
Mean no. of pups delivered	10.27	10.67	9.89	9.44*	9.40 – 11.18 (n=19)
Total no. of pups delivered	267	256	277	236	
Live birth index (%)	100	100	99	100	
No. of pups lost (dying, missing and/ or cannibalized) on:					
Days 1-4	3	20***	10	14**	
Days 5-7	0	0	0	0	
Days 8-14	0	0	0	0	
Days 15-21	0	0	0	0	
Mean no. live pups/litter (PN1)	10.27	10.63	9.79	9.44*	
Sex ratio on PN1 (M/F)	156/111	129/127	143/134	112*/124	
No. pups alive Day 21	198	178	213	190	
F1:					
Mean no. of pups delivered	10.56	10.00	9.13*	8.68***	9.40 – 11.18 (n=19)
Total no. of pups delivered	264	240	219	191	
Live birth index (%)	100	99	100	100	
No. of pups lost (dying, missing and/ or cannibalized) on:					
Days 1-4	1	0	2	12***	
Days 5-7	0	0	0	0	
Days 8-14	0	0	0	0	
Days 15-21	0	0	0	0	
Mean no. live pups/litter (PN1)	10.52	9.92	9.08**	8.68**	
Sex ratio on PN1 (M/F)	140/124	123/117	113106	94/97	
No. pups alive Day 21	198	186	181	155	

*/**/*** statistically significantly different to the control group p< 0.05/ 0.01/ 0.001

In the F0 generation, the mean number of runts was statistically significantly increased in all dose groups on PN1 and persisted to PN21 in the mid and high dose groups. In F1 generation, the number of runts was increased in the high dose group on PN14 and in all dose groups on PN21. In both generations, the number of runts in the high dose groups increased during the course of the lactation period. **Table 4.54** below summarises the number of runts in F0 and F1 generations.

Table 4.54 Clinical observations in pups of F0 and F1 generations on Days 1-21 of lactation

Dose Group	0	Low	Mid	High
F0				
Runts				
Day 1	0	14***(7)**	23***(7)**	11***(3)
Day 4	2(2)	11**(3)	7(5)	6(2)
Day 7	2(2)	13**(3)	20***(7)	21***(6)
Day 14	1	6(2)	15***(7)	26***(9)**
Day 21	1	4(2)	30***(10)**	97***(19)***
F1				
Runts				
Day 1	10(4)	1	17(5)	14(4)
Day 4	4(3)	0	15(3)	16(3)
Day 7	4(3)	2(2)	17(4)	38(8)
Day 14	11(6)	14(3)	19(5)	78***(13)*
Day 21	5(3)	17** (4)	36***(9)	127***(19)***

*/**/*** statistically significantly different to the control group p< 0.05/ 0.01/ 0.001

Figures in brackets represent the number of litters with pups showing the observation

The increased numbers of runts in all dose groups of the F0 generation on PN1 could indicate systemic toxicity to the pups *in utero*, although it is noted that no similar significant increase in the number of runts was observed in the F1 generation or in the preliminary study at PN1.

One pup of the mid dose group showed a missing eye, which was noticed on PN21.

There was no effect on pup weight at PN1 in either generation. There was no effect on pup weight on PN1 in both generations. Mean pup weights of the high dose group were significantly decreased in F0 generation from PN14 onwards and in the F1 generation from PN 7 onwards. Mean pup weights were decreased in mid dose groups on PN21.

No difference in anogenital distance of the male or female F2 pups was observed between the treated and control animals. Vaginal opening was delayed (not significantly) in the high dose group. Preputial separation was statistically significantly delayed in the high dose group. The mean age of pups reaching these criterion are presented in **Table 4.55**, below.

Table 4.55 Sexual maturation of F2 pups

Dose Group	0	Low	Mid	High
Vaginal opening				
Pups reaching criteria (%)	92	92	83	80
Day reaching criteria (mean)	39.61	40.77	42.58	46.44
Preputial separation				
Pups reaching criteria (%)	96	96	100	100
Day reaching criteria (mean)	43.96	44.13	44.79	47.10#

Statistically significantly different to the control group p< 0.05/ 0.01/ 0.001

The body weight of the high dose male and females of the F2 generation was significantly decreased from PN28 until PN42 (91% and 89% of control at PN42 for females and males of this group, respectively). The effects observed in this dose group on vaginal opening and preputial separation is most likely secondary to toxicity.

At necropsy of the pups there were no treatment related macroscopic findings. Absolute and relative spleen weights of the F1 and F2 pups of the mid and high dose groups were statistically significantly decreased. No missing 13th rib or cervical ribs were observed in the skeletons of the F1-pups.

In deriving a N(L)OAEL for developmental toxicity, consideration is given to the increased number of runts observed in all TCPP-treated groups in F0 generation on PN1, which may indicate toxicity to the offspring *in utero*. It is noted that an increase in runts on PN1 was not observed in F1 generation or in the preliminary study, and pup weights were also not affected on PN1 in either generation. A decrease in the mean number of pups delivered was observed in the mid dose group of the F1 generation and in the high dose groups of both generations. As discussed above and also in section 4.1.2.9.1, it is not clear whether this effect is possibly due to maternal toxicity, decreased fertility of the parental animals or a developmental effect on the pups. A decrease in pup weights during the lactation period and a decrease in spleen weight were also observed in the mid and high dose groups.

Based on a weight of evidence approach, a LOAEL of 99 mg/kg, based on the increase in runts seen in F0 generation is derived for developmental toxicity, and this value will be taken forward to risk characterisation. This may be considered to be a relatively precautionary LOAEL, as the effect on runts was not observed in both generations.

It is noted that over the course of the lactation period, increasing numbers of runts were observed in the mid dose of F0 and in high dose groups of both generations. While this could be attributed to a lactational effect, it is known that pups begin to eat treated feed during the second week of the lactation period and therefore the increase in runts during the lactational period may be due to pups eating the TCPP-treated diet. Also, as the effect on pup weight was not, or barely, observed during the first weeks of lactation, it is possibly due to consumption of TCPP-containing diets rather than a lactational effect. The numbers of pups dying in PN 1-4 could indicate enhanced toxicity of TCPP to the pups. Again, while this is possibly due to a lactational effect, the increased mortality may also be attributed to systemic toxicity to the pups *in utero.* Overall, it is considered that there is no concern for a lactational effect.

The developmental effects of TCPP were investigated in a non-GLP study (Kawasaki *et al*., 1982). In the range finding study, groups of 5 female rats were dosed (forcibly by mouth)

each day for 7 days with 8, 40, 200 or 1000 mg/kg TCPP suspended in olive oil. Body weight gain was unaffected and no abnormal behaviour or adverse symptoms were recorded. One animal dosed at 1000 mg/kg died on day 2. Kidney weights were significantly increased at 40 mg/kg (10% increase when compared to controls), 200 (increased by 20%) and 1000 mg/kg (increased by 10%). No dose-response effect was observed. Liver weight was also significantly increased at 1000 mg/kg (increased by 10% when compared to control values).

Pregnant Wistar rats were administered TCPP in solid food from days 0 – 20 of gestation. Final TCPP doses administered were 5.7 (13 dams), 57 (12 dams) or 571 (14 dams) mg/kg/day in food. 11 control dams were used. Approximately two-thirds of live foetuses were necropsied on day 20 of gestation and examined for skeletal abnormalities, with the remaining third fixed in Bouin's solution and examined for visceral abnormalities. In the post-natal phase, dams were given 0.01 (7 dams), 0.1 (6 dams) and 1% (5 dams) TCPP in the diet up to weaning. 6 control dams were used. Pups were weaned 21 days after birth and monitored until 4 weeks.

Food consumption and body weight gain among pregnant dams did not differ from controls. No other effects of TCPP were identified in the dams. **Table 4.56** below summarises the effects on the dams and the foetuses on day 20 of gestation. There were no treatment-related effects on foetal mortality, implantation number, resorption or foetal weight.

Table 4.56 Effects of TCPP on foetuses and dams fed from day 0 to day 20 of gestation

Dose (%)	**0**	**0.01**	**0.1**	**1.0**
No. of animals(dams)	11	13	12	14
No. of implants	124	135	132	158
No. of resorptions	12	5	6	8
No. of dead foetuses	0	0	0	0
Live foetuses:	Male/Female	Male/Female	Male/Female	Male/Female
No.	56/56	63/67	52/74	77/73
Weight (grams)	4.3/4.1	4.4/4.2	4.3/4.1	4.3/4.1
No. of foetuses with ext. malformations	0/0	0/0	0/0	0/0

The litters of dams fed TCPP in the diet throughout pregnancy were adjusted to an average of 8 newborns each within each group and were reared for three weeks with the dams. **Table 4.57** summarises the results obtained following examination of the newborns for abnormalities and growth.

Table 4.57 Effects of TCPP on neonatal growth

Dose (%)	**0**	**0.01**	**0.1**	**1.0**
No. of litters	5	6	7	6
At birth:				
No. of live neonates	47	60	74	61
No. of dead neonates	1	3	0	3
Live birth index (%)	89.1	89.4	96	93
Abnormality of neonates	0	0	0	0
At weaning:				
No. of dead neonates				
Male:	1	1	1	0
Female:	1	0	0	1
No. of weanlings	38	47	55	47
Weanling rate (%)	95.0	97.9	98.2	97.9
Abnormality of neonates	0	0	0	0

There were no gross abnormalities observed at the birth in any group and there was no difference in the birth rate between the test and control groups. There were no differences between the test and control groups for the weaning rate at three weeks with no abnormalities observed.

Skeletal examination was performed on foetuses from the control and treatment groups. Cervical ribs and missing 13th ribs were encountered in all treatment groups, but not in the control group. 65 control foetuses were examined and none showed cervical ribs. In the 0.01%, 0.1% and 1% treatment groups, 77, 73 and 64 foetuses were examined and 1, 1, and 3 of them showed cervical ribs, respectively. No control foetuses demonstrated missing 13th rib, while 1, 2 and 5 foetuses treated with 0.01%, 0.1% and 1% TCPP showed missing 13th ribs. The incidence of cervical ribs and missing 13th ribs was not reported on a per litter basis and therefore, it is not possible to determine whether the increase in the incidence of these effects was seen only in one litter or spread across a number of litters. Also, due to the relatively low number of foetuses examined, it is difficult to conclude on the dose-dependence and therefore, the significance of the increase in missing 13th rib. Historical control data on the incidence of missing 13th rib was also not available. However, the rib count undertaken as part of the two generation reproductive toxicity study (TNO Quality of Life, 2007) described above did not reveal any increase in missing 13th ribs or cervical ribs. Therefore, it is considered that this finding is not toxicologically significant.

Delayed ossification of the sternebrae was seen in 2 foetuses in the control group compared to 3, 7 and 1 foetuses in the 0.01%, 0.1% and 1.0% treatment groups. The authors of the report concluded that these effects were not significant. Following visceral examination of the foetuses only one case of dilatation of the renal pelvis was noted in the 0.1% treatment group. There were no other instances of abnormalities observed in any group following visceral examination. Weaning rate and rearing condition were unaffected by treatment and there was no evidence of any abnormality.

Studies in humans

No studies are available.

4.1.2.9.3 Summary of toxicity for reproduction

In a two-generation reproductive toxicity study with TCPP, there were no treatment related effects in pre-coital time, mating index, female fecundity index, male and female fertility index, duration of gestation and post-implantation loss. There was no effect on sperm parameters at necropsy. In females, the length of the longest oestrus cycle and the mean number of cycles per animal were statistically significantly increased in high dose animals of both generations. A decrease in uterus weight was observed in all dosed females in F0 and in high dose females in F1. Effects were also noted on pituitary weights, significant in high dose females of both generations. A LOAEL of 99 mg/kg is derived for effects on fertility. This is based on effects on the effect on uterus weight seen in all dosed females in F0 and high dose females in F1.

From the same study, a LOAEL of 99 mg/kg is derived for developmental toxicity. This is based on a treatment related effect on the number of runts observed in all TCPP-treated groups of the F0 generation.

In a separate study, no treatment-related effects on foetal mortality, implantation number, resorption or foetal weight were observed following treatment of pregnant dams with TCPP. Cervical ribs and missing 13th ribs were noted at a low incidence in all treatment groups, but not in the control group. However, as a specific rib count undertaken in the 2-generation study did not reveal an increase in this effect, it is concluded that this is not toxicologically significant. Weaning rate and rearing condition were unaffected by treatment and there was no evidence of any abnormality.

4.1.3 Risk characterisation [21]

4.1.3.1 General aspects

This section provides an overview of the occupational use, exposure and toxicological profile of TCPP.

Occupational exposure to TCPP may occur during the:

1. Manufacture of TCPP
2. Manufacture of flexible PUR foam
3. Cutting of flexible PUR foam
4. Production of foam granules and rebonded PUR foam
5. Formulation of systems and manufacture of spray foam
6. Use of spray foams
7. Manufacture of rigid PUR foam

[21] 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.

8. Use of rigid PUR foam
9. Manufacture of one-component foams
10. Use of one-component foams

TCPP is a liquid at room temperature, with a low vapour pressure of 1.4×10^{-3} Pa at 25^0C and a calculated saturated vapour concentration (SVC) of 0.19 mg/m^3 at 21^0C. Exposure to TCPP will be in the form of inhalation and by skin contact. Personal exposures to TCPP vapours at ambient temperature in the workplace will be low, the maximum theoretical vapour concentration being 0.19 mg/m^3. This prediction for maximum vapour concentration based on the SVC will still hold where the process is at a higher temperature, since the actual working environment will usually be about 20^0C.

The sole use of TCPP is as a flame retardant. The main downstream use of TCPP is in the production of flexible or rigid polyurethane foam. The flame retardant is not chemically reacted, but physically bound within the matrix and therefore has the potential for migration.

The TCPP 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 TCPP manufacturing was carried out by industry in the four 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 polyurethane foam, there is the potential for worker exposure to TCPP.

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.

TCPP inhalation exposures varied across the industry sectors. The highest inhalation exposure was estimated to be during the manufacture of rigid foam, with the reasonable worst case estimated to be 150 μg/m^3 and the typical exposures estimated to be 20 μg/m^3. During the production of TCPP, the typical inhalation exposure (8 hr TWA) is 25 μg/m^3. The lowest inhalation exposures are considered to occur during the use of 1K foams, with typical exposures around 2.5×10^{-3} μg/m^3.

TCPP dermal exposures again varied across the industry sectors. The highest worst-case dermal exposure was estimated to be during the production of TCPP, with a predicted worst-case exposure of 1 mg/cm^2/day. Dermal exposure was estimated to be low during scenarios such as use of rigid foams and the use of 1K foams, with typical exposures estimated to be 6×10^{-3} mg/cm^2/day and 9.3×10^{-4} mg/cm^2/day, respectively.

Information on the toxicokinetics of TCPP indicates less than 100% absorption following oral administration in animals. 80% oral absorption is used in the risk characterisation, based on available information. For the inhalation route, 100% absorption is assumed.

An *in vitro* percutaneous absorption study determined the percentage dermal penetration of TCPP through human skin at three doses. The mean total absorption was found to be 22.7%, 13.6% and 3.7%, for doses 0.002, 0.1 and 1 mg/cm^2, respectively. In a separate *in vitro* percutaneous absorption study, the percentage of TCPP absorbed across the skin as a result of

handling flexible PUR foam containing TCPP was determined to be 40%. Therefore, a figure of 23% dermal absorption is assumed for scenarios where there is exposure to "neat" TCPP and 40% dermal absorption has been taken forward for scenarios 3, 4 and 8 where there is exposure due to handling of foam containing TCPP.

TCPP was widely distributed, but concentrations in tissues were low and so bioaccumulation potential is considered to be low. TCPP was extensively metabolised, with the parent substance accounting for less than 2% of urinary or faecal radioactivity. The observed biliary/faecal excretion suggested enterohepatic recirculation.

No toxicological information is available on the effects of single exposure to TCPP in humans. In animals, TCPP is of moderate toxicity by the oral and inhalation routes and low toxicity via the dermal route. A NOAEL of 200 mg/kg was identified for the oral route.

No data are available in humans relating to skin or eye irritation. Animal studies have shown that TCPP is non-irritating to skin and eyes. It is not expected to be a respiratory tract irritant.

No data are available on the skin sensitisation potential of TCPP in humans but an animal study in guinea pigs and an LLNA showed no evidence of skin sensitisation. No information is available on the potential for TCPP to cause respiratory sensitisation.

No information is available on the effects of repeated exposure in humans. In animals, there are no data relating to repeated inhalation or dermal exposure. A study is available in which male and female rats were fed diets containing TCPP for 13 weeks at concentrations corresponding to mean substance intake values of up to 1349 mg/kg/day and 1745 mg/kg/day for males and females respectively. This study indicated that the liver and thyroid are the main target organs affected by TCPP. Effects observed included significant increases in absolute and relative liver weights in males at all doses and females at the two highest doses, periportal hepatocyte swelling in high dose groups and mild thyroid follicular cell hyperplasia in males at all doses and females at the highest dose. Although the effects on the liver may not be considered very serious, a LOAEL of 52 mg/kg/day based on increased liver weights observed in the male animals is derived from this study and taken forward to risk characterisation.

The mutagenic potential of TCPP has been well investigated *in vitro*. Evidence from several bacterial mutagenicity studies shows that TCPP is not a bacterial cell mutagen. TCPP was also shown to be non-mutagenic in fungi. In mammalian cell studies, TCPP was not genotoxic in a DNA repair assay in rat hepatocytes. It did not induce forward mutations at the TK locus in L5178Y mouse lymphoma cells in one study, but in a second mouse lymphoma study, the result was considered equivocal (in the presence of rat liver S9 fraction). A confirmatory assay was conducted, with the positive results in the presence of metabolic activation indicating that TCPP has possible clastogenic activity.

In one GLP study, TCPP did not induce unscheduled DNA synthesis *in vitro*. Two other *in vitro* UDS studies are reported. In one, TCPP gave a negative result; in the second, the result is considered equivocal. *In vivo*, TCPP was not clastogenic in a mouse micronucleus test nor did it induce an increase in chromosomal aberrations in a rat bone marrow cytogenetics assay. However, both of these studies were not in full compliance with current regulatory guidelines.

In an *in vitro/in vivo* UDS assay statistical significant increases in NNG counts and a dose response effect at one time point were observed. However, as the counts did not exceed zero at either of the doses tested, the biological significance of the effect is doubtful and thus the result is considered equivocal.

As indicated above, the results of the most recent MLA study were positive. In particular in this study, there was a clear increase in the proportion of small colony mutants. This gives rise to concern for a possible clastogenic effect of TCPP and in order to further investigate this an *in vivo* Comet assay was conducted to assess the potential for DNA strand breaks and DNA damage in the livers of rats treated with either 750 or 1500 mg/kg TCPP. Comet analysis of liver tissue provided tail moment and tail intensity values that were considered consistent with control groups and it was concluded that TCPP did not induce DNA damage in the liver of treated rats. Therefore, TCPP is not considered to be genotoxic *in vivo*.

No carcinogenicity studies have been carried out with TCPP. The study of longest duration for TCPP is a 90-day dietary study in rats. Increased liver weights (both relative and absolute) were observed in males at 52 mg/kg and above and periportal hepatocyte swelling was noted at highest dose (1349 mg/kg in males and 1745 mg/kg in females). In addition, mild follicular cell hyperplasia was noted in females at 1745 mg/kg and in all dosed males. In the kidney, vacuolation in females at highest dose was also observed. A slightly excessive fatty infiltration indicative of mild bone marrow hypoplasia was noted in three high dose females. The LOAEL of 52 mg/kg/day is based on increased liver weights observed in males. In the absence of carcinogenicity data, it cannot be excluded that the effects observed in this study with TCPP may progress to cancer. Therefore, as a reasonable worse case approach, this data will be used in a quantitative way to carry out a risk characterisation for carcinogenicity.

This initial concern for carcinogenicity is further supported by the fact that TCPP is structurally similar to two other chlorinated alkyl phosphate esters, TDCP and TCEP. TDCP and TCEP are considered to be non-genotoxic carcinogens and have agreed classifications of Carc. Cat. 3; R40[22]). It is considered that there is sufficient information from the structures, physical-chemical properties, toxicokinetics and mutagenic profiles of TCPP and the structurally similar substances, TCEP and TDCP, to support a qualitative read-across for carcinogenicity. However, differences in the metabolism, target organs, the severity of the effects observed and the potency of the three substances indicates that a quantative read-across for carcinogenicity from either TDCP or TCEP may not be appropriate. The proposal for read-across to TDCP and TCEP is presented in full in Appendix D.

Therefore, it is proposed that the LOAEL of 52 mg/kg/day, identified from the 90-day study with TCPP, should be used as a basis for risk characterisation of the carcinogenicity endpoint.

A 2-generation reproductive toxicity study with TCPP found no treatment related differences in pre-coital time, mating index, female fecundity index, male and female fertility index, duration of gestation and post-implantation loss. In females, the length of the longest oestrus cycle and the mean number of cycles per animal were statistically significantly increased in high dose animals of both generations. A decrease in uterus weight was observed in all dosed females in F0 generation and in high dose females of F1 generation. There was no effect on sperm parameters at necropsy. No treatment related microscopic effects were observed at necropsy. A LOAEL of 99 mg/kg is derived for effects on fertility, based on effects on the uterus weight seen in all dosed females in F0 and high dose females in F1.

In the same study, an increase in the number of runts was observed in all dose groups of F0 generation on PN1 and persisted to PN21 in the mid and high dose groups. In the F1 generation, the number of runts was increased in the high dose group on PN14 and all dose groups on PN21. A decrease in mean pup weight was noted in high dose group of F0 from

[22] 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,

PN14 onwards and of F1 from PN 7. Mean pups weights were decreased in the mid dose group of both generations on PN21. A decrease in the mean number of pups delivered was observed in the mid and high dose groups and could be due either to decreased fertility of parental animals or a developmental effect on the pups. No treatment related macroscopic alterations were observed at necropsy of the pups. No missing 13^{th} rib or cervical ribs were observed in the skeletons of the F1-pups. There were no treatment related differences on anogenital distance, vaginal opening and preputial separation between the TCPP fed groups and the controls. Based on the increased number of runts observed in all dose groups of F0 generation, a LOAEL of 99 mg/kg is derived for developmental toxicity.

4.1.3.2 Workers

The total number of persons occupationally exposed to TCPP in the EU through the various exposure scenarios is unknown.

Occupational exposure to TCPP occurs primarily by the dermal and inhalation routes of exposure. Ingestion is not considered for workers in this risk assessment. Exposure levels used for the manufacture and use of TCPP have been derived from both measured data supplied by industry and EASE modelling.

For most toxicological endpoints, data on TCPP have been generated from oral studies. Therefore, it is important to consider route-to-route extrapolation (to the dermal and inhalation exposure) in the risk characterisation. The available toxicokinetic data following oral administration of TCPP indicate that it is extensively metabolised by first pass metabolism in the liver to more polar metabolites. If no such extensive metabolism occurs after dermal and inhalation exposure, route-to-route extrapolation will potentially result in an underestimation of systemic exposure to the parent compound. However, the route-independent low acute toxicity of TCPP, and the comparable toxicokinetic behaviour of the structurally related TDCP after oral and dermal exposure indicate that such a correction appears not to be needed here.

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 exposures for all of the identified exposure scenarios.

Scenario 1: Manufacture of TCPP

With regard to TCPP production, the reasonable worst-case inhalation exposure is 25 $\mu g/m^3$. 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 3.5×10^{-3} mg/kg. For dermal exposure in this scenario, the reasonable worst-case exposure is 1 mg/cm^2/day. Using default values of a 70kg worker with 210 cm^2 of exposed skin and assuming 23% absorption, the dermal body burden is 0.69 mg/kg. Combining the two values gives a calculated total body burden of 0.69 mg/kg for this scenario.

The typical inhalation exposure for this scenario is 12.5 $\mu g/m^3$. Using the default values stated above, the inhalation body burden is 1.8×10^{-3} mg/kg. For dermal exposure in this scenario, the typical exposure is 0.1 mg/cm^2/day, leading to a dermal body burden of 6.9×10^{-2} mg/kg. Combining the two values gives a calculated total body burden of 7.1×10^{-2} mg/kg.

Scenario 2: Manufacture of flexible PUR foam

Regarding the manufacture of flexible polyurethane 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 0.07 mg/cm^2/day. Using default values of a 70 kg worker with 420 cm^2 of exposed skin and assuming 23% absorption, the dermal body burden is 9.7 x 10^{-2} mg/kg. Combining the two values gives a calculated total body burden of 9.8 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 0.002 mg/cm^2/day, leading to a dermal body burden of 2.8 x 10^{-3} mg/kg. Combining the two values gives a calculated total body burden of 2.9 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 40% absorption, the dermal body burden is 1.7 x 10^{-2} mg/kg. Combining the two values gives a calculated total body burden of 1.8 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 2.4 x 10^{-3} mg/kg. Combining the two values gives a calculated total body burden of 2.7 x 10^{-3} mg/kg.

Scenario 4: Production of foam granules and rebonded PUR foam

Regarding the exposure scenario of the production of foam granules and rebonded foam, the reasonable worst-case inhalation exposure during handling of the foam blocks 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 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 40% absorption, the dermal body burden is 4.1 x 10^{-3} mg/kg. Combining the two values gives a total reasonable worst-case body burden of 4.7 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, giving a dermal body burden of 1.3 x 10^{-3} mg/kg. The total body burden following typical exposure is 1.4 x 10^{-3}.

Scenario 5: Formulation of systems and manufacture of spray foam

Regarding the formulation of systems and manufacture of spray foams, the reasonable worst-case inhalation exposure is 5 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 7.1 x 10^{-4} mg/kg. For dermal exposure in this scenario, the reasonable worst-case exposure is 0.11 mg/cm^2/day. Using default values of a 70 kg worker with 420 cm^2 of exposed skin and

assuming 23% absorption, the dermal body burden is 0.15 mg/kg. Combining the two values gives a calculated total body burden of 0.15 mg/kg for this scenario.

The typical inhalation exposure for this scenario is 2.5 µg/m^3. Using the default values stated above, the inhalation body burden is 3.6 x 10^{-4} mg/kg. For dermal exposure in this scenario, the typical exposure is 0.05 mg/cm^2/day, leading to a dermal body burden of 6.9 x 10-2 mg/kg. Combining the two values gives a calculated total body burden of 6.9 x 10^{-2} mg/kg.

Scenario 6: Use of spray foams

Regarding exposure during the use of spray foams, the reasonable worst-case inhalation exposure is 187.5 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 2.7 x 10^{-2} mg/kg. For dermal exposure in this scenario, the reasonable worst-case exposure is 0.23 mg/cm^2/day. Using default values of a 70 kg worker with 420 cm^2 of exposed skin and assuming 23% absorption, the dermal body burden is 0.32 mg/kg. Combining the two values gives a calculated total body burden of 0.35 mg/kg for this scenario.

The typical inhalation exposure for this scenario is 25 µg/m^3. Using the default values stated above, the inhalation body burden is 3.6 x 10^{-3} mg/kg. For dermal exposure in this scenario, the typical exposure is 0.12 mg/cm^2/day, leading to a dermal body burden of 0.17 mg/kg. Combining the two values gives a calculated total body burden of 0.17 mg/kg.

Scenario 7: Manufacture of rigid PUR foam

Regarding the manufacture of rigid PUR foam, the reasonable worst-case inhalation exposure is 150 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 2.1 x 10^{-2} mg/kg. For dermal exposure in this scenario, the reasonable worst-case exposure is 6.5 x 10^{-2} mg/cm^2/day. Using default values of a 70kg worker with 210 cm^2 of exposed skin and assuming 23% absorption, the dermal body burden is 4.5 x 10^{-2} mg/kg. Combining the two values gives a calculated total body burden of 6.6 x 10^{-2} mg/kg for this scenario.

The typical inhalation exposure for this scenario is 20 µg/m^3. Using the default values stated above, the inhalation body burden is 2.9 x 10^{-3} mg/kg. For dermal exposure in this scenario, the typical exposure is 3.2 x 10^{-2} mg/cm^2/day, leading to a dermal body burden of 2.2 x 10^{-2} mg/kg. Combining the two values gives a calculated total body burden of 2.5 x 10^{-2} mg/kg.

Scenario 8: Use of rigid PUR foam

With regard to the use of rigid 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 1.3 x 10^{-2} mg/cm^2/day. Using default values of a 70 kg worker with 210 cm^2 of exposed skin and 40% absorption, the dermal body burden is 1.6 x 10^{-2} mg/kg. Combining the two values gives a calculated total body burden of 1.6 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 6 x 10^{-3} mg/cm^2/day, leading to a dermal body burden of 7.2 x 10^{-3} mg/kg. Combining the two values gives a calculated total body burden of 7.5 x 10^{-3}mg/kg.

Scenario 9: Manufacture of one-component foams

With regard to the manufacture of one-component (1-K) foams, the reasonable worst-case inhalation exposure is 12.5 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 1.8 x 10^{-3} mg/kg. For dermal exposure in this scenario, the reasonable worst-case exposure is 5.2 x 10^{-3} mg/cm^2/day. Using default values of a 70 kg worker with 210 cm^2 of exposed skin and assuming 23% absorption, the dermal body burden is 3.6 x 10^{-3} mg/kg. Combining the two values gives a calculated total body burden of 5.4 x 10^{-3} mg/kg for this scenario.

The typical inhalation exposure for this scenario is 6.7 µg/m^3. Using the default values stated above, the inhalation body burden is 9.6 x 10^{-4} mg/kg. For dermal exposure in this scenario, the typical exposure is 1 x 10^{-3} mg/cm^2/day, leading to a dermal body burden of 6.9 x 10^{-4} mg/kg. Combining the two values gives a calculated total body burden of 1.7 x 10^{-3} mg/kg.

Scenario 10: Use of one-component foams

Regarding exposure during the use of 1-K foams, the reasonable worst-case inhalation exposure is 5 x 10^{-3} 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 x 10^{-7} mg/kg. For dermal exposure in this scenario, the reasonable worst-case exposure is 1.9 x 10^{-3} mg/cm^2/day. Using default values of a 70 kg worker with 420 cm^2 of exposed skin and assuming 23% absorption, the dermal body burden is 2.6 x 10^{-3} mg/kg. Combining the two values gives a calculated total body burden of 2.6 x 10^{-3} mg/kg for this scenario.

The typical inhalation exposure for this scenario is 2.5 x 10^{-3} µg/m^3. Using the default values stated above, the inhalation body burden is 3 x 10^{-7} mg/kg. For dermal exposure in this scenario, the typical exposure is 9.3 x 10^{-4}mg/cm^2/day, leading to a dermal body burden of 1.3 x 10^{-3} mg/kg. Combining the two values gives a calculated total body burden of 1.3 x 10^{-3} mg/kg.

Table 4.58 summarises the dermal and inhalation body burden values for all TCPP exposure scenarios.

Table 4.58 Summary of dermal and inhalation body burden values for all TCPP 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	3.5×10^{-3}	0.69	0.69	1.8×10^{-3}	6.9×10^{-2}	7.1×10^{-2}
2	7.3×10^{-4}	9.7×10^{-2}	9.8×10^{-2}	8.9×10^{-5}	2.8×10^{-3}	2.9×10^{-3}
3	5.9×10^{-4}	1.7×10^{-2}	1.8×10^{-2}	2.7×10^{-4}	2.4×10^{-3}	2.7×10^{-3}
4	6.6×10^{-4}	4.1×10^{-3}	4.7×10^{-3}	8.4×10^{-5}	1.3×10^{-3}	1.4×10^{-3}
5	7.1×10^{-4}	0.15	0.15	3.6×10^{-4}	6.9×10^{-2}	6.9×10^{-2}
6	2.7×10^{-2}	0.32	0.35	3.6×10^{-3}	0.17	0.17
7	2.1×10^{-2}	4.5×10^{-2}	6.6×10^{-2}	2.9×10^{-3}	2.2×10^{-2}	2.5×10^{-2}
8	5.9×10^{-4}	1.6×10^{-2}	1.6×10^{-2}	2.7×10^{-4}	7.2×10^{-3}	7.5×10^{-3}
9	1.8×10^{-3}	3.6×10^{-3}	5.4×10^{-3}	9.6×10^{-4}	6.9×10^{-4}	1.7×10^{-3}
10	7×10^{-7}	2.6×10^{-3}	2.6×10^{-3}	3×10^{-7}	1.3×10^{-3}	1.3×10^{-3}

The exposure scenarios referred to by numbers in the above table are:

1. Manufacture of TCPP
2. Manufacture of flexible PUR foam
3. Cutting of flexible PUR foam
4. Production of foam granules & rebonded PUR foam
5. Formulation of systems and manufacture of spray foam
6. Use of spray foams
7. Manufacture of rigid PUR foam
8. Use of rigid PUR foam
9. Manufacture of one-component foams
10. Use of one-component foams

4.1.3.2.1 Acute toxicity

No significant signs of toxicity were seen in experimental animals via the inhalation and dermal routes.

With respect to oral exposure, a NOAEL of 200 mg/kg was identified from the acute oral toxicity studies. Assuming 80% absorption by the oral route, this leads to an internal body burden of 160 mg/kg.

In line with the draft TGD (2005), the minimal MOS for acute toxicity is 50. This mMOS is established taking into account an interspecies factor of 10 (4 for metabolic size differences * 2.5 for sensitivity differences) and an intraspecies factor of 5.

For scenario 1, manufacture of TCPP, with respect to inhalation exposure, the body burden for reasonable worst case is 3.5×10^{-3} mg/kg. When this is compared with the internal body burden of 160 mg/kg the MOS value is 45,714. Regarding dermal exposure, the body burden for reasonable worst case is 0.69 mg/kg, leading to a MOS of 232. The combined reasonable worst case body burden is also 0.69 mg/kg, giving a MOS of 232. The typical body burden for the inhalation exposure is1.8×10^{-3} mg/kg, which when compared with the internal body

burden results in a MOS of 88,889. For the dermal exposure, the typical body burden is 0.069 mg/kg, leading to a MOS of 2,319. The combined typical exposure body burden for this scenario is 0.071 mg/kg, leading to a MOS of 2,254.

When compared to the minimal MOS of 50, it is concluded that the MOSs are sufficient and there are no concerns for acute toxicity for this scenario and so **conclusion (ii)** is drawn.

Regarding scenario 2, the manufacture of flexible PUR foam, the body burden for reasonable worst-case inhalation exposure is 7.3 x 10^{-4} mg/kg, which when compared with the internal body burden results in a MOS of 219,178. The body burden for reasonable worst-case dermal exposure is 9.7 x 10^{-2} mg/kg. This gives a MOS of 1,649. The combined body burden for reasonable worst-case exposure is 9.8 x 10^{-2} mg/kg, leading to a MOS of 1,633. For this scenario, the typical inhalation body burden is 8.9 x 10^{-5} mg/kg. When this is compared with the internal body burden, the MOS is >1,000,000. The typical dermal body burden is estimated to be 2.8 x 10^{-3} mg/kg, leading to a MOS of 57,143. The combined body burden for the typical exposures is 2.9 x 10^{-3}, resulting in a MOS of 55,172

When compared to the minimal MOS of 50, it is concluded that the MOSs are sufficient and there are no concerns for acute toxicity for this scenario and so **conclusion (ii)** is drawn.

For scenario 5, formulation of systems and manufacture of spray foam, the body burden for reasonable worst-case inhalation exposure is 7.1 x 10^{-4} mg/kg. When compared with the internal body burden of 160 mg/kg, the MOS is 225,352. The body burden for reasonable worst-case dermal exposure is 0.15 mg/kg, leading to a MOS of 1,067. The combined body burden for reasonable worst-case exposure is also 0.15 mg/kg, resulting in a MOS of 1,067. For this scenario, the inhalation body burden for the typical exposure is 3.6 x 10^{-4} mg/kg, which when compared with the internal body burden results in a MOS of 444,444. The typical dermal and combined body burdens are estimated to be 6.9 x 10^{-2} mg/kg, leading to a MOSs of 2,319 for both.

When compared to the minimal MOS of 50, it is concluded that the MOSs are sufficient and there are no concerns for acute toxicity for this scenario and so **conclusion (ii)** is drawn.

With respect to scenario 6, the use of spray foams, the body burden for reasonable worst-case inhalation exposure is 2.7 x 10^{-2} mg/kg, resulting in a MOS of 5,926. The body burden for reasonable worst-case dermal exposure is 0.32 mg/kg, giving a MOS of 500. The combined body burden is 0.35 mg/kg, resulting in a MOS of 457. For this scenario, the inhalation body burden for the typical exposure is 3.6 x 10^{-3} mg/kg, leading to a MOS of 44,444. The dermal 1and combined typical exposure body burdens for this scenario are 0.17 mg/kg. This gives a MOS of 941 for both.

When compared to the minimal MOS of 50, it is concluded that the MOSs are sufficient and there are no concerns for acute oral toxicity for this scenario and so **conclusion (ii)** is drawn.

As **conclusions (ii)**s are drawn for the 4 exposure scenarios detailed above, and these scenarios gave the highest potential reasonable worst-case and typical exposures, a risk characterisation will not be carried out for the remaining exposure scenarios, as they would also result in a conclusion of no concern. Therefore, **conclusion (ii)** is drawn for all remaining exposure scenarios for acute toxicity.

Tables 4.59 and **4.60** summarise the MOSs and conclusions for acute toxicity for the reasonable worst case and typical exposures, respectively.

Table 4.59 MOS values and conclusions for acute toxicity of TCPP – 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 TCPP	3.5×10^{-3}	45,714	(ii)	0.69	232	(ii)	0.69	232	(ii)
2.Manufacture of flexible PUR foam	7.3×10^{-4}	219,178	(ii)	9.7×10^{-2}	1,649	(ii)	9.8×10^{-2}	1,633	(ii)
5.Formulation of systems & manufacture of spray foam	7.1×10^{-4}	225,352	(ii)	0.15	1,067	(ii)	0.15	1,067	(ii)
6. Use of spray foams	2.7×10^{-2}	5,926	(ii)	0.32	500	(ii)	0.35	457	(ii)

Table 4.60 MOS values and conclusions for acute toxicity of TCPP – 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 TCPP	1.8×10^{-3}	88,889	(ii)	6.9×10^{-2}	2,319	(ii)	7.1×10^{-2}	2,254	(ii)
2.Manufacture of flexible PUR foam	8.9×10^{-5}	>1,000,000	(ii)	2.8×10^{-3}	57,143	(ii)	2.9×10^{-3}	55,172	(ii)
5.Formulation of systems & manufacture of spray foam	3.6×10^{-4}	444,444	(ii)	6.9×10^{-2}	2,319	(ii)	6.9×10^{-2}	2,319	(ii)
6. Use of spray foams	3.6×10^{-3}	44,444	(ii)	0.17	941	(ii)	0.17	941	(ii)

4.1.3.2.2 Irritation and corrosivity

TCPP 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.

4.1.3.2.3 Sensitisation

Skin

Based on available data, TCPP is not considered to be a skin sensitiser. **Conclusion (ii)** is drawn for this end-point, for all exposure scenarios.

Respiratory tract

No data are available on the respiratory sensitisation potential of TCPP. There is currently no validated test method available to identify respiratory sensitisers. As TCPP is produced in a closed system, and has a low vapour pressure, it is expected that exposure of the respiratory tract will be low. TCPP 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.

4.1.3.2.4 Repeated dose toxicity

In relation to repeated dose toxicity, a LOAEL of 52 mg/kg/day was derived from a 13-week study in which male and female rats were dosed with TCPP at concentrations of up to 1349 mg/kg/day and 1745 mg/kg/day, respectively. This LOAEL was based on increased liver weights observed in male animals. Assuming 80% absorption by the oral route, this leads to an internal body burden of 42 mg/kg/day.

In line with the draft TGD (2005), the minimal MOS for repeated dose toxicity is 50. 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 5. Normally a further factor of 3 would be used to take into account the use of a LOAEL rather than a NOAEL. However, this is not considered necessary here, as the adverse effect in the repeated dose toxicity study (mainly liver weight changes) are not considered particularly toxicologically significant and the LOAEL is probably quite close to the NOAEL. In addition, a factor to allow for semi chronic to chronic extrapolation (usually a factor of 2) was not used here. It is not considered necessary, as relatively similar effects on the liver, at doses of a comparable order of magnitude were observed in both the 28-day and the 90-day studies and so it is felt that exposure duration is not significant.

For scenario 1, manufacture of TCPP, with respect to inhalation exposure, the body burden for reasonable worst-case exposure is 3.5×10^{-3} mg/kg. When this is compared with the internal body burden for repeat dose toxicity of 42 mg/kg, the MOS is 12,000. With respect to dermal exposure, the body burden for reasonable worst-case exposure is 0.69 mg/kg, leading to a MOS of 61. The total body burden for reasonable worst case for this scenario is also 0.69 mg/kg, again leading to a MOS of 61. For this scenario, the body burden for the typical inhalation exposure is 1.8×10^{-3} mg/kg, leading to a MOS of 23,333. For the typical dermal exposure, the body burden is 6.9×10^{-2} mg/kg, which results in a MOS of 609. The combined body burden for the typical exposure is 7.1×10^{-2} mg/kg, leading to a MOS of 592.

When the MOSs are compared with the minimal MOS of 50, there is no concern for this scenario. Therefore, **conclusion (ii)** is drawn for worst-case and typical exposures.

Regarding scenario 2, the manufacture of flexible PUR foam, the body burden for reasonable worst-case inhalation exposure is 7.3×10^{-4} mg/kg, which when compared with the internal body burden gives a MOS of 57,534. The body burden for reasonable worst-case dermal exposure is 9.7×10^{-2} mg/kg. This gives a MOS of 433. The combined body burden for reasonable worst-case exposure is 9.8×10^{-2} mg/kg, leading to a MOS of 429. For the typical exposures, the inhalation body burden is 8.9×10^{-5} mg/kg, leading to a MOS of 471,910. The dermal body burden for this scenario is 2.8×10^{-3} mg/kg, leading to a MOS of 15,000. The body burden for the combined exposure is 2.9×10^{-3}, resulting in a MOS of 14,483.

When the MOSs are compared with the minimal MOS of 50, there is no concern for this scenario. Therefore, **conclusion (ii)** is drawn for reasonable worst case and typical exposures.

For scenario 3, cutting of flexible foam, the body burden for reasonable worst-case inhalation exposure is 5.9 x 10^{-4} mg/kg. When this is compared with the internal body burden of 42 mg/kg, the MOS is 71,186. The body burden for reasonable worst-case dermal exposure is 1.7 x 10^{-2} mg/kg, leading to a MOS of 2,471. The combined body burden for reasonable worst-case exposure is 1.8 x 10^{-2} mg/kg, resulting in a MOS of 2,333. The typical inhalation body burden is 2.7 x 10^{-4} mg/kg, which gives a MOS of 155,556. The typical dermal body burden is 2.4 x 10^{-3} mg/kg, resulting in a MOS of 17,500. The combined typical exposure for this scenario is 2.7 x 10^{-3} mg/kg, leading to a MOS of 15,556.

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.

Regarding scenario 4, the production of foam granules and rebonded foam, with respect to inhalation exposure, the body burden for reasonable worst-case exposure is 6.6 x 10^{-4} mg/kg, resulting in a MOS value of 63,636. Regarding dermal exposure for this exposure scenario, the body burden for reasonable worst-case exposure is 4.1 x 10^{-3} mg/kg, leading to a MOS of 10,244. The total body burden for reasonable worst-case for this scenario is 4.7 x 10^{-3} mg/kg, and so results in a MOS of 8,936. For the typical exposure, the inhalation body burden is 8.4 x 10^{-5} mg/kg, leading to a MOS of 500,000. For the typical dermal exposure, the body burden is 1.3 x 10^{-3} mg/kg, leading to a MOS of 32,308. The combined body burden for the typical exposure is 1.4 x 10^{-3} mg/kg, resulting in a MOS of 30,000.

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 5, the formulation of systems and manufacture of spray foams, with respect to inhalation exposure, the body burden for reasonable worst-case exposure is 7.1 x 10^{-4} mg/kg. When this is compared with the internal body burden of 42 mg/kg, the MOS is 59,155. Regarding dermal exposure, the body burden for reasonable worst-case exposure is 0.15 mg/kg, leading to a MOS of 280. The total body burden for reasonable worst-case for this scenario is also 0.15 mg/kg, and so this also results in a MOS of 280. For the typical exposure, the inhalation body burden is 3.6 x 10^{-4} mg/kg, which when compared with the internal body burden results in a MOS of 116,667. For the dermal and combined exposure, the body burdens are 6.9 x 10^{-2} mg/kg, leading to a MOS of 609 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.

Regarding scenario 6, the use of spray foams, the body burden for reasonable worst-case inhalation exposure is 2.7 x 10^{-2} mg/kg, which when compared with the internal body burden leads to a MOS of 1,556. The body burden for reasonable worst-case dermal exposure is 0.32 mg/kg, giving a MOS of 131. The combined body burden is 0.35 mg/kg and results in a MOS of 120. The body burden for the typical inhalation exposure is 3.6 x 10^{-3} mg/kg, leading to a MOS of 11,667. The dermal and combined typical exposure body burdens for this scenario are both 0.17 mg/kg (the combined body burden value was driven by the dermal exposure estimate), which give a MOS of 247.

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 7, the manufacture of rigid PUR foam, with respect to inhalation exposure, the body burden for reasonable worst-case exposure is 2.1 x 10^{-2} mg/kg. When this is compared with the internal body burden, the resulting MOS value is 2,000. Regarding dermal exposure, the body burden for reasonable worst-case exposure is 4.5 x 10^{-2} mg/kg, leading to a MOS of 933. The total body burden for reasonable worst-case for this scenario is 6.6 x 10^{-2} mg/kg, and so results in a MOS of 636. For the typical inhalation exposure, the body burden is 2.9 x 10^{-3} mg/kg, leading to a MOS of 14,483. The dermal exposure body burden for the typical exposure is 2.2 x 10^{-2} mg/kg, giving a MOS of 1,909. For the combined exposure, body burden is 2.5 x 10^{-2} mg/kg, leading to a MOS of 1,680.

When the MOSs are compared with the minimal MOS of 50, there is no concern for this scenario. Therefore, **conclusion (ii)** is drawn for reasonable worst case and typical exposures.

For scenario 8, the use of rigid PUR foam, with respect to inhalation exposure, the body burden for reasonable worst-case exposure is 5.9 x 10^{-4} mg/kg. Comparing this with the internal body burden of 42 mg/kg results in a MOS value of 71,186. Regarding dermal exposure, the body burden for reasonable worst-case exposure is 1.6 x 10^{-2} mg/kg, leading to a MOS of 2,625. The total body burden for reasonable worst-case for this scenario is also 1.6 x 10^{-2} mg/kg, resulting in a MOS of 2,625. For the typical inhalation exposure, the body burden is 2.7 x 10^{-4} mg/kg, leading to a MOS of 155,556. The dermal typical body burden for this scenario is 7.2 x 10^{-3} mg/kg, leading to a MOS of 5,833. The combined body burden is 7.5 x 10^{-3} mg/kg, giving a MOS of 5,600.

When all of 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.

Regarding scenario 9, the manufacture of one-component foams, the body burden for reasonable worst-case inhalation exposure is 1.8 x 10^{-3} mg/kg. Comparing this to the internal body burden of results in a MOS of 23,333. The body burden for reasonable worst-case dermal exposure is 3.6 x 10^{-3} mg/kg, giving a MOS of 11,667. The combined body burden is 5.4 x 10^{-3} mg/kg and so results in a MOS of 7,778. For the typical exposure, the inhalation body burden is 9.6 x 10^{-4} mg/kg, giving a MOS of 43,750. The typical dermal exposure body burden for this scenario is 6.9 x 10^{-4} mg/kg. This gives a MOS of 60,870. The combined body burden for the typical exposure is 1.7 x 10^{-3} mg/kg, leading to a MOS of 24,706.

When the MOSs are compared with the minimal MOS of 50, there is no concern for this scenario, and so a **conclusion (ii)** can be drawn.

Regarding scenario 10, the use of one-component foams, the body burden for reasonable worst-case inhalation exposure is 7 x 10^{-7} mg/kg, resulting in a MOS of >1,000,000. The body burden for reasonable worst-case dermal exposure is 2.6 x 10^{-3} mg/kg, giving a MOS of 16,154. The combined body burden is also 2.6 x 10^{-3} mg/kg and so also results in a MOS of 16,154. The typical inhalation exposure body burden for this scenario is 3 x 10^{-7} mg/kg, resulting in a MOS of > 1,000,000. The typical dermal and combined body burdens are 1.3 x 10^{-3} mg/kg, resulting in a MOS of 32,308 for both.

When the MOSs are compared with the minimal MOS of 50, there is no concern for this scenario, and so a **conclusion (ii)** can be drawn.

Tables 4.61 and **4.62** summarise the MOSs and conclusions for repeated dose toxicity for the reasonable worst case and typical exposures, respectively.

Table 4.61 MOS values and conclusions for repeated dose toxicity of TCPP – 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 TCPP	3.5×10^{-3}	12,000	(ii)	0.69	61	(ii)	0.69	61	(ii)
2.Manufacture of flexible PUR foam	7.3×10^{-4}	57,534	(ii)	9.7×10^{-2}	433	(ii)	9.8×10^{-2}	429	(ii)
3.Cutting of flexible PUR foam	5.9×10^{-4}	71,186	(ii)	1.7×10^{-2}	2,471	(ii)	1.8×10^{-2}	2,333	(ii)
4.Production of foam granules & rebonded foam	6.6×10^{-4}	63,636	(ii)	4.1×10^{-3}	10,244	(ii)	4.7×10^{-3}	8,936	(ii)
5.Formulation of systems and mfgr of spray foams	7.1×10^{-4}	59,155	(ii)	0.15	280	(ii)	0.15	280	(ii)
6.Use of spray foams	2.7×10^{-2}	1,556	(ii)	0.32	131	(ii)	0.35	120	(ii)
7.Manufacture of rigid PUR foam	2.1×10^{-2}	2,000	(ii)	4.5×10^{-2}	933	(ii)	6.6×10^{-2}	636	(ii)
8.Use of rigid foam	5.9×10^{-4}	71,186	(ii)	1.6×10^{-2}	2,625	(ii)	1.6×10^{-2}	2,625	(ii)
9.Production of 1-K foams	1.8×10^{-3}	23,333	(ii)	3.6×10^{-3}	11,667	(ii)	5.4×10^{-3}	7,778	(ii)
10.Use of 1-K foams	7×10^{-7}	>1,000,000	(ii)	2.6×10^{-3}	16,154	(ii)	2.6×10^{-3}	16,154	(ii)

Table 4.62 MOS values and conclusions for repeated dose toxicity of TCPP – 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 TCPP	1.8×10^{-3}	23,333	(ii)	6.9×10^{-2}	609	(ii)	7.1×10^{-2}	592	(ii)
2.Manufacture of flexible PUR foam	8.9×10^{-5}	471,910	(ii)	2.8×10^{-3}	15,000	(ii)	2.9×10^{-3}	14,483	(ii)
3.Cutting of flexible PUR foam	2.7×10^{-4}	155,556	(ii)	2.4×10^{-3}	17,500	(ii)	2.7×10^{-3}	15,556	(ii)
4.Production of foam granules & rebonded foam	8.4×10^{-5}	500,000	(ii)	1.3×10^{-3}	32,308	(ii)	1.4×10^{-3}	30,000	(ii)
5.Formulation of systems and mfgr of spray foams	3.6×10^{-4}	116,667	(ii)	6.9×10^{-2}	609	(ii)	6.9×10^{-2}	609	(ii)
6.Use of spray foams	3.6×10^{-3}	11,667	(ii)	0.17	247	(ii)	0.17	247	(ii)
7.Manufacture of rigid PUR foam	2.9×10^{-3}	14,483	(ii)	2.2×10^{-2}	1,909	(ii)	2.5×10^{-2}	1,680	(ii)
8.Use of rigid foam	2.7×10^{-4}	155,556	(ii)	7.2×10^{-3}	5,833	(ii)	7.5×10^{-3}	5,600	(ii)
9.Production of 1-K foams	9.6×10^{-4}	43,750	(ii)	6.9×10^{-4}	60,870	(ii)	1.7×10^{-3}	24,706	(ii)
10.Use of 1-K foams	3×10^{-7}	>1,000,000	(ii)	1.3×10^{-3}	32,308	(ii)	1.3×10^{-3}	32,308	(ii)

4.1.3.2.5 Mutagenicity

A TCPP *in vitro* mouse lymphoma assay was positive, indicating TCPP has possible clastogenic activity. *In vivo*, TCPP was not clastogenic in a mouse micronucleus test, nor did it induce an increase in chromosomal aberrations in a rat bone marrow cytogenetics assay. However, both of these studies were not in full compliance with current regulatory guidelines. An *in vitro/in vivo* UDS assay was considered to be equivocal. In an *in vivo* Comet assay, TCPP did not induce DNA damage in the livers of treated rats.

Based on the weight of available information, TCPP is considered to be non-genotoxic *in vivo* and a **conclusion (ii)** is drawn for this endpoint for all exposure scenarios.

4.1.3.2.6 Carcinogenicity

No carcinogenicity studies have been carried out with TCPP. As described in section 4.1.2.8, it is proposed that the LOAEL of 52 mg/kg/day, identified from the 90-day study with TCPP will be taken forward for risk characterisation. Assuming 80% absorption by the oral route, this leads to an internal body burden of 42 mg/kg/day.

The minimal MOS for carcinogenicity 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. Normally, a factor of 3 would be used to take into account the use of a LOAEL rather than a NOAEL. However, this is not considered necessary here, as the LOAEL derived from the repeat dose toxicity study was based on liver weight changes which are not considered to be particularly toxicologically significant and the LOAEL is probably quite close to the NOAEL.

For scenario 1, manufacture of TCPP, with respect to inhalation exposure, the body burden for reasonable worst-case exposure is 3.5×10^{-3} mg/kg. When this is compared with the internal body burden for repeat dose toxicity of 42 mg/kg, the MOS is 12,000. With respect to dermal exposure, the body burden for reasonable worst-case exposure is 0.69 mg/kg, leading to a MOS of 61. The reasonable worst combined body burden is also 0.69 mg/kg, again leading to a MOS of 61. For this scenario, the body burden for the typical inhalation exposure is 1.8×10^{-3} mg/kg, leading to a MOS of 23,333. For the typical dermal exposure, the body burden is 6.9×10^{-2} mg/kg, which results in a MOS of 609. The combined body burden for the typical exposure is 7.1×10^{-2} mg/kg, leading to a MOS of 592.

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 the reasonable worst case and typical dermal and inhalation exposures.

Regarding scenario 2, the manufacture of flexible PUR foam, the body burden for reasonable worst-case inhalation exposure is 7.3×10^{-4} mg/kg, resulting in a MOS of 57,534. The body burden for reasonable worst-case dermal exposure is 9.7×10^{-2} mg/kg, leading to a MOS of 433. The combined body burden for reasonable worst-case exposure is 9.8×10^{-2} mg/kg, leading to a MOS of 429. The body burden for the typical inhalation exposure is 8.9×10^{-5} mg/kg, leading to a MOS of 471,910. The body burden for the typical dermal exposure is 2.8×10^{-3} mg/kg, leading to a MOS of 15,000. The typical combined exposure is 2.8×10^{-3} mg/kg, resulting in a MOS of 14,483.

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 the reasonable worst case and typical dermal and inhalation exposures.

For scenario 3, cutting of flexible foam, with respect to inhalation exposure, the body burden for reasonable worst-case exposure is 5.9×10^{-4} mg/kg, which when compared with the internal body burden of 42 mg/kg leads to a MOS of 71,186. The body burden for reasonable worst-case dermal exposure is 1.7×10^{-2} mg/kg, leading to a MOS of 2,471. The combined body burden for reasonable worst-case exposure is 1.8×10^{-2} mg/kg, resulting in a MOS of 2,333. The typical inhalation body burden is 2.7×10^{-4} mg/kg. This gives a MOS of 155,556. The typical dermal body burden is 2.4×10^{-3} mg/kg, resulting in a MOS of 17,500. The combined typical exposure for this scenario is 2.7×10^{-3} mg/kg, leading to a MOS of 15,556.

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 the reasonable worst case and typical dermal and inhalation exposures.

Regarding scenario 4, the production of foam granules and rebonded foam, the body burden for reasonable worst-case inhalation exposure is 6.6×10^{-4} mg/kg, resulting in a MOS value of 63,636. The body burden for reasonable worst-case dermal exposure is 4.1×10^{-3} mg/kg, leading to a MOS of 10,244. The total body burden for reasonable worst-case for this scenario is 4.7×10^{-3} mg/kg, and results in a MOS of 8,936. For the typical exposure, the inhalation body burden is 8.4×10^{-5} mg/kg, leading to a MOS of 500,000. For the typical dermal exposure, the body burden is 1.3×10^{-3} mg/kg, resulting in a MOS of 32,308. The combined body burden for the typical exposure is 1.4×10^{-3} mg/kg, resulting in a MOS of 30,000.

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 the reasonable worst case and typical dermal and inhalation exposures.

For scenario 5, the formulation of systems and manufacture of spray foams, the body burden for reasonable worst-case inhalation exposure is 7.1×10^{-4} mg/kg. When this is compared with the internal body burden of 42 mg/kg, the MOS was 59,155. Regarding dermal exposure, the body burden for reasonable worst-case exposure is 0.15 mg/kg, leading to a MOS of 280. The total body burden for reasonable worst-case for this scenario is also 0.15 mg/kg, and so this also results in a MOS of 280. For the typical exposure, the inhalation body burden is 3.6×10^{-4} mg/kg, which when compared with the internal body burden results in a MOS of 116,667. For the dermal and combined exposure, the body burdens are 6.9×10^{-2} mg/kg, leading to a MOS of 609 for both.

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 the reasonable worst case and typical dermal and inhalation exposures.

Regarding scenario 6, the use of spray foams, the body burden for reasonable worst-case inhalation exposure is 2.7×10^{-2} mg/kg. When this is compared with the internal body burden the MOS is 1,556. For the dermal exposure, the body burden for reasonable worst-case is 0.32 mg/kg, giving a MOS of 131. The combined body burden is 0.35 mg/kg, resulting in a MOS of 120. The body burden for the typical inhalation exposure is 3.6×10^{-3} mg/kg, leading to a MOS of 11,667. The dermal and combined typical exposure body burdens for this scenario are both 0.17 mg/kg, which give a MOS of 247 for both.

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 7, the manufacture of rigid PUR foam, the inhalation body burden for reasonable worst-case exposure is 2.1×10^{-2} mg/kg. When this is compared with the internal body burden, the resulting MOS value is 2,000. Regarding dermal exposure, the body burden for reasonable worst-case exposure is 4.5×10^{-2} mg/kg, leading to a MOS of 933. The total reasonable worst case body burden for this scenario is 6.6×10^{-2} mg/kg, and results in a MOS of 636. For the typical inhalation exposure, the body burden is 2.9×10^{-3} mg/kg. This gives a MOS of 14,483. The dermal exposure body burden for the typical exposure is 2.2×10^{-2} mg/kg, giving a MOS of 1,909. For the combined exposure, body burden is 2.5×10^{-2} mg/kg, leading to a MOS of 1680.

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 the reasonable worst case and typical dermal and inhalation exposures.

For scenario 8, the use of rigid PUR foam, with respect to inhalation exposure, the body burden for reasonable worst-case exposure is 5.9 x 10^{-4} mg/kg. When this is compared with the internal body burden of 42 mg/kg, the MOS value is 71,186. The body burden for reasonable worst-case dermal exposure is 1.6 x 10^{-2} mg/kg, leading to a MOS of 2,625. The reasonable worst case combined body burden for this scenario is also 1.6 x 10^{-2} mg/kg, and so this also results in a MOS of 2,625. For the typical exposure, the body burden for the inhalation exposure is 2.7 x 10^{-4} mg/kg, leading to a MOS of 155,556. The dermal body burden is 7.2 x 10^{-3} mg/kg, leading to a MOS of 5,833. The combined body burden is 7.5 x 10^{-3} mg/kg, giving a MOS of 5,600.

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 the reasonable worst case and typical dermal and inhalation exposures.

Regarding scenario 9, the manufacture of one-component foams, for the reasonable worst case inhalation exposure, the body burden is 1.8 x 10^{-3} mg/kg. Comparing this value to the internal body burden, results in a MOS of 23,333. The body burden for reasonable worst-case dermal exposure is 3.6 x 10^{-3} mg/kg, leading to a MOS of 11,667. The combined body burden is 5.4 x 10^{-3} mg/kg and so results in a MOS of 7,778. For the typical exposure, the inhalation body burden is 9.6 x 10^{-4} mg/kg, which results in a MOS of 43,750. The typical dermal body burden for this scenario is 6.9 x 10^{-4} mg/kg. This gives a MOS of 60,870. The combined body burden for the typical exposure is 1.7 x 10^{-3} mg/kg, leading to a MOS of 24,706.

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 the reasonable worst case and typical dermal and inhalation exposures.

Regarding scenario 10, the use of one-component foams, for the reasonable worst-case inhalation exposure, the body burden is 7 x 10^{-7} mg/kg, resulting in a MOS of >1,000,000. The body burden for reasonable worst-case dermal exposure is 2.6 x 10^{-3} mg/kg, leading to a MOS of 16,154. The combined body burden is also 2.6 x 10^{-3} mg/kg and so also results in a MOS of 16,154. The typical inhalation body burden for this scenario is 3 x 10^{-7} mg/kg, resulting in a MOS of > 1,000,000. The typical dermal and combined body burdens are 1.3 x 10^{-3} mg/kg, both resulting in a MOS of 32,308.

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 the reasonable worst case and typical dermal and inhalation exposures.

Tables 4.63 and **4.64** summarise the MOSs and conclusions for carcinogenicity for worst case and typical exposure, respectively.

Table 4.63 MOS values and conclusions for carcinogenicity of TCPP – 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 TCPP	3.5×10^{-3}	12,000	(ii)	0.69	61	(ii)	0.69	61	(ii)
2.Manufacture of flexible PUR foam	7.3×10^{-4}	57,534	(ii)	9.7×10^{-2}	433	(ii)	9.8×10^{-2}	429	(ii)
3.Cutting of flexible PUR foam	5.9×10^{-4}	71,186	(ii)	1.7×10^{-2}	2,471	(ii)	1.8×10^{-2}	2,333	(ii)
4.Production of foam granules & rebonded foam	6.6×10^{-4}	63,636	(ii)	4.1×10^{-3}	10,244	(ii)	4.7×10^{-3}	8,936	(ii)
5.Formulation of systems and mfgr of spray foams	7.1×10^{-4}	59,155	(ii)	0.15	280	(ii)	0.15	280	(ii)
6.Use of spray foams	2.7×10^{-2}	1,556	(ii)	0.32	131	(ii)	0.35	120	(ii)
7.Manufacture of rigid PUR foam	2.1×10^{-2}	2,000	(ii)	4.5×10^{-2}	933	(ii)	6.6×10^{-2}	636	(ii)
8.Use of rigid foam	5.9×10^{-4}	71,186	(ii)	1.6×10^{-2}	2,625	(ii)	1.6×10^{-2}	2,625	(ii)
9.Production of 1-K foams	1.8×10^{-3}	23,333	(ii)	3.6×10^{-3}	11,667	(ii)	5.4×10^{-3}	7,778	(ii)
10.Use of 1-K foams	7×10^{-7}	>1,000,000	(ii)	2.6×10^{-3}	16,154	(ii)	2.6×10^{-3}	16,154	(ii)

Table 4.64 MOS values and conclusions for carcinogenicity of TCPP – 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 TCPP	1.8×10^{-3}	23,333	(ii)	6.9×10^{-2}	609	(ii)	7.1×10^{-2}	592	(ii)
2.Manufacture of flexible PUR foam	8.9×10^{-5}	471,910	(ii)	2.8×10^{-3}	15,000	(ii)	2.9×10^{-3}	14,483	(ii)
3.Cutting of flexible PUR foam	2.7×10^{-4}	155,556	(ii)	2.4×10^{-3}	17,500	(ii)	2.7×10^{-3}	15,556	(ii)
4.Production of foam granules & rebonded foam	8.4×10^{-5}	500,000	(ii)	1.3×10^{-3}	32,308	(ii)	1.4×10^{-3}	30,000	(ii)
5.Formulation of systems and mfgr of spray foams	3.6×10^{-4}	116,667	(ii)	6.9×10^{-2}	609	(ii)	6.9×10^{-2}	609	(ii)
6.Use of spray foams	3.6×10^{-3}	11,667	(ii)	0.17	247	(ii)	0.17	247	(ii)
7.Manufacture of rigid PUR foam	2.9×10^{-3}	14,483	(ii)	2.2×10^{-2}	1,909	(ii)	2.5×10^{-2}	1,680	(ii)
8.Use of rigid foam	2.7×10^{-4}	155,556	(ii)	7.2×10^{-3}	5,833	(ii)	7.5×10^{-3}	5,600	(ii)
9.Production of 1-K foams	9.6×10^{-4}	43,750	(ii)	6.9×10^{-4}	60,870	(ii)	1.7×10^{-3}	24,706	(ii)
10.Use of 1-K foams	3×10^{-7}	>1,000,000	(ii)	1.3×10^{-3}	32,308	(ii)	1.3×10^{-3}	32,308	(ii)

4.1.3.2.7 Toxicity for reproduction

Effects on fertility

In a two-generation oral reproductive toxicity study in rats with TCPP, a LOAEL of 99 mg/kg is derived for effects on fertility. This is based on a decrease in relative uterus weight seen in all dosed females in F0 and the high dose females in F1. Assuming 80% absorption by the oral route, this leads to an internal body burden of 79 mg/kg.

In line with the draft TGD (2005), the minimal MOS for effects on fertility is 150. 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 5. A factor of 3 to account for the use of a LOAEL rather than a NOAEL is also employed. Although the effects seen at the low dose were slight, they did reach statistical significance and were considered to be biologically significant as they followed a dose dependent trend.

For scenario 1, manufacture of TCPP, with respect to inhalation exposure, the body burden for the reasonable worst-case exposure is 3.5 x 10^{-3} mg/kg. When this is compared with the internal body burden for fertility of 79 mg/kg, the MOS is 22,571. With respect to dermal exposure, the body burden for the reasonable worst-case exposure is 0.69 mg/kg, leading to a MOS of 114. The total body burden for the reasonable worst case for this scenario is also 0.69 mg/kg, again leading to a MOS of 114. The body burden for the typical inhalation exposure is 1.8 x 10^{-3} mg/kg, leading to a MOS of 43,889. For the typical dermal exposure, the body burden is 6.9 x 10^{-2} mg/kg, which results in a MOS of 1,145. The combined body burden for the typical exposure is 7.1 x 10^{-2} mg/kg, leading to a MOS of 1,113.

When the MOSs are compared with the minimal MOS of 150, there is a concern for the reasonable worst case dermal 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.

Regarding scenario 2, the manufacture of flexible PUR foam, the body burden for the reasonable worst-case inhalation exposure is 7.3 x 10^{-4} mg/kg. When this is compared with the internal body burden of 79 mg/kg, it results in a MOS of 108,219. The body burden for the reasonable worst-case dermal exposure is 9.7 x 10^{-2} mg/kg. This gives a MOS of 814. The combined body burden for the reasonable worst-case exposure is 9.8 x 10^{-2} mg/kg, leading to a MOS of 806. The body burden for the typical inhalation exposure is 8.9 x 10^{-5} mg/kg, leading to a MOS of 887,640. The body burden for the typical dermal exposure for this scenario is 2.8 x 10^{-3} mg/kg, leading to a MOS of 28,214. The typical combined body burden is 2.9 x 10^{-3}, leading to a MOS of 27,241.

When the MOSs are compared with the minimal MOS of 150, there is no concern for this scenario. Therefore, **conclusion (ii)** is drawn for reasonable worst case and typical exposures.

For scenario 3, cutting of flexible foam, the body burden for the reasonable worst-case inhalation exposure is 5.9 x 10^{-4} mg/kg, which when compared the internal body burden of 79 mg/kg gives a MOS of 133,898. For the reasonable worst-case dermal exposure, the body burden is 1.7 x 10^{-2} mg/kg, leading to a MOS of 4,647. The combined body burden for the reasonable worst-case exposure is 1.8 x 10^{-2} mg/kg, resulting in a MOS of 4,389. The typical inhalation body burden is 2.7 x 10^{-4} mg/kg, which gives a MOS of 292,593. The typical dermal body burden is 2.4 x 10^{-3} mg/kg, resulting in a MOS of 32,917. The combined typical exposure is 2.7 x 10^{-3} mg/kg, leading to a MOS of 29,259.

When the MOSs are compared with the minimal MOS of 150, there is no concern for this scenario. Therefore, **conclusion (ii)** is drawn for this scenario.

Regarding scenario 4, the production of foam granules and rebonded foam, the inhalation body burden for reasonable worst-case exposure is 6.6 x 10^{-4} mg/kg, resulting in a MOS value of 119,697. For dermal exposure for this exposure scenario, the body burden for reasonable worst-case exposure is 4.1 x 10^{-3} mg/kg, and when compared with the internal body burden of 79 mg/kg, results in a MOS of 19,268. The total body burden for reasonable worst-case for this scenario is 4.7 x 10^{-3} mg/kg, and so results in a MOS of 16,809. For the typical exposure, the inhalation body burden is 8.4 x 10^{-5} mg/kg, leading to a MOS of 940,476. The body burden for the typical dermal exposure is 1.3 x 10^{-3} mg/kg, leading to a MOS of 60,769. The combined body burden for the typical exposure is 1.4 x 10^{-3} mg/kg, resulting in a MOS of 56,429.

When the MOSs are compared with the minimal MOS of 150, there is no concern for this scenario. Therefore, **conclusion (ii)** is drawn for this scenario.

For scenario 5, the formulation of systems and manufacture of spray foams, with respect to inhalation exposure, the body burden for the reasonable worst-case exposure is 7.1 x 10^{-4} mg/kg. When this is compared with the internal body burden of 79 mg/kg, the MOS is 111,268. The body burden for the reasonable worst-case dermal exposure is 0.15 mg/kg, leading to a MOS of 527. The total reasonable worst case body burden is also 0.15 mg/kg, and so this also results in a MOS of 527. The body burden for the typical inhalation exposure is 3.6 x 10^{-4} mg/kg, which when compared with the internal body burden results in a MOS of 219,444. For the dermal and combined exposure, the body burden is 6.9 x 10^{-2} mg/kg, leading to a MOS of 1,145 for both.

When the MOSs are compared with the minimal MOS of 150, there is no concern for this scenario and so **conclusion (ii)** is drawn.

Regarding scenario 6, the use of spray foams, the body burden for the reasonable worst-case inhalation exposure is 2.7 x 10^{-2} mg/kg, which when compared with the internal body burden leads to a MOS of 2,926. The body burden for the reasonable worst-case dermal exposure is 0.32 mg/kg, giving a MOS of 247. The combined body burden for the reasonable worst case exposure is 0.35 mg/kg and results in a MOS of 226. The body burden for the typical inhalation exposure is 3.6 x 10^{-3} mg/kg, leading to a MOS of 21,944. The dermal and combined typical exposure body burdens for this scenario are both 0.17 mg/kg, which give a MOS of 465.

When the MOSs are compared with the minimal MOS of 150, there is no concern for this scenario and so **conclusion (ii)** is drawn.

For scenario 7, the manufacture of rigid PUR foam, for the reasonable worst case exposure, the inhalation body burden is 2.1 x 10^{-2} mg/kg. When this is compared with the internal body burden, the MOS is 3,762. Regarding dermal exposure, the body burden is 4.5 x 10^{-2} mg/kg, leading to a MOS of 1,756. The total reasonable worst case body burden for this scenario is 6.6 x 10^{-2} mg/kg, and results in a MOS of 1,197. For the typical inhalation exposure, the body burden is 2.9 x 10^{-3} mg/kg, leading to a MOS of 27,241. The body burden for the typical dermal exposure is 2.2 x 10^{-2} mg/kg, giving a MOS of 3,591. For the combined exposure, body burden is 2.5 x 10^{-2} mg/kg, leading to a MOS of 3,160.

When the MOSs are compared with the minimal MOS of 150, there is no concern for this scenario. Therefore, **conclusion (ii)** is drawn for reasonable worst case and typical exposures.

For scenario 8, the use of rigid PUR foam, with respect to inhalation exposure, the body burden for the reasonable worst-case exposure is 5.9 x 10^{-4} mg/kg. When this is compared with the internal body burden of 79 mg/kg, the MOS is 133,898. Regarding dermal exposure, the reasonable worst case body burden is 1.6 x 10^{-2} mg/kg, leading to a MOS of 4,938. The total reasonable worst case body burden for this scenario is also 1.6 x 10^{-2} mg/kg, and so this also results in a MOS of 4,938. For the typical inhalation exposure, the body burden is 2.7 x 10^{-4} mg/kg, leading to a MOS of 292,593. The dermal typical body burden is 7.2 x 10^{-3} mg/kg, leading to a MOS of 10,972. The combined body burden is 7.5 x 10^{-3} mg/kg, giving a MOS of 10,533.

When all of the MOSs are compared with the minimal MOS of 150, there is no concern for this scenario. Therefore, **conclusion (ii)** is drawn for this scenario.

For scenario 9, the manufacture of one-component foams, the body burden for the reasonable worst-case inhalation exposure is 1.8 x 10^{-3} mg/kg, and when this is compared to the internal body burden of 79 mg/kg, results in a MOS of 43,889. The body burden for the reasonable worst-case dermal exposure is 3.6 x 10^{-3} mg/kg, giving a MOS of 21,944. The combined body burden for this scenario is 5.4 x 10^{-3} mg/kg, resulting in a MOS of 14,630. For the typical exposure, the inhalation body burden is 9.6 x 10^{-4} mg/kg, giving a MOS of 82,292. The typical dermal exposure body burden is 6.9 x 10^{-4} mg/kg. This gives a MOS of 114,493. The combined body burden for the typical exposure is 1.7 x 10^{-3} mg/kg, leading to a MOS of 46,471.

When the MOSs are compared with the minimal MOS of 150, there is no concern for this scenario, and so a **conclusion (ii)** can be drawn.

Regarding scenario 10, the use of one-component foams, the body burden for the reasonable worst-case inhalation exposure is 7 x 10^{-7} mg/kg, resulting in a MOS of >1,000,000. The body burden for the reasonable worst-case dermal exposure is 2.6 x 10^{-3} mg/kg, giving a MOS of 30,385. The combined body burden is also 2.6 x 10^{-3} mg/kg and so also results in a MOS of 30,385. The typical inhalation exposure body burden is 3 x 10^{-7} mg/kg, resulting in a MOS of >1,000,000. The typical dermal and combined body burdens are 1.3 x 10^{-3} mg/kg, both resulting in a MOS of 60,769.

When the MOSs are compared with the minimal MOS of 150, there is no concern for this scenario, and so a **conclusion (ii)** can be drawn.

Tables 4.65 and **4.66** summarise the MOSs and conclusions for fertility for the reasonable worst case and typical exposures, respectively.

Table 4.65 MOS values and conclusions for effects on fertility for TCPP – 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 TCPP	3.5×10^{-3}	22,571	(ii)	0.69	114	**(iii)**	0.69	114	**(iii)**
2.Manufacture of flexible PUR foam	7.3×10^{-4}	108,219	(ii)	9.7×10^{-2}	814	(ii)	9.8×10^{-2}	806	(ii)
3.Cutting of flexible PUR foam	5.9×10^{-4}	133,898	(ii)	1.7×10^{-2}	4,647	(ii)	1.8×10^{-2}	4,389	(ii)
4.Production of foam granules & rebonded foam	6.6×10^{-4}	119,697	(ii)	4.1×10^{-3}	19,268	(ii)	4.7×10^{-3}	16,809	(ii)
5.Formulation of systems and mfgr of spray foams	7.1×10^{-4}	111,268	(ii)	0.15	527	(ii)	0.15	527	(ii)
6.Use of spray foams	2.7×10^{-2}	2,926	(ii)	0.32	247	(ii)	0.35	226	(ii)
7.Manufacture of rigid PUR foam	2.1×10^{-2}	3,762	(ii)	4.5×10^{-2}	1,756	(ii)	6.6×10^{-2}	1,197	(ii)
8.Use of rigid foam	5.9×10^{-4}	133,898	(ii)	1.6×10^{-2}	4,938	(ii)	1.6×10^{-2}	4,938	(ii)
9.Production of 1-K foams	1.8×10^{-3}	43,889	(ii)	3.6×10^{-3}	21,944	(ii)	5.4×10^{-3}	14,630	(ii)
10.Use of 1-K foams	7×10^{-7}	>1,000,000	(ii)	2.6×10^{-3}	30,385	(ii)	2.6×10^{-3}	30,385	(ii)

Table 4.66 MOS values and conclusions for effects on fertility for TCPP – 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 TCPP	1.8×10^{-3}	43,889	(ii)	6.9×10^{-2}	1,145	(ii)	7.1×10^{-2}	1,113	(ii)
2.Manufacture of flexible PUR foam	8.9×10^{-5}	887,640	(ii)	2.8×10^{-3}	28,214	(ii)	2.9×10^{-3}	27,241	(ii)
3.Cutting of flexible PUR foam	2.7×10^{-4}	292,593	(ii)	2.4×10^{-3}	32,917	(ii)	2.7×10^{-3}	29,259	(ii)
4.Production of foam granules & rebonded foam	8.4×10^{-5}	940,476	(ii)	1.3×10^{-3}	60,769	(ii)	1.4×10^{-3}	56,429	(ii)
5.Formulation of systems and mfgr of spray foams	3.6×10^{-4}	219,444	(ii)	6.9×10^{-2}	1,145	(ii)	6.9×10^{-2}	1,145	(ii)
6.Use of spray foams	3.6×10^{-3}	21,944	(ii)	0.17	465	(ii)	0.17	465	(ii)
7.Manufacture of rigid PUR foam	2.9×10^{-3}	27,241	(ii)	2.2×10^{-2}	3,591	(ii)	2.5×10^{-2}	3,160	(ii)
8.Use of rigid foam	2.7×10^{-4}	292,593	(ii)	7.2×10^{-3}	10,972	(ii)	7.5×10^{-3}	10,533	(ii)
9.Production of 1-K foams	9.6×10^{-4}	82,292	(ii)	6.9×10^{-4}	114,493	(ii)	1.7×10^{-3}	46,471	(ii)
10.Use of 1-K foams	3×10^{-7}	>1,000.000	(ii)	1.3×10^{-3}	60,769	(ii)	1.3×10^{-3}	60,769	(ii)

Developmental toxicity

In a two-generation oral reproductive toxicity study in rats with TCPP, a LOAEL of 99 mg/kg is derived for developmental toxicity. This is based on a treatment related effect on the number of runts observed in all TCPP-treated groups of the F0 generation. Assuming 80% absorption by the oral route, this leads to an internal body burden of 79 mg/kg.

In line with the draft TGD (2005), the minimal MOS for developmental 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) and an intraspecies factor of 5. A factor of 3 to account for the use of a LOAEL rather than a NOAEL is also used.

For scenario 1, manufacture of TCPP, with respect to inhalation exposure, the body burden for the reasonable worst-case exposure is 3.5×10^{-3} mg/kg. When this is compared with the internal body burden of 79 mg/kg, the MOS is 22,571. For the dermal and combined

exposures, the body burden is 0.69 mg/kg, leading to a MOS of 114 in both cases. For the typical inhalation exposure, the body burden is 1.8 x 10^{-3} mg/kg, leading to a MOS of 43,889. The body burden for the typical dermal exposure is 6.9 x 10^{-2} mg/kg, which results in a MOS of 1,145. The combined body burden for the typical exposure is 7.1 x 10^{-2} mg/kg, leading to a MOS of 1,113.

When the MOSs are compared with the minimal MOS of 150, there is a concern for the reasonable worst case dermal 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.

Regarding scenario 2, the manufacture of flexible PUR foam, the body burden for the reasonable worst-case inhalation exposure is 7.3 x 10^{-4} mg/kg, which when compared with the internal body burden of 79 mg/kg, results in a MOS of 108,219. The body burden for the reasonable worst-case dermal exposure is 9.7 x 10^{-2} mg/kg, leading to a MOS of 814. The combined body burden for the reasonable worst-case exposure is 9.8 x 10^{-2} mg/kg, leading to a MOS of 806. For the typical inhalation exposure, the body burden is 8.9 x 10^{-5} mg/kg, leading to a MOS of 887,640. The body burden for the typical dermal exposure is 2.8 x 10^{-3} mg/kg, leading to a MOS of 28,214. The combined body burden is 2.9 x 10^{-3} mg/kg. Leading to a MOS of 27,241.

When the MOSs are compared with the minimal MOS of 150, there is no concern for this scenario. Therefore, **conclusion (ii)** is drawn for reasonable worst case and typical exposures.

For scenario 3, cutting of flexible foam, the body burden for the reasonable worst-case inhalation exposure is 5.9 x 10^{-4} mg/kg. When this is compared with the internal body burden of 79 mg/kg, a MOS of 133,898 is derived. For the reasonable worst-case dermal exposure, the body burden is 1.7 x 10^{-2} mg/kg, leading to a MOS of 4,647. The combined body burden for the reasonable worst-case exposure is 1.8 x 10^{-2} mg/kg, resulting in a MOS of 4,389. The typical inhalation body burden is 2.7 x 10^{-4} mg/kg, which gives a MOS of 292,593. The typical dermal body burden is 2.4 x 10^{-3} mg/kg, resulting in a MOS of 32,917. The combined typical exposure is 2.7 x 10^{-3} mg/kg, leading to a MOS of 29,259.

When the MOSs are compared with the minimal MOS of 150, there is no concern for this scenario. Therefore, **conclusion (ii)** is drawn for this scenario.

Regarding scenario 4, the production of foam granules and rebonded foam, the inhalation body burden for the reasonable worst-case exposure is 6.6 x 10^{-4} mg/kg. This results in a MOS value of 119,697. The body burden for the reasonable worst-case dermal exposure is 4.1 x 10^{-3} mg/kg, resulting in a MOS of 19,268. The total body burden for reasonable worst-case is 4.7 x 10^{-3} mg/kg, and so results in a MOS of 16,809. For the typical exposure, the inhalation and dermal body burdens are 8.4 x 10^{-5} mg/kg and 1.3 x 10^{-3} mg/kg, leading to MOS of 940,476 and 60,769, respectively. The combined body burden for the typical exposure is 1.4 x 10^{-3} mg/kg, resulting in a MOS of 56,429.

When the MOSs are compared with the minimal MOS of 150, there is no concern for this scenario. Therefore, **conclusion (ii)** is drawn for this scenario.

For scenario 5, the formulation of systems and manufacture of spray foams, the body burden for the reasonable worst-case inhalation exposure is 7.1 x 10^{-4} mg/kg. When this was compared with the internal body burden, the MOS is 111,268. The dermal body burden for

the reasonable worst-case exposure is 0.15 mg/kg, leading to a MOS of 527. The reasonable worst case combined body burden is also 0.15 mg/kg, and so this also results in a MOS of 527. The body burden for the typical inhalation exposure is 3.6×10^{-4} mg/kg, leading to a MOS of 219,444. For the typical dermal and combined exposures, the body burdens are 6.9×10^{-2} mg/kg, leading to a MOS of 1,145 for both.

When the MOSs are compared with the minimal MOS of 150, there is no concern for this scenario and so **conclusion (ii)** is drawn.

Regarding scenario 6, the use of spray foams, the body burden for the reasonable worst-case inhalation exposure is 2.7×10^{-2} mg/kg. When this is compared with the internal body burden of 79 mg/kg, the MOS is 2,926. The body burden for the reasonable worst-case dermal exposure is 0.32 mg/kg, giving a MOS of 247. The combined body burden for the reasonable worst case exposure is 0.35 mg/kg and resulting in a MOS of 226. For the typical exposure, the body burden for the inhalation exposure is 3.6×10^{-3} mg/kg, leading to a MOS of 21,944. The dermal and combined body burdens are both 0.17 mg/kg, which gives a MOS of 465 for both.

When the MOSs are compared with the minimal MOS of 150, there is no concern for this scenario and so **conclusion (ii)** is drawn.

For scenario 7, the manufacture of rigid PUR foam, the body burden for the reasonable worst-case inhalation exposure is 2.1×10^{-2} mg/kg and when compared with the internal body burden, results in a MOS of 3,762. The body burden for the reasonable worst-case dermal exposure is 4.5×10^{-2} mg/kg, leading to a MOS of 1,756. The total body burden for this scenario is 6.6×10^{-2} mg/kg, resulting in a MOS of 1,197. For the typical exposure, the inhalation body burden is 2.9×10^{-3} mg/kg, leading to a MOS of 27,241. The dermal body burden is 2.2×10^{-2} mg/kg, giving a MOS of 3,591. For the combined exposure, body burden is 2.5×10^{-2} mg/kg, leading to a MOS of 3,160.

When the MOSs are compared with the minimal MOS of 150, there is no concern for this scenario. Therefore, **conclusion (ii)** is drawn for reasonable worst case and typical exposures.

For scenario 8, the use of rigid PUR foam, with respect to the reasonable worst case exposures, the inhalation body burden is 5.9×10^{-4} mg/kg. When this is compared with the internal body burden of 79 mg/kg, the MOS is 133,898. The dermal and combined body burdens are 1.6×10^{-2} mg/kg, leading to a MOS of 4,938 for both. For the typical inhalation exposure, the body burden is 2.7×10^{-4} mg/kg, leading to a MOS of 292,593. The typical dermal body burden is 7.2×10^{-3} mg/kg, leading to a MOS of 10,972. The combined body burden is 7.5×10^{-3} mg/kg, giving a MOS of 10,533.

When all of the MOSs are compared with the minimal MOS of 150, there is no concern for this scenario. Therefore, **conclusion (ii)** is drawn for this scenario.

For scenario 9, the manufacture of one-component foams, the body burden for the reasonable worst-case inhalation exposure is 1.8×10^{-3} mg/kg, leading to a MOS of 43,889. The body burden for the reasonable worst-case dermal exposure is 3.6×10^{-3} mg/kg, giving a MOS of 21,944. The combined body burden is 5.4×10^{-3} mg/kg, resulting in a MOS of 14,630. For the typical exposure, the inhalation body burden is 9.6×10^{-4} mg/kg, giving a MOS of 82,292. The dermal body burden is 6.9×10^{-4} mg/kg, leading to a MOS of 114,493. The combined body burden for the typical exposure is 1.7×10^{-3} mg/kg, leading to a MOS of 46,471.

When the MOSs are compared with the minimal MOS of 150, there is no concern for this scenario, and so a **conclusion (ii)** can be drawn.

Regarding scenario 10, the use of one-component foams, with respect to the reasonable worst case exposures, the inhalation body burden is 7 x 10^{-7} mg/kg, resulting in a MOS of >1,000,000. The dermal body burden is 2.6 x 10^{-3} mg/kg, giving a MOS of 30,385. The combined body burden is also 2.6 x 10^{-3} mg/kg and so also results in a MOS of 30,385. For the typical exposures, inhalation body burden is 3 x 10^{-7} mg/kg, resulting in a MOS of >1,000,000. The dermal and combined body burdens are 1.3 x 10^{-3} mg/kg, both resulting in a MOS of 60,769.

When the MOSs are compared with the minimal MOS of 150, there is no concern for this scenario, and so a **conclusion (ii)** can be drawn.

Tables 4.67 and **4.68** summarise the MOSs and conclusions for fertility for the reasonable worst case and typical exposures, respectively.

Table 4.67 MOS values and conclusions for developmental toxicity for TCPP – 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 TCPP	3.5 x 10^{-3}	22,571	(ii)	0.69	114	**(iii)**	0.69	114	**(iii)**
2.Manufacture of flexible PUR foam	7.3 x 10^{-4}	108,219	(ii)	9.7 x 10^{-2}	814	(ii)	9.8 x 10^{-2}	806	(ii)
3.Cutting of flexible PUR foam	5.9 x 10^{-4}	133,898	(ii)	1.7 x 10^{-2}	4,647	(ii)	1.8 x 10^{-2}	4,389	(ii)
4.Production of foam granules & rebonded foam	6.6 x 10^{-4}	119,697	(ii)	4.1 x 10^{-3}	19,268	(ii)	4.7 x 10^{-3}	16,809	(ii)
5.Formulation of systems and mfgr of spray foams	7.1 x 10^{-4}	111,268	(ii)	0.15	527	(ii)	0.15	527	(ii)
6.Use of spray foams	2.7 x 10^{-2}	2,926	(ii)	0.32	247	(ii)	0.35	226	(ii)
7.Manufacture of rigid PUR foam	2.1 x 10^{-2}	3,762	(ii)	4.5 x 10^{-2}	1,756	(ii)	6.6 x 10^{-2}	1,197	(ii)
8.Use of rigid foam	5.9 x 10^{-4}	133,898	(ii)	1.6 x 10^{-2}	4,938	(ii)	1.6 x 10^{-2}	4,938	(ii)
9.Production of 1-K foams	1.8 x 10^{-3}	43,889	(ii)	3.6 x 10^{-3}	21,944	(ii)	5.4 x 10^{-3}	14,630	(ii)
10.Use of 1-K foams	7 x 10^{-7}	>1,000,000	(ii)	2.6 x 10^{-3}	30,385	(ii)	2.6 x 10^{-3}	30,385	(ii)

Table 4.68 MOS values and conclusions for developmental toxicity for TCPP – 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 TCPP	1.8×10^{-3}	43,889	(ii)	6.9×10^{-2}	1,145	(ii)	7.1×10^{-2}	1,113	(ii)
2.Manufacture of flexible PUR foam	8.9×10^{-5}	887,640	(ii)	2.8×10^{-3}	28,214	(ii)	2.9×10^{-3}	27,241	(ii)
3.Cutting of flexible PUR foam	2.7×10^{-4}	292,593	(ii)	2.4×10^{-3}	32,917	(ii)	2.7×10^{-3}	29,259	(ii)
4.Production of foam granules & rebonded foam	8.4×10^{-5}	940,476	(ii)	1.3×10^{-3}	60,769	(ii)	1.4×10^{-3}	56,429	(ii)
5.Formulation of systems and mfgr of spray foams	3.6×10^{-4}	219,444	(ii)	6.9×10^{-2}	1,145	(ii)	6.9×10^{-2}	1,145	(ii)
6.Use of spray foams	3.6×10^{-3}	29,944	(ii)	0.17	465	(ii)	0.17	465	(ii)
7.Manufacture of rigid PUR foam	2.9×10^{-3}	27,241	(ii)	2.2×10^{-2}	3,591	(ii)	2.5×10^{-2}	3,160	(ii)
8.Use of rigid foam	2.7×10^{-4}	292,593	(ii)	7.2×10^{-3}	10,972	(ii)	7.5×10^{-3}	10,533	(ii)
9.Production of 1-K foams	9.6×10^{-4}	82,292	(ii)	6.9×10^{-4}	114,493	(ii)	1.7×10^{-3}	46,471	(ii)
10.Use of 1-K foams	3×10^{-7}	>1,000.000	(ii)	1.3×10^{-3}	60,769	(ii)	1.3×10^{-3}	60,769	(ii)

4.1.3.2.8 Summary of risk characterisation for workers

With respect to worker scenario 1 (manufacture of TCPP), the MOS for reasonable worst case dermal exposures for fertility and developmental toxicity are below the minimal MOS and therefore **conclusion (iii)** is drawn. There is no concern for the typical dermal exposures or inhalation exposures for this exposure scenario.

A **conclusion (ii)** is drawn for all other worker exposure scenarios for all other endpoints.

4.1.3.3 Consumers

The current use pattern provided by industry indicates that most of the TCPP produced in the EU is used in the production of polyurethane foam in Europe. Most of the TCPP used in flexible foam is used in upholstery and bedding. 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 expected that consumer exposure to TCPP from these foams is very low.

There are two consumer exposure scenarios from which exposure to TCPP could occur. These are exposure due to release of the substance from TCPP-containing flexible PUR foam and exposure during the use of 1-K foams. Exposure due to release of TCPP in rooms containing closed-cell rigid foam is not considered further here, as consumer exposure is believed to negligible.

For exposure to TCPP 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, 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 filame retardant from foam over years rather than hours seems to indicate that the loss of flame 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.

Regarding exposure due to the use of 1-K foams, a RWC inhalation exposure for a consumer can be estimated as 0.005 mg/m^3 and a typical exposure as 0.0025 mg/m^3. Dermal exposure is estimated (as a worst case scenario, assuming direct contact with the foam) as being 174 μg/cm^2. However, most consumers would not be spraying foam regularly. It is very unlikely that they would spray foam more than once per year, and more probably would use spray once or twice in a lifetime, if at all. Exposure for consumers in this scenario is considered to be negligible over a lifetime, but could be significant in the short-term. Therefore, the only end-point considered in the risk characterisation for this exposure scenario is acute toxicity.

The reasonable worst-case inhalation exposure is 0.005 mg/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 1.4 x 10^{-3} mg/kg. For dermal exposure in this scenario, the reasonable worst-case exposure is 174 μg/cm^2/day. Using default values of a 70 kg person with 420 cm^2 of exposed skin and 23 % absorption, the dermal body burden is 0.24 mg/kg.

Combining the two values gives a calculated total body burden of 0.24 mg/kg for this scenario.

4.1.3.3.1 Acute toxicity

No significant signs of toxicity were seen in experimental animals via the inhalation and dermal routes. With respect to oral exposure, a NOAEL of 200 mg/kg was identified from the acute oral toxicity studies. Assuming 80 % absorption by the oral route, this leads to an internal body burden of 160 mg/kg.

In line with the draft TGD (2005), the minimal MOS for acute toxicity is 100. This mMOS is established 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 only consumer exposure scenario for which the acute toxicity end-point is considered is the use of 1-K foams. For that scenario, with respect to inhalation exposure, the body burden for reasonable worst-case exposure was 1.4×10^{-3} mg/kg. This gives a MOS value of 114,286. With respect to dermal exposure, the body burden for reasonable worst-case exposure was 0.24 mg/kg. This gives a MOS of 667.

When compared to the minimal MOS of 100, it is concluded that the MOSs are sufficient and there are no concerns for acute toxicity to consumers for this scenario and so **conclusion (ii)** is drawn.

4.1.3.3.2 Irritation and corrosivity

TCPP 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.

4.1.3.3.3 Repeated dose toxicity

In relation to repeated dose toxicity, a LOAEL of 52 mg/kg/day was derived from a 13-week study in which male and female rats were dosed with TCPP at concentrations of up to 1349 mg/kg/day and 1745 mg/kg/day, respectively. This LOAEL was based on increased liver weights observed in male animals. Assuming 80 % absorption by the oral route, this leads to an internal body burden of 42 mg/kg/day.

In line with the draft TGD (2005), the minimal MOS for repeated dose toxicity 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. As discussed in section 4.1.3.3.3, it is proposed that additional factors to take account of the use of a LOAEL rather than a NOAEL and semi-chronic to chronic exposure are not considered necessary.

Regarding potential inhalation exposure to TCPP 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 42,000. 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 TCPP from flexible PUR foam, the reasonable worst-case body burden is taken as 0.0011 mg/kg, leading to a MOS of 38,182.

Given the size of 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/day (assuming a body weight of 9.1 kg) has been taken from the TCEP risk assessment report. When this is compared to the internal body burden of 42 mg/kg taken from the repeated dose toxicity study, then an MOS of 210,000 results. 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).**

4.1.3.3.4 Mutagenicity

As with the worker section above, **conclusion (ii)** is drawn for consumers in relation to mutagenicity.

4.1.3.3.5 Carcinogenicity

As described in section 4.1.2.8, it is proposed that the LOAEL of 52 mg/kg/day, identified from the 90-day study with TCPP will be taken forward for risk characterisation for carcinogenicity. Assuming 80% absorption by the oral route, this leads to an internal body burden of 42 mg/kg/day.

The minimal MOS for carcinogenicity 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. Normally, a factor of 3 would be used to take into account the use of a LOAEL rather than a NOAEL. However, this is not considered necessary here, as the LOAEL derived from the repeat dose toxicity study was based on liver weight changes which is not considered to be particularly toxicologically significant and the LOAEL is probably quite close to the NOAEL.

Regarding potential inhalation exposure to TCPP due to its release from flexible PUR, the estimated body burden for the reasonable worst case exposure was 1 µg/kg. When this is compared with the internal body burden of 42 mg/kg, the MOS is 42,000. It is concluded that this MOS is sufficient and there are no concerns for carcinogenicity to consumers for this scenario and so **conclusion (ii)** is drawn.

Regarding potential dermal exposure due to the release of TCPP from flexible PUR foam, the reasonable worst-case body burden is taken as 0.0011 mg/kg, leading to a MOS of 38,182. 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/day (assuming a body weight of 9.1 kg) has been taken from the TCEP risk assessment report. When this is compared to the internal body burden the MOS is 210,000. 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).**

4.1.3.3.6 Toxicity for reproduction

Effects on fertility

In a two-generation oral reproductive toxicity study in rats with TCPP, a LOAEL of 99 mg/kg is derived for effects on fertility. This is based on a decrease in relative uterus weight seen in all dosed females in F0 and the high dose females in F1. Assuming 80% absorption by the oral route, this leads to an internal body burden of 79 mg/kg.

In line with the draft TGD (2005), the minimal MOS for effects on fertility is 300. 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. A factor of 3 to account for the use of a LOAEL rather than a NOAEL is also employed.

Regarding potential inhalation exposure to TCPP due to its release from flexible PUR foam, the body burden for the reasonable worst case exposure was 1 µg/kg. This gives a MOS of 79,000. It is concluded that this MOS is sufficient and there are no concerns for effects on fertility for consumers for this scenario and so **conclusion (ii)** is drawn.

Regarding potential dermal exposure due to the release of TCPP from flexible PUR foam, the reasonable worst-case body burden is 0.0011 mg/kg, resulting in a MOS of 71,818. When this is compared with the minimal MOS it is concluded that there is a sufficient margin of safety and a **conclusion (ii)** is drawn.

For children, the oral route of exposure is also considered. A RWC exposure for oral ingestion of 0.2 µg/kg/day (assuming a body weight of 9.1 kg) has been taken from the TCEP risk assessment report. When this is compared to the internal body burden of 79 mg/kg, the MOS is 395,000. It is considered that this MOS is sufficient and so there is no concern for exposure of children via the oral route and **conclusion (ii**) is drawn.

Developmental toxicity

In a two-generation reproductive toxicity study with TCPP, a LOAEL of 99 mg/kg is derived for developmental toxicity. This is based on a treatment related effect on the number of runts observed in all TCPP-fed groups in the F0 generation. Assuming 80% absorption by the oral route, the internal body burden is 79 mg/kg.

In line with the draft TGD (2005), the minimal MOS for developmental 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) and an intraspecies factor of 10. As described in section 4.1.3.2.7, a factor of 3 to account for the use of a LOAEL rather than an NOAEL is also employed.

Regarding potential inhalation exposure to TCPP due to its release from flexible PUR foam, the body burden for the reasonable worst case exposure was 1 µg/kg, resulting in a MOS of 79,000. It is considered that this MOS is sufficient and therefore **conclusion (ii)** is drawn.

Regarding potential dermal exposure due to the release of TCPP from flexible PUR foam, the reasonable worst case body burden is 0.0011 mg/kg, giving a MOS of 71,818. It is concluded that there is no concern for dermal exposure to consumers and **conclusion (ii)** is drawn.

For children, the oral route of exposure is also considered. A reasonable worst case exposure for oral ingestion of 0.2 µg/kg/day (assuming a body weight of 9.1 kg) has been taken from

the TCEP risk assessment report. When this figure is compared with the internal body burden of 79 mg/kg, the MOS is 395,000 and **conclusion (ii)** is drawn.

4.1.3.3.7 Summary of risk characterisation for consumers

Conclusion (ii) is drawn for consumers for all exposure scenarios. This conclusion applies to all endpoints.

4.1.3.4 Humans exposed via the environment

4.1.3.4.1 Regional exposure

Repeated dose toxicity

In relation to repeated dose toxicity, a LOAEL of 52 mg/kg/day was derived from a 13-week study in which male and female rats were dosed with TCPP at concentrations of up to 1349 mg/kg/day and 1745 mg/kg/day, respectively. This LOAEL was based on increased liver weights observed in male animals. Assuming 80% absorption by the oral route, this leads to an internal body burden of 42 mg/kg/day.

The minimal MOS for repeated dose toxicity 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.As discussed in section 4.1.3.3.3, it is proposed that additional factors to take account of the use of a LOAEL rather than a NOAEL and semi-chronic to chronic exposure are not considered necessary.

The total daily human exposure to TCPP from regional sources is 2×10^{-4} mg/kg/day, which when compared to the internal body burden of 42 mg/kg results in a MOS of 210,000. Given the size of this MOS, c**onclusion (ii)** is drawn.

Mutagenicity

As with the worker section above, **conclusion (ii)** is drawn for man exposed via regional exposure in relation to mutagenicity.

Carcinogenicity

As described in section 4.1.2.8, it is proposed that the LOAEL of 52 mg/kg/day, identified from the 90-day study with TCPP will be taken forward for risk characterisation for carcinogenicity. Assuming 80% absorption by the oral route, this leads to an internal body burden of 42 mg/kg/day.

The minimal MOS for carcinogenicity 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. Normally, a factor of 3 would be used to take into account the use of a LOAEL rather than a NOAEL. However, this is not considered necessary here, as the LOAEL derived from the repeat dose toxicity study was based on liver weight changes which is not considered to be particularly toxicologically significant and the LOAEL is probably quite close to the NOAEL.

The total daily exposure to TCPP from regional sources is estimated as 2 x 10^{-4} mg/kg/day. When this is compared with the internal body burden of 42 mg/kg, the MOS is 210,000. The MOS is considered sufficient and so **conclusion (ii)** is drawn.

Reproductive toxicity

Effects on fertility

In a two-generation oral reproductive toxicity study in rats with TCPP, a LOAEL of 99 mg/kg is derived for effects on fertility. This is based on a decrease in relative uterus weight seen in all dosed females in F0 and the high dose females in F1. Assuming 80% absorption by the oral route, this leads to an internal body burden of 79 mg/kg.

In line with the draft TGD (2005), the minimal MOS for effects on fertility is 300. 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. A factor of 3 to account for the use of a LOAEL rather than a NOAEL is also employed. Although the effects seen at the low dose were slight, they did reach statistical significance and were considered to be biologically significant as they followed a dose dependent trend.

The total daily human exposure to TCPP from regional sources is 2 x 10^{-4} mg/kg/day. When this is compared with the internal body burden of 79 mg/kg, the MOS is 395,000. When the MOS is compared with the minimal MOS of 300, there is no concern and **conclusion (ii)** is drawn.

Developmental toxicity

From a two-generation reproductive toxicity study with TCPP, a LOAEL of 99 mg/kg is derived for developmental toxicity. Assuming 80% absorption by the oral route, this leads to an internal body burden of 79 mg/kg.

In line with the draft TGD (2005), the minimal MOS for developmental 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) and an intraspecies factor of 10. As described in section 4.1.3.2.7, a factor of 3 to account for the use of a LOAEL rather than an NOAEL is also employed.

The total daily human exposure to TCPP from regional sources is 2 x 10^{-4} mg/kg/day and comparing this with the internal body burden results in a MOS of 395,000. It is considered that there is a sufficient margin of safety and so **conclusion (ii)** is drawn.

4.1.3.4.2 Local exposure

Repeated dose toxicity

In relation to repeated dose toxicity, a LOAEL of 52 mg/kg/day was derived from a 13-week study in which male and female rats were dosed with TCPP at concentrations of up to 1349 mg/kg/day and 1745 mg/kg/day, respectively. This LOAEL was based on increased liver weights observed in male animals. Assuming 80% absorption by the oral route, this leads to an internal body burden of 42 mg/kg/day.

The minimal MOS for repeated dose toxicity 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. As discussed in section 4.1.3.3.3, it is proposed that additional factors to take account of the use of a LOAEL rather than a NOAEL and semi-chronic to chronic exposure are not considered necessary.

From section 4.1.1.3, the highest continuous local exposure is estimated to be 0.104 mg/kg/day, which is taken from **Table 4.39**. This figure (for A1a: large systems houses life cycle stage) has been derived using site-specific data for releases to waste water and default values for releases to air. The latter is driving the high value for 'leaf crops' for this life cycle stage, which results in the high local exposure value.

Comparing this to a body burden of 42 mg/kg results in a MOS of 404. When this is compared to the minimal MOS of 100, it is concluded that there is no concern and so c**onclusion (ii)** is drawn.

Mutagenicity

As with the worker section above, **conclusion (ii)** is drawn for man exposed via local exposure in relation to mutagenicity.

Carcinogenicity

As described in section 4.1.2.8, it is proposed that the LOAEL of 52 mg/kg/day, identified from the 90-day study with TCPP will be taken forward for risk characterisation for carcinogenicity. Assuming 80% absorption by the oral route, this leads to an internal body burden of 42 mg/kg/day.

The minimal MOS for carcinogenicity 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. Normally, a factor of 3 would be used to take into account the use of a LOAEL rather than a NOAEL. However, this is not considered necessary here, as the LOAEL derived from the repeat dose toxicity study was based on liver weight changes which is not considered to be particularly toxicologically significant and the LOAEL is probably quite close to the NOAEL.

From section 4.1.1.3, the highest continuous local exposure is estimated to be 0.104 mg/kg/day, which is taken from **Table 4.39**. This figure (for A1a: large systems houses life cycle stage) has been derived using site-specific data for releases to waste water and default values for releases to air. The latter is driving the high value for 'leaf crops' for this life cycle stage, which results in the high local exposure value.

When this is compared with the internal body burden of 42 mg/kg, the MOS is 404. When the MOS is compared with the minimal MOS of 100, there is no concern for this scenario and so **conclusion (ii)** is drawn.

A **conclusion (ii)** is drawn for all other life cycle stages.

Reproductive toxicity

Effects on fertility

In a two-generation oral reproductive toxicity study with TCPP in rats, a LOAEL of 99 mg/kg was derived for effects on fertility. This is based on a decrease in relative uterus weight seen in all dosed females in F0 and the high dose females in F1. Assuming 80% absorption by the oral route, this leads to an internal body burden of 79 mg/kg.

In line with the draft TGD (2005), the minimal MOS for effects on fertility is 300. 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. A factor of 3 to account for the use of a LOAEL rather than a NOAEL is also employed. Although the effects seen at the low dose were slight, they did reach statistical significance and were considered to be biologically significant as they followed a dose dependent trend.

From section 4.1.1.3, the highest continuous local exposure is estimated to be 0.104 mg/kg/day, which is taken from **Table 4.39**. This figure (for A1a: large systems houses life cycle stage) has been derived using site-specific data for releases to waste water and default values for releases to air. The latter is driving the high value for 'leaf crops' for this life cycle stage, which results in the high local exposure value.

When this is compared with the internal body burden of 79 mg/kg, the MOS is 760. When the MOS is compared with the minimal MOS of 300, there is no concern and **conclusion (ii)** is drawn.

Developmental toxicity

From a two-generation reproductive toxicity study with TCPP, a LOAEL of 99 mg/kg is derived for developmental toxicity. Assuming 80% absorption by the oral route, this leads to an internal body burden of 79 mg/kg.

In line with the draft TGD (2005), the minimal MOS for developmental 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) and an intraspecies factor of 10. As described in section 4.1.3.2.7, a factor of 3 to account for the use of a LOAEL rather than an NOAEL is also employed.

From **Table 4.39** in section 4.1.1.3, the highest continuous local exposure is estimated to be 0.104 mg/kg/kg, which has been derived for A1a:large systems houses. When this value is compared with the internal body burden of 79 mg/kg, the MOS is 760. It is considered that there is a sufficient margin of safety and **conclusion (ii)** is drawn.

4.1.3.4.3 Summary of risk characterisation for exposure via the environment

Conclusion (ii) is drawn for both regional and local exposures for all endpoints.

4.1.3.5 Combined exposure

The combined exposure to TCPP 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 reasonable worst case 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 TCPP indirectly from a) flexible foam used in upholstery and bedding and b) closed-cell rigid foam used for insulation. Consumers may also be exposed from the use of 1-K foams containing TCPP, which are used in DIY applications. 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 4.69**, below.

Table 4.69 Combined regional and local exposure to TCPP (excluding occupational exposure)

Source of exposure	Exposures	Body burdens (mg/kg bw)
Consumer		
Release of TCPP from flexible polyurethane foam		
Inhalation	0.0038 mg/m^3	0.001
Dermal	0.0011 mg/kg	0.0011
Use of 1-K foam		
Inhalation	0.005 mg/m^3	0.0014
Dermal	174 µg/cm^2	0.24
Release of TCPP from closed cell right foam	Negligible	Negligible
Man via the environment		
Local exposure	0.104* mg/kg/day	0.104
Regional exposure	0.0002 mg/kg/day	0.0002
Combined local (acute)	-	0.34
Combined local (repeated)	-	0.106
Combined regional (acute)	-	0.24
Combined regional (repeated)	-	0.0023

*highest exposure scenario for local exposure (A1a: large systems houses)

It should be noted that most consumers would not regularly use 1-K foams. It is most likely that consumers would use them less than once per year and more probably once or twice in a lifetime. Therefore, exposure for consumers in this scenario is considered to negligible over a lifetime, but could be significant in the short-term. Therefore, combined exposures with (acute) and without (repeated exposure) 1-K foam use have been calculated.

As discussed in section 4.1.1.4, occupational exposures are not included in the combined exposure calculation. As can be seen from **Table 4.58** 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.

Acute toxicity

No significant signs of toxicity were seen in experimental animals via the inhalation and dermal routes. With respect to oral exposure, a NOAEL of 200 mg/kg was identified from the acute oral toxicity studies. Assuming 80 % absorption by the oral route, this leads to an internal body burden of 160 mg/kg.

In line with the draft TGD (2005), the minimal MOS for acute toxicity is 100. This mMOS is established taking into account an interspecies factor of 10 (4 for metabolic size differences * 2.5 for sensitivity differences) and an intraspecies factor of 10.

From **Table 4.69** above, the body burden for the combined local exposure (acute) is 0.34 mg/kg. When this is compared with the internal body burden of 160 mg/kg, the MOS is 471. There are no concerns for the combined local exposure and so **conclusion (ii)** is drawn.

The body burden for the combined regional exposure (acute) is 0.24 mg/kg, which gives a MOS of 667. There are no concerns for the combined regional exposure and so **conclusion (ii**) is drawn.

Repeated dose toxicity

A LOAEL of 52 mg/kg/day was derived from a 13-week oral study in rats with TCPP. This LOAEL was based on increased liver weights observed in male animals. Assuming 80% absorption by the oral route, this leads to an internal body burden of 42 mg/kg/day.

In line with the draft TGD (2005), the minimal MOS for repeated dose toxicity is 100. This mMOS is established taking into account an interspecies factor of 10 (4 for metabolic size differences * 2.5 for sensitivity differences) and an intraspecies factor of 10. As discussed in section 4.1.3.3.3, it is proposed that an additional factor to take account of the use of a LOAEL rather than a NOAEL is not necessary in this case.

From **Table 4.63** above, the body burden for the combined local exposure (repeated) is 0.106 mg/kg. When this is compared with the internal body burden of 42 mg/kg, the MOS is 396. There are no concerns for the combined local exposure and so **conclusion (ii)** is drawn.

The body burden for the combined regional exposure (repeated) is 0.0023 mg/kg, which gives a MOS of 18,261. There are no concerns for the combined regional exposure and so **conclusion (ii**) is drawn.

Mutagenicity

As with the worker and consumer sections above, **conclusion (ii)** is drawn for combined exposures in relation to mutagenicity.

Carcinogenicity

As described in section 4.1.2.8, it is proposed that the LOAEL of 52 mg/kg/day, identified from the 90-day study with TCPP will be taken forward for risk characterisation. Assuming 80% absorption by the oral route, this leads to an internal body burden of 42 mg/kg/day.

The minimal MOS for carcinogenicity 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. Normally, a factor of 3 would be used to take into account the use of a LOAEL rather than a NOAEL. However, this is not considered necessary here, as the

LOAEL derived from the repeat dose toxicity study was based on liver weight changes which is not considered to be particularly toxicologically significant and the LOAEL is probably quite close to the NOAEL.

From **Table 4.69** above, the body burden for the combined local exposure (repeated) is 0.106 mg/kg. When this is compared with the internal body burden of 42 mg/kg, the MOS is 396. When this is compared with the minimal MOS of 100, there is considered to be a sufficient margin of safety. Therefore, it is concluded that there is no concern for the combined local exposure and so **conclusion (ii)** is drawn.

The body burden for the combined regional exposure (repeated) is 0.0023 mg/kg, which gives a MOS of 18,261. There are no concerns for the combined regional exposure and so **conclusion (ii**) is drawn.

Reproductive toxicity

Effects on fertility

In a two-generation oral reproductive toxicity study with TCPP in rats, a LOAEL of 99 mg/kg was derived for effects on fertility. This is based on a decrease in relative uterus weight seen in all dosed females in F0 and the high dose females in F1. Assuming 80% absorption by the oral route, this leads to an internal body burden of 79 mg/kg.

In line with the draft TGD (2005), the minimal MOS for effects on fertility is 300. 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. A factor of 3 to account for the use of a LOAEL rather than a NOAEL is also employed. Although the effects seen at the low dose were slight, they did reach statistical significance and were considered to be biologically significant as they followed a dose dependent trend.

From **Table 4.69** above, the body burden for the combined local exposure (repeated) is 0.106 mg/kg. When this is compared with the internal body burden of 79 mg/kg, the MOS is 745. There are no concerns for the combined local exposure and so **conclusion (ii)** is drawn.

The body burden for the combined regional exposure (repeated) is 0.0023 mg/kg, which gives a MOS of 34,348. There are no concerns for the combined regional exposure and so **conclusion (ii**) is drawn.

Developmental toxicity

From a two-generation reproductive toxicity study with TCPP, a LOAEL of 99 mg/kg is derived for developmental toxicity. Assuming 80% absorption by the oral route, this leads to an internal body burden of 79 mg/kg.

In line with the draft TGD (2005), the minimal MOS for developmental 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) and an intraspecies factor of 10. As described in section 4.1.3.2.7, a factor of 3 to account for the use of a LOAEL rather than an NOAEL is also employed.

The body burden for the combined local exposure (repeated) is 0.106 mg/kg, which results in a MOS of 745. When this is compared with the minimal MOS of 300, there is no concern for this scenario and so **conclusion (ii)** is drawn.

The body burden for the combined regional exposure (repeated) is 0.0023 mg/kg, resulting in a MOS of 34,348. It is considered that there is a sufficient margin of safety and therefore **conclusion (ii)** is drawn for this scenario.

Summary of risk characterisation for the combined exposure

Conclusion (ii) is drawn for combined exposure for all endpoints.

4.2 HUMAN HEALTH (PHYSICO-CHEMICAL PROPERTIES)

4.2.1 Exposure assessment

Exposure potentially occurs in the workplace during the manufacture of TCPP and during the manufacture of flexible and rigid PUR foam containing TCPP.

4.2.2 Effects assessment: Hazard identification

4.2.2.1 Explosivity

Explosive properties have not been tested. Based on its chemical structure and the known synthesis route of manufacture via an exothermic chemical reaction, there is no indication that the substance is thermodynamically unstable. The DSC test used for boiling point measurement showed no exotherms. The substance does not contain any of the more commonly known endothermic groups such as azides, cyano-, dienes, peroxide or chlorate. Therefore, TCPP is not expected to possess explosive properties.

4.2.2.2 Flammability

Based on the known chemical and physical properties of TCPP and its chemical structure, it is not expected to produce flammable gases in contact with water or damp air.

4.2.2.3 Oxidizing potential

Oxidising properties have not been tested. By reference to the structural formula, it can be seen that TCPP 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.

4.2.3 Risk characterisation

TCPP 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)).**

5 RESULTS [23]

5.1 INTRODUCTION

The conclusions from the risk characterisation processes are brought together and summarised below.

5.2 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 to all compartments for all local life cycle stages, and at the regional scale in all compartments.

With regard to secondary poisoning, 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, due to the low ratios and lack of any significant bioaccumulation potential of TCPP, it is reasonable to conclude that there are no risks.

TCPP does not meet all of the PBT criteria (it meets the screening criteria for P or vP).

5.3 HUMAN HEALTH

5.3.1 Human health (toxicity)

5.3.1.1 Workers

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 worse case dermal exposure during the manufacture of TCPP (worker scenario 1) in relation to effects on fertility and developmental toxicity.

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.

[23] 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 (ii) applies to all worker exposure scenarios for the endpoints acute toxicity, irritation, sensitisation, repeated dose toxicity, mutagenicity and carcinogenicity.

Conclusion (ii) applies to typical dermal exposure and inhalation exposures, both reasonable worst case and typical, during the manufacture of TCPP (worker scenario 1) in relation to effects on fertility and developmental toxicity.

Conclusion (ii) applies to all other worker exposure scenarios (worker scenarios 2-10) for both reasonable worst case and typical exposures in relation to effects on fertility and developmental toxicity.

5.3.1.2 Consumers

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 in relation to all toxicological endpoints.

5.3.1.3 Humans exposed via the 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 to both regional and local exposures in relation to all toxicological endpoints.

5.3.1.4 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 in relation to all toxicological endpoints.

5.3.2 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 TCPP - 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 FORMULATION OF SYSTEMS: USE A

Overview

TCPP is added to polyols in the formulation of PUR systems; while some PUR producers buy polyols, isocyanates and other raw materials direct from manufacturers, others purchase pre-mixed, ready-to-use systems.

The suppliers of raw materials (i.e. polyols, isocyanates) are members of ISOPA, the European Diisocyanate and Polyol Producers Association. ISOPA is the European trade association for the producers of di-isocyanates. It was formed in 1987 by seven chemical companies that have European interests in the production of raw materials for PUR and is an affiliate of European Chemical Industry Council (CEFIC) (ISOPA 2002a). ISOPA has provided information for the development of this risk assessment and has acted as a focal point for input from other downstream users of TCPP.

Small to medium-sized systems houses tend to manufacture small volumes of systems to supply local manufacturers and smaller PUR processors. They often supply niche markets where the major manufacturers are unwilling to manufacture in small enough volumes. Some systems houses manufacture only a number of standard systems for various applications, whilst others also offer custom manufacture. There are in excess of 50 small to medium-sized systems houses in the EU (IAL 2000).

Systems houses tend to purchase TCPP direct, but some of the smaller houses may purchase TCPP-containing polyols from the raw materials suppliers. Discussions with industry (pers. comm. 31st July 2002, producers and downstream users) indicate a number of relevant points:

- It was indicated that very few systems houses will purchase pre-formulated polyols as it is economically inefficient to include a "middle man" in the supply chain. Thus, companies using pre-formulated polyols will be very specialist companies.
- It was suggested that where TCPP-containing polyols were purchased for further processing this would tend to be from the producers of PUR raw materials (i.e. ISOPA members).
- It was also suggested that this stage of the chain of trade may be associated with producers of PIR foam (see Section 2.2.2.3.6 of the main risk assessment report) who use very small amounts of TCPP as a viscosity reducer.
- It was estimated that less than 1% of the TCPP used by systems houses would be used as pre-formulated polyol.

The Market for Systems

Polyurethane systems in general are used in the manufacture of (IAL, 2000):

- flexible foam: block, semi-rigid and moulded components
- rigid foam: insulation, appliances, moulded components, panels, sprays
- elastomers: hot and cold cure, microcellular, thermoplastic polyurethanes (TPUs) and technical parts
- coatings
- adhesives
- sealants.

The market for systems is given in **Table A.1**. The principal use of polyols is the fabrication of polyurethane foam, a minor proportion (9%) being used for production of coatings, adhesives, sealants and elastomers ("CASE" applications) (EC, 2000).

Table A.1 Market for Systems

Rigid Foam	Comment[1]	Tonnes	Flexible Foam	Comment[1]	Tonnes
Appliances	Mostly NFR	175,000	Auto seating		71,000
OCF aerosols		62,000	Moulded components	NFR	47,000
Pipe in pipe		27,000	Semi rigid foam	NFR	25,500
Sandwich panels		165,000	Integral skin	NFR	25,500
Boardstock		165,000	Total Flexible foam		169,000
In situ foam		25,000			
Spray foams		56,000	**CASE**		
Moulded foams	Mostly NFR	6,000	Elastomers – footwear	NFR	195,000
Total rigid foam in EU		>700,000	Elastomers – other	Mostly NFR	101,000
			Other CASE		330,000
			Total CASE		626,000
Total of PUR market		45%	across all uses		

Note: 1 – the Industry has indicated that several of these applications are largely non-flame retarded (NFR). This is recorded in the Comments column.

TCPP-containing systems are used almost exclusively in the manufacture of rigid foams (Pers. comm. 16th October 2001). In this regard, while "short chain" polyether polyols are used for rigid foams, "long chain" polyether polyols are used for flexible foams (and 90% of those used in CASE applications are of the long chain type). Short and long chain polyols have different technical specifications and different physical properties, and users consider them to be non-substitutable (EC, 2000).

2 FLEXIBLE FOAM FOR FURNITURE: USE B – PRODUCTION

Slabstock foams[24]

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. 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 discolouration 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 20ºC 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 a 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 1982 in EC 2000). The blocks may for example be up to 60 metres 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

[24] The majority of the description of foam production presented in this section is taken from the risk assessment for pentabromodiphenyl ether (EC, 2000).

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/m3, 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).

Polyether versus polyester foams

Slabstock foam exists in both polyether and polyester form, depending on the nature of the polyol used (i.e. polyethers or polyesters). Polyether foams are different 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). 80% to 90% of the polyols used today are polyetherols (BASF, undated 1). TCPP is used exclusively in comfort foam for the UK market.

Polyether PUR 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). The market for comfort foam is influenced by downstream producers moving production to Eastern Europe, and by excess in production capacity for all producers (EC, 1997). Eastern Europe could therefore be an important source of finished goods.

Combustion Modified Foam

Combustion Modified High Resilience (CMHR) foams were introduced in 1987/1988. These are high resilience foams modified with melamine and other flame retardants. In the mid 1990s Combustion Modified Polyethers (CME) were introduced by a UK based foamer, and other foamers followed suit. While CME and CMHR use different polyols, the technologies are the same. There are various grades of CMHR and CME foams, with the grade reflecting different hardnesses, densities and colours.

CMHR foam needs to be crushed to create the required density of cell windows, while other foams have this naturally. Without crushing, CMHR foam would be crushed in use, e.g. as people sat on the furniture, which would not be acceptable to the customer (pers. comm.).

3 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
- 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. This document was revised and reissued in 2005, but the options under discussion are the same in the new edition (ISOPA, 2005).

A description of the range of PUR recycling options currently available is given in **Table A.2**. This includes a discussion of recycling for rigid foam applications.

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 2.2.2.2.5 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 2.2.2.2.5 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: PUR 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	PUR 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):

a) densification of low density, voluminous PUR foams, allowing for cost-effective transportation from collection point to recycling operation
b) 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 section 2.2.2.2.3). 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. 16th October 2001).

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 batch or through continuous moulding. The foam blocks are further processed to fabricate parts and articles, resulting in trim which 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 (EUROMOULDERS 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).
- Foam scrap 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 are 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. Most scrap foam currently goes to mainland Europe (e.g. Italy, the Netherlands and Germany), and there is a shortage of scrap foam in the UK (pers. comm.).

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 which contains TCPP. 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 TCPP in the EU.

4 RIGID PUR FOAMS FOR USE IN CONSTRUCTION: USE C

There are two major differences between the production of flexible and that of rigid PUR foam. The first is the closed-cell nature of the rigid foam and the second is the point that almost all products are covered from the point of manufacture by impermeable or semi-permeable barriers.

For the production of PUR rigid foam, MDI is mixed with a polyol component in a mixing head. Driven by catalysts, the reaction starts within seconds while the mixture is poured on a transport belt, shielded by flexible or rigid facings, depending on the type of rigid foam required. The foam rises and cures and after several metres, the foam is sufficiently stable to be cut into blocks or panels.

Key products

The following describes some of the key products associated with PUR insulating foam and is taken in the main from Jeffs (2000) and, in the case of sandwich panels, Koschade (2002). The description includes some discussion of production processes. The general discussion of PUR flexible slabstock foam production in section 2 of this appendix is also relevant.

Flexible-Faced Laminate

Flexible faced laminate is a major product of the rigid foam industry and is based on PUR or polyisocyanurate (PIR) foam in a range of thickness between 30 and 120 mm. The flexible facing materials, supplied in rolls, include glass fleece, aluminium foil, kraft paper and combinations of these. The continuous production process involves the pouring of the foam chemicals onto the lower facing material which is carried by a conveyor belt, the chemicals

react, the foam is formed and the upper facing is unrolled to meet the upper surface of the foam. The whole is conveyed into a curing tunnel and, at the end of the process the product is cut into the size to be used in buildings, usually 2.4 x 1.2m (Jeffs 2000).

The production process involved in the manufacture of flexible-faced laminate generally occurs in a closed system, with only a very short period (seconds) where the chemicals are in the open work environment.

Exhaust systems are installed to ensure compliance with national occupational hygiene limits. Depending on the production line, the air of the lay down area and of the cutting area may be emitted to separate stacks or via a single stack (Schupp 2001).

The uses are in the insulation of the walls and roofs of buildings. In walls they can be used in the cavity between bricks, on the outside with a cover (e.g. a "ventilated" façade) or on the inside of a structure. In roofs they are used over concrete, steel or other decks and covered with a "weather" protective cover such as bitumenised felt (Jeffs 2000).

When these products were based on hydrofluorochlorocarbons (HCFCs), they have the advantages of having the highest degree of insulation efficiency and meet the most stringent fire standards applicable to organic-based materials (Jeffs 2000). However the use of HCFCs has been banned in the EU since end 2003. They have been replaced by either further use of pentane or the HFCs (ISOPA and the rigid polyurethane foam industry, 2006).

Sandwich Panels

Sandwich panels are of similar importance and are made of a PUR or PIR foam core of thickness 30 to 200 mm and faced with rigid materials. The most common is profiled steel and the production process is similar to that for flexible faced laminate except that the steel is supplied in rolls and fed through profiling rollers just before the polyurethane is applied. Other facing materials include copper, aluminium and gypsum board. The metal-faced products are cut into lengths of up to 15-20m and the gypsum board-faced products into panels of size 2.4 x 1.2m (Jeffs 2000).

The most important process for manufacturing sandwich panels with metallic facings and a foamed PUR core is continual production on double conveyor belts (i.e. double belt machines). Both metallic facings are unrolled from coils on metal rollers up to 1.3 m wide, are profiled according to the profile form desired and then fed into the double belt machine. The mixing head feeds the liquid PUR reaction mixture with an optimum oscillation frequency evenly onto the lower facing that is moved along by the double belt machine. The foaming mixture adheres to the lower as well as the upper metallic cover layer under the influence of heat and free rise foaming pressure. This results in a continuous sandwich panel that is then further processed step by step along the double belt machine, cut to supply lengths and packed (Koschade 2002).

To separate the continuous panel into individual sandwich panels, twin bladed circular saws, a band saw or a length cutting machine with combined saws are used. The chemical reaction of the polyol and isocyanate creates intense heat. The thickness of the insulation also influences the intensity of the heat of reaction. For this reason a cooling space to allow stretch is placed after the cross-cut section. Panels are stacked in cooling racks so that they are evenly and rapidly cooled. In the final step the cooled sandwich panels arrive at a run-out section of the double belt machine, the stacking installation. The panels are stacked onto pallets by surface-protecting vacuum suction and packed for dispatch (Koschade 2002).

The production capacity of the whole plant depends primarily on the length of the double belt. Normal double belt lengths for the production of roof and wall panels are 30 m with a maximum speed of 15 m per minute. The normal production speed for 40 mm thick sandwich panels is between 10 m and 12 m per minute. For a thickness of 80 mm the speed is reduced to approximately 6 m to 8 m per minute. With a two shift operation of 4,000 hours per year and a production speed of 8 m per minute, such a machine can produce around 2 million m^2 of sandwich panels (with a width of 1,000 mm and not taking re-tooling time into account). The largest double belt machines have speed of up to 30 m per minute and production capacities of 2,160 m^2 of sandwich panels per hour. This corresponds theoretically to a total capacity (not taking re-tooling time into account) in a three-shift operation of 19 million m^2 per year (Koschade 2002).

The metal-faced panels are used to construct many types of buildings including factories and stores, especially those which need hygienic, temperature-controlled environments such as food processing, electronics and pharmaceuticals manufacturing. Their uses also include food cold stores (hence the 200 mm thick products), schools, sports halls and in the conversion of existing buildings for new uses. The gypsum-faced products are used as internal linings for walls and ceilings in many types of buildings including houses and are especially useful in retrofitting existing buildings (Jeffs 2000).

The steel facings on the panels fully protect the core. In addition, panel joints are fully engineered to provide excellent weather and air-tightness and also to protect the core materials in the event of a fire (EPIC 2002).

Discontinuous Panels

Discontinuous panels are similar to the continuously produced variety in appearance but are produced by injecting the PUR or PIR foam chemicals in-between pre-cut steel sheets. They are used in a variety of applications including cold rooms for food stores for supermarkets. The same advantages for HCFCs apply for these panels as for the continuously produced versions (Jeffs 2000).

Block Foams

Block foams of section about 1.5 by 1.0 m are produced either discontinuously in blocks of length about 2m or produced continuously. They are cut into shapes such as pipe sections or sheets. The latter are glued to facing materials to make panels. The production process can be strongly exothermic and the temperature in the middle of the block can reach over 200ºC (an alternative source (pers. comm. 11/02/03) indicates that maximum temperature in rigid block foams is 150ºC) and the block will take a long time to cool after manufacture. Without care the centre of the block can scorch. Because of these high temperatures the use of hydrocarbons is obviously avoided (Jeffs 2000).

Injected Foam

Injected foam is a general term, widely used in the USA, to describe a general foam process where the foam is injected into a cavity in a discontinuous process. Thus, it is used for making domestic and commercial refrigerators and freezers, discontinuous sandwich panels, pipe-in-pipe products and several others (Jeffs 2000).

End of life

Co-combustion of insulation foams from the building and construction industry with municipal solid waste is seen as the most environmentally friendly option. This is because crushing and compressing the foam to reduce its density for disposal by landfill can be problematic due to the presence of ozone depleting blowing agents (chlorofluorocarbons) in old foams that would be released by the process (Vehlow and Mark1996).

In the Netherlands, all building and construction waste is collected and manually separated. 98% of the light fraction incinerated. In Belgium it is the same. However, there will be national or regional requirements, and thus regional differences (pers. comm. 31st July 2002, producers and downstream users).

Re-use is not significant for construction panels; it is not practised to a large extent (pers. comm. 31st July 2002, producers and downstream users). Koschade (2002) states that there is some re-use, and significant amounts of incineration with energy recovery. In addition, it is evident from Koschade (2002) that the quantity of panels being manufactured has been increasing for some years, and is likely to continue to increase.

When insulation foams are removed from buildings at the end of life the usual practice is to bury these foams in landfill. In the longer term it is recognised however that most insulants will be eventually excluded from landfill, principally because of organic content and/or stability requirements of the landfill sites (ISOPA 1996b). In this regard, several countries in Western Europe have already limited landfill to "earth like" mineral substances, i.e. those having a very low content of organic material (Vehlow and Mark, 1995).

5 SPRAY FOAMS: USE D

Overview

Elastogran (one of the key manufacturers of spray foams) has produced a brochure entitled *Sealing and Insulation with Elastopor® H PUR Spray Foam* (BASF undated 2). Its product, Elastopur H roof spray, is a polyurethane rigid foam with up to 95% closed cell content. It is produced through the mixing of two initially liquid components, namely the A-component (polyol) and the B-component (diphenylmethane di-isocyanate - MDI). The mixing of the two components produces a reactive mixture which forms under heat evolution. At the end of the reaction phase the foam starts to solidify and cure.

The foam is applied by a spray gun in several layers, with an experienced processing team able to cover more than 1000 m^2 of roof surface per day. The spray guns are mobile high pressure spraying units. The A and B components are pumped through heated high pressure hoses to the spray gun where they are completely mixed by counter-flow injection (BASF undated 2). The temperature reached in the spray 'gun' is typically 120-140°C, but this is not considered to be an issue for risk assessment since the foam surface would cool rapidly once sprayed.

The building supervisory/building regulations approval prescribes at least three layers of foam with a total minimum overall thickness of 30 mm. Within a few minutes of coating the foam is cured and hard enough to walk on. The foam provides thermal protection on roofs and provides a jointless seam against precipitation. To protect against ultra violet radiation foams are coated, for example using a silver reflective coating or a gravel layer (BASF undated 2).

All other use scenarios are described in detail in the Confidential Annex.

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:

2. Review of measured data
3. A new mathematical model
4. 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

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+ (µg 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

+ Based on total emission measured from PUR plug and walls of test vessel.
*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:

1. Weight loss following thermal ageing at room temperature, 40°C and 60°C.
2. Analysis of flame retardant content following solvent extraction of foam aged at 60°C.
3. Analysis of flame retardant emissions in aqueous media designed to model dermal absorption (contact blotting tests) and chewing (head over heels tests).
4. Measurement of volatile emissions during thermal ageing in sealed vials.
5. 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:

1. A combustion modified (CM) ether foam containing 8.47% by weight TCPP.
2. A combustion modified high resilience (CMHR) foam containing 5.2% by weight TCPP.
3. 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:

1. Dust (diameter less than 1 mm)
2. Small crumbs (diameter 3 mm to 1 cm)
3. 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.

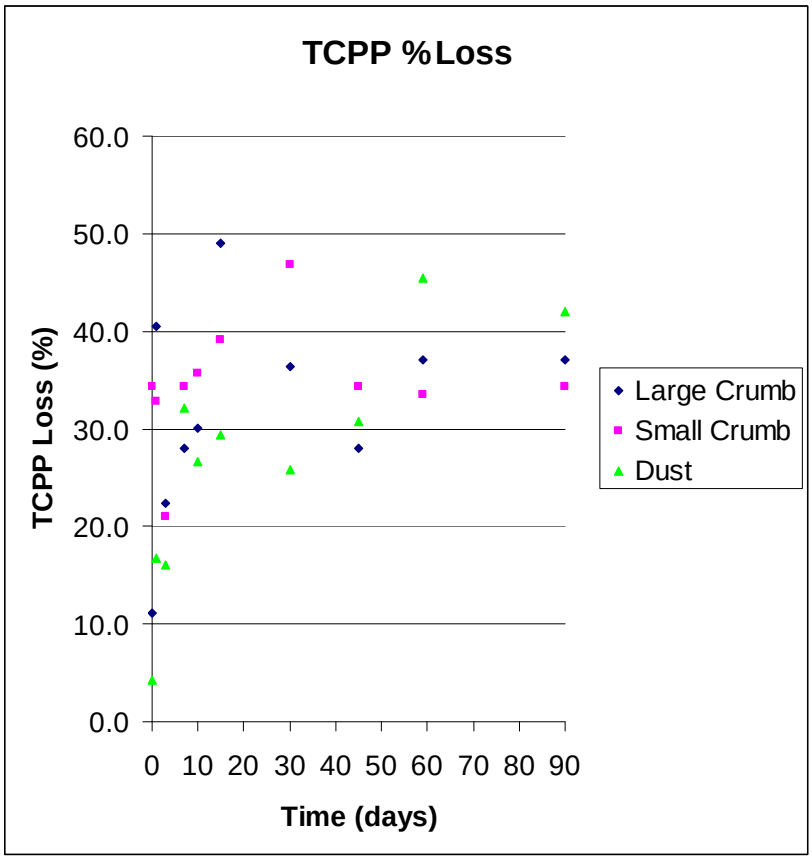
2.1.6.2 Results

Results of the study are presented in **Table B.7** and **Figure B.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 releases 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 μgm^{-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:

1. 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.
2. 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.
3. 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.

4. 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.
5. 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 B2 shows the total amount lost versus timescale for a 1 m^2 x 0.1 m block of foam, while Figure B3 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 B.13**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 m2)

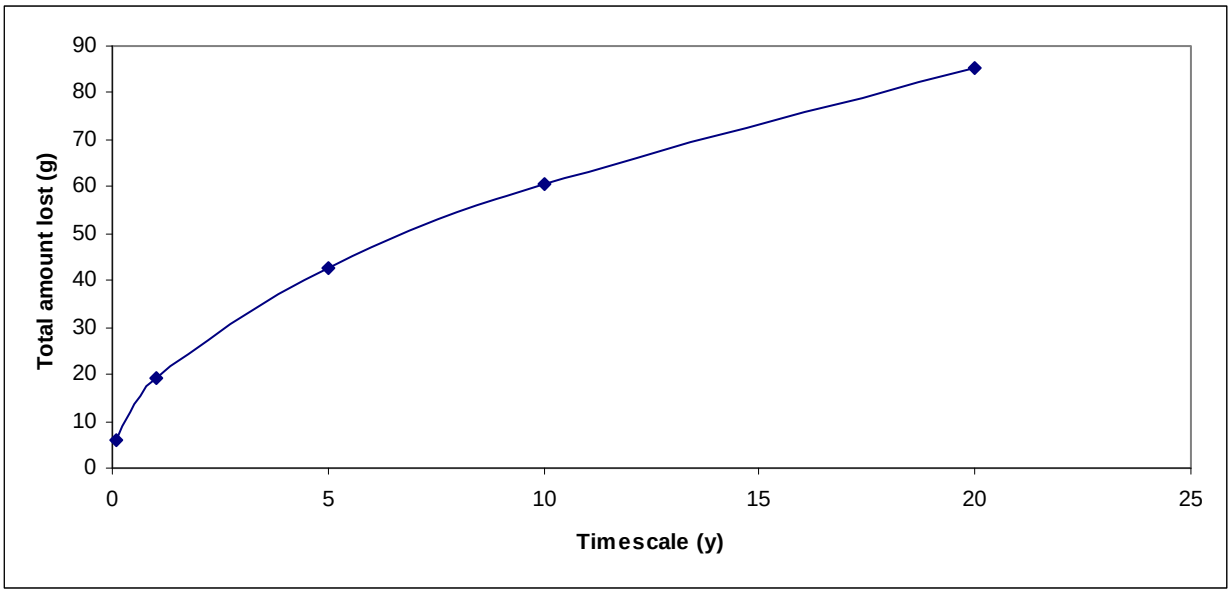
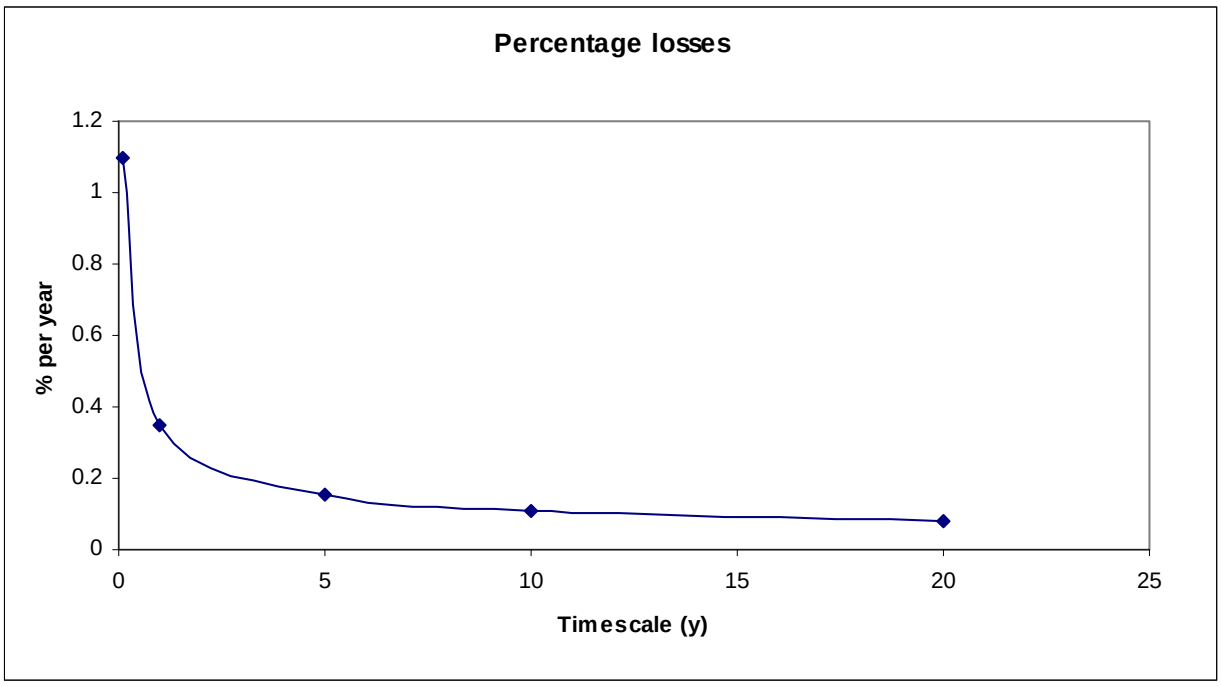


Figure B.3 Annual average percentage loss as a function of timescale (thickness = 0.1 m)



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:

$D\ (m^2/s) = 10^{(-7.83 - 0.0062MW)} /10000$

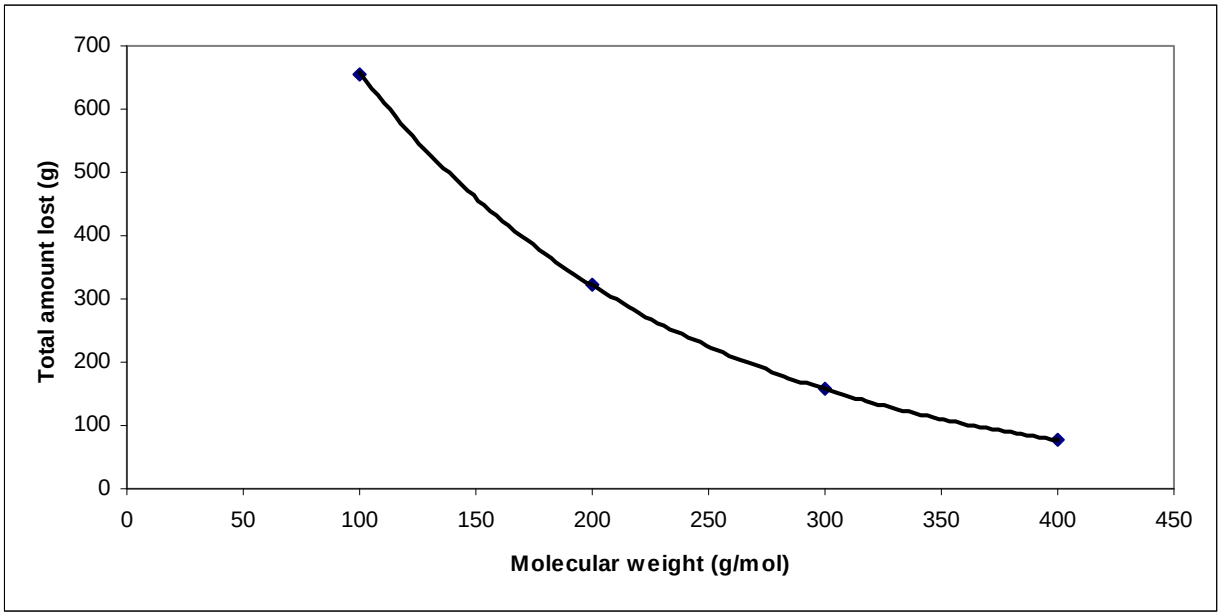
Using diffusion coefficients predicted by the model, releases for varying molecular weights are shown in **Table B.14** and **Figure B.4**.

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).

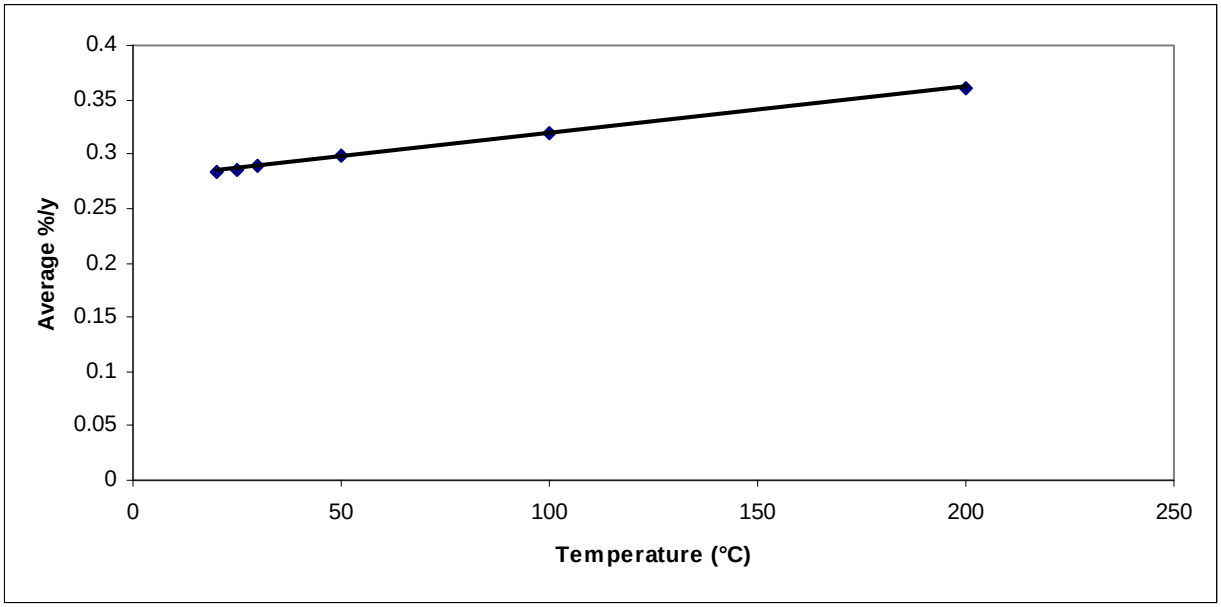
Table B.15 Amount lost as a function of temperature

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 furnishing & sound insulation; including rebonded foam)	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 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.

Application area	Conclusions
Loose crumb	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 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. Correcting for availability, the release rate used in the risk assessment is: TCPP: 4.8E-06% to air and 4.8E-06% to wastewater

Application area	Conclusions
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
Organization
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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
(2) Al-Malaika, S; Migration of 4-substituted 2-hydroxybenzophenones in low-density polyethylene. Part I. Diffusion characteristics; Polym Degrad Stability 1991, V32, P231
(3) Berens, A; Diffusion of organic vapors at low concentrations in glassy PVC, polystyrene, and PMMA; J Membrane Sci 1982, V10, P283
(4) Brandsch, J; Plastic Packaging Materials for Food. Barrier Function, Mass Transport, Quality Assurance and Legislation 2000
(5) Feigenbaum, A; Safety and quality of foodstuffs in contact with plastic materials: a structural approach; J Chem Educ 1993, V70, P883
Display from CAplus database

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
(1) Adcock, L; The migration of non-volatile compounds from plastics. Part 3. Further experiments with model systems and development of the descriptive and pictorial concept of migration; Plastics and Rubber Processing and Applications 1984, V4, P53
(2) Al Malaika, S; Migration of 4-substituted 2-hydroxybenzophenones in low-density polyethylene. Part I. Diffusion characteristics; Polymer Degradation and Stability 1991, V32, P231
(3) Baner, A; The application of a predictive migration model for evaluating the compliance of plastic materials with European food regulations; Food Advances and Contaminants 1996, V13, P587
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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
0265-203X
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
Cited Reference or Reference
(1) Baner, A; Alternative methods for the determination and evaluation of migration potential from polymeric food contact materials. Continued; Deutsch Lebensmittel-Rundschau 1994, V90, P181
(2) Baner, A; Alternative fatty food simulants for migration testing of polymeric food contact materials; Food Additives and Contaminants 1992, V9, P137
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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.
Document Type
Journal
Language
English
Accession Number
2000:582442 CAPLUS
Document Number
134:148224
Cited Reference or Reference

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(6) Urban, M; Attenuated Total Reflectance Spectroscopy of Polymers: Theory and Practice 1996
Display from CAplus database
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.
Organization
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
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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.
Document Type
Journal
Language
English
Accession Number
2000:336335 CAPLUS
Document Number
133:104009
Cited Reference or Reference
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(4) Baner, A; The application of a predictive migration model for evaluating the compliance of plastic materials with European food regulations; Fd Addit Contam 1996, V13, P587
(5) Baner, A; Food Packaging and Preservation 1994
Display from CAplus database
ANSWER 69 ©2002 ACS
Title
Polymer additive migration to foods-a direct comparison of experimental data and values calculated from
migration models for high density polyethylene (HDPE)
Author
O'Brien, Anthony; Goodson, Anne; Cooper, Ian
Organization
Pira International, Surrey, KT22 7RU, UK
Publication Source
Food Addit. Contam. (1999), 16(9), 367-380
Identifier-CODEN
FACOEB
ISSN
0265-203X
Publisher
Taylor & Francis Ltd.
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.

Document Type
Journal
Language
English
Accession Number
1999:574969 CAPLUS
Document Number
131:285593
Cited Reference or Reference
(1) Baner, A; Alternative methods for the determination and evaluation of migration potential from polymeric food contact materials. Continued; Deutsche Lebensmittel-Rundschau 1994, V90, P181
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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	ca. 288 (decomp)	1	ca. 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	
2.11	Oxidising properties	-	ND	-	ND	-	ND	-	ND	Not possible or necessary

IUCLID ref	Endpoint	TCEP	R [a]	TCPP	R	TDCP	R	V6	R	Comment on the data, QSAR or read-across
Environmental fate and behaviour										
3.5	Ready biodegradability	No	1	No	2	No	2	No (not GLP)	2	Weight of evidence is that none 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	
3.3	Log Koc			2.24 (Koc = 174, calculated from TDCP value)	1	3.25 (OECD 106) (Koc = 1780)	1 1	2.39 (Koc = 245, calculated from TDCP	1	Full study more reliable than HPLC estimation.

IUCLID ref	Endpoint	TCEP	R [a]	TCPP	R	TDCP	R	V6	R	Comment on the data, QSAR or read-across
	Log Koc (estimated by HPLC method) (Estimated using TGD QSAR for TCEP)	*2.04* (Koc estimated from log Kow)	1	2.76	1	4.09		value) 4.04	1	

IUCLID ref	Endpoint	TCEP	R [a]	TCPP	R	TDCP	R	V6	R	Comment on the data, QSAR or read-across
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).
	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	

IUCLID ref	Endpoint	TCEP	R [a]	TCPP	R	TDCP	R	V6	R	Comment on the data, QSAR or read-across
	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
	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)

IUCLID ref	Endpoint	TCEP	R [a]	TCPP	R	TDCP	R	V6	R	Comment on the data, QSAR or read-across
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 OECD 209	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
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 2000): Earthworm Reproduction Test*	1	14 d LC50 >1000 (>340) 14 d NOEC >1000 (>340) (not GLP) *Eisenia foetida* *OECD207*	1	
	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	

IUCLID ref	Endpoint	TCEP	R [a]	TCPP	R	TDCP	R	V6	R	Comment on the data, QSAR or read-across
	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

Appendix D: TCPP – Carcinogenicity endpoint.

Proposal to perform a qualitative read-across from data on structurally similar substances TDCP and TCEP

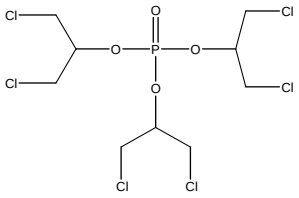
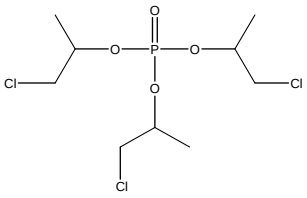
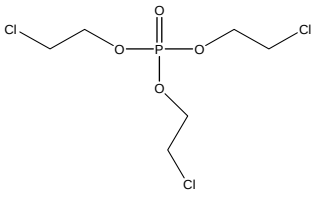
There are no carcinogenicity data available for TCPP.

TCPP is structurally similar to two other chlorinated alkyl phosphate esters, TDCP and TCEP. TDCP and TCEP are non-genotoxic carcinogens and have agreed classifications of Carc Cat 3 R40[25]. Therefore, the acceptability of read-across from TCEP and TDCP to address the potential carcinogenicity of TCPP is considered in this appendix.

1. Structure

TDCP, TCPP and TCEP are structurally similar (see **Table D.1** below); all contain a central phosphate group covalently linked to three chloroalkyl chains. Where these compounds differ structurally, is in the nature of the chloroalkyl chains attached to the central phosphate group.

Table D.1 Structures of TCEP, TCPP and TDCP

Name	Tris [2-chloro-1-(chloromethyl)ethyl] phosphate (TDCP)	Tris (2-chloro-1-methylethyl) phosphate (TCPP)	Tris (2-chloroethyl) phosphate (TCEP)
Structure			

It is thought that TDCP is the most sterically hindered of the three substances, as it contains three branched (dichloromethyl)ethyl groups which are thought to 'crowd' the central phosphate group. For this reason it is expected that reactivity at the P=O in TDCP would be lower than in the less sterically hindered P=O in either TCPP or TCEP. TCPP contains the less bulky chloromethylethyl groups, hence it is thought that this reduction in steric hindrance would lead to greater reactivity at the P=O in TCPP, when compared with TDCP. It is expected that the unbranched monochloroethyl chains of TCEP would cause the least amount of steric hindrance around the P=O, therefore TCEP is thought to be most reactive when compared to TCPP or TDCP.

It is thought that the electronegative chlorine atoms of TDCP, TCPP and TCEP may have an effect on the lability of the phosphate ester groups to differing degrees. It is expected that the chlorine atoms in TDCP will create a strong $^{-}$I-effect whereas in TCPP, the $^{-}$I-effect created by the chlorine atoms will be counteracted by the $^{+}$I effect of the adjacent methyl groups. As a result, the phosphate ester group of TDCP is expected to be more labile than the phosphate ester group of TCPP.

Based on this structural assessment, it is expected that TDCP and TCPP are most similar based on the nature of the three branched chloroalkyl chains surrounding the central phosphate group in both.

[25] 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.

The substances were also evaluated using a hierarchical clustering with the QSAR data-mining tool, Leadscope (Patlewicz *et al*., 2007). The modified Tanimoto index within the tool was used as a means of comparing the substances for structural similarity. The Tanimoto index is used to quantitatively relate two or more chemicals together on the basis of the commonality of features between those chemicals. In addition, the model also compares the absence of structural features. When the cluster threshold distance (i.e. a cut-off value to determine whether a chemical belongs to one cluster or another) was set to the default value recommended for similar substances, all three substances were found to be in the same cluster and thus very similar to each other. When the substances were then clustered based on structural features, TCEP and TCPP were found to be most structurally similar, with TDCP less similar than the other two (Patlewicz *et al.,* 2007).

Although the conclusion of the visual assessment and QSAR analysis of the structures differ slightly, overall it can be considered that TCPP is sufficiently similar to both TCEP and TDCP to support a read-across.

2. Physical Chemical Properties

The key physical chemical properties of each are presented in **Table D.2** below.

Table D.2 Physical chemical properties of TCEP, TCPP and TDCP

Name	*TCEP	TCPP	**TDCP
Molecular weight	285.49	327.57	430.91
Physical state	Liquid	Liquid	Liquid
Melting point	<-70 ^{0}C	<-20 ^{0}C	<-20 ^{0}C
Boling point	320 ^{0}C (decomp)	Ca. 288 ^{0}C (decomp)	Ca. 326 ^{0}C (decomp)
Relative density	1.4193 at 25 ^{0}C	1.288 at 20 ^{0}C	1.513
Vapour Pressure	1.14 x 10^{-3} Pa at 20 ^{0}C (extrapol)	1.4 x 10^{-3} Pa at 25 ^{0}C	5.6 x 10^{-6} Pa at 25 ^{0}C
Water solubility	7820 mg/l at 20 ^{0}C	1080 mg/l at 20 ^{0}C	18.1 mgl
Log Kow	1.78	2.68 ± 0.36	3.69 ± 0.36

* Values taken from BAUA, 2006

**Values taken from HSA/EA 2008a

All three substances are liquid at room temperature. The molecular weights, boiling points and relative densities of the substances are comparable. There are slight differences in the water solubility's of the substances, with TDCP having a lower water solubility value (18.1 mg/l) than the other two substances. All three substances have log Kow within the range 1-4, indicating favourable absorption. The vapour pressure of TDCP is lower than the comparable TCEP and TCPP. However, the vapour pressures of all three substances are not considered to be toxicologically significant. Although there are some minor differences in the physical chemical properties, the substances can be considered comparable.

The physiochemical similarity of the substances was also evaluated using Leadscope software (Patlewicz *et al.,* 2007). Clustering analysis was conducted based on physicochemical descriptors: lipophilicity (log P and water solubility) and molecular size (including molecular mass and molecular refraction). TDCP and TCPP were found to be most similar to each other based on the chosen physical chemical parameters. When the cluster threshold distance was

increased, all three substances were clustered into one group, indicating that all three substances can be considered similar (Patlewicz *et al.,* 2007).

It can be concluded, therefore, that the physical chemical properties of TCPP are sufficiently comparable to TDCP and TCEP to support a read-across.

3. Reactivity

The reactivity profiles of the three substances were analysed using quantum-mechanical calculations with the TSAR software (Patlewicz *et al.,* 2007). For each structure, the LUMO (energy of the lowest unoccupied molecular orbital), HOMO (energy of the highest occupied molecular orbital) and the partial charge values were calculated. The LUMO can be used as a means of modelling the overall electrophilicity of a chemical: the lower the LUMO value the greater the electrophilicity. TDCP had the lowest LUMO value of the three substances, indicating that it is the most electrophilic and therefore may be expected to be most reactive. TCPP had the highest LUMO value and TCEP was approximately mid-way between the two. In order to try to identify the reaction centres in the structures, the partial charges of each structure were calculated. However, these were found to be more or less constant between the substances and therefore inconclusive as to which part of the molecule is influencing the reactivity. The HOMO values, which provide information on a chemical's propensity to act as a nucleophile, were constant between the three substances and indicating no evidence of nucleophilicity.

It can be concluded that TDCP is the most electrophilic of the substances and TCPP the least, however the comparable partial charges between all three substances mean that it is not possible to identify which part of the structure influences the reactivity. Therefore, while the electronic parameters of the three substances are similar, no further insight into the reactivity of the substances is gained from this analysis.

4. Toxicokinetics

Absorption, distribution & excretion

Following oral administration, all three substances are well absorbed from the gastrointestinal tract. In a study conducted by Minegishi *et al*. (1988), the comparative absorption, distribution and excretion of ^{14}C-TCPP, ^{14}C-TDCP and ^{14}C-TCEP were evaluated following a single oral dose in rats. The percentage radioactivities recovered after 7 days are presented in **Table D.3** below.

Table D.3 Percentage recovery of radioactivity in rats following oral administration of ^{14}C-TCEP, ^{14}C-TCPP and ^{14}C-TDCP

	Percentage recovery of radioactivity		
	TCEP	**TCPP**	**TDCP**
Urine	93%	67.2%	43.2%
Faeces	5.6%	22.2%	39.2%
Expired air	1.7%	7.7%	16.24%
Carcass	0.8%	0.7%	2.51%
Total	101.5%	97.8%	101.8%

From this study, TCEP appears to be excreted to a higher degree in urine than either TCPP or TDCP. The distribution of radioactivity between urine and faeces is more evenly balanced for TDCP. For TCPP, the distribution appears to be mid-way between TCEP and TDCP, with the majority of TCPP (67%) excreted in urine but a significant amount (22%) is also excreted in faeces.

The biliary excretion of radioactivity for TDCP and TCPP was found to be comparable, 40% for TDCP and 45% for TCPP (compared with 25% for TCEP). The biliary: faecal ratios at 48 hours for TCEP, TCPP and TDCP were determined to be 4.62, 2.23 and 1.04, respectively. A ratio of greater than 1 indicates re-absorption of biliary metabolites from the gastrointestinal tract and therefore it is anticipated that that some degree of enterohepatic re-circulation of TCEP occurs, and to a lesser extent with TCPP. This would prolong the half-life of both substances in plasma. TDCP, in comparison, is expected to exhibit only limited enterohepatic recirculation and would therefore be expected to have a shorter half-life.

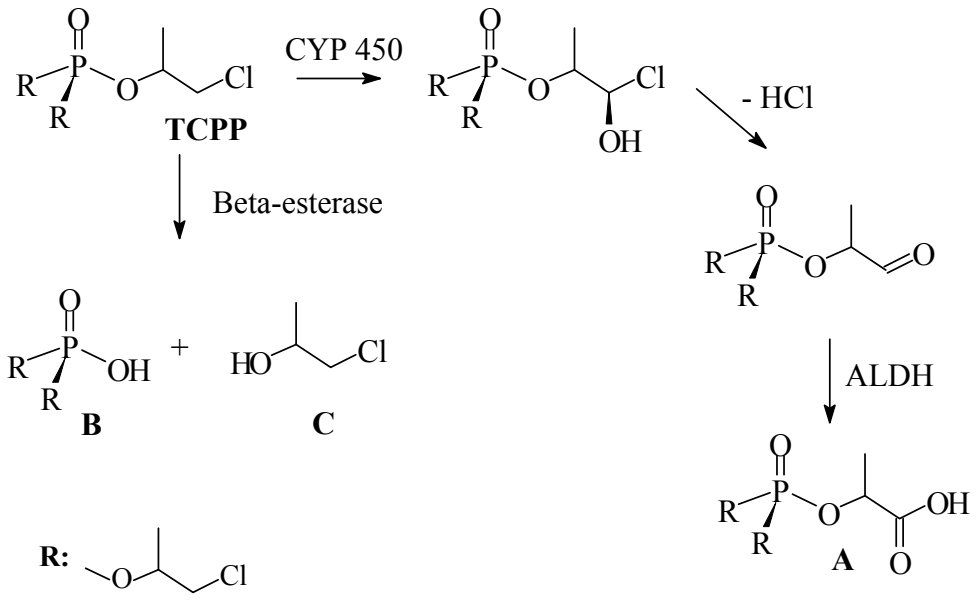
The distribution profiles of the substances differ slightly. Minegishi *et al.,* (1988) found that at 72 hours after oral administration the distribution of TCEP in tissues was kidney > liver > blood > spleen. TCPP and TDCP had similar distribution profiles: liver > kidney > lung for TCPP and liver > kidney > adipose > blood for TDCP. At 7 days after dosing, the tissue distribution for TCEP was comparable to the other two substances: liver > kidney> blood=lung. In a study by Nomeir *et al*. in 1981 (HSA/EA, 2008a), the distribution of TDCP 24 hours following oral administration was kidney > liver > lung > blood > muscle.

Metabolism

Phosphate esters behave similarly to carboxylic acid esters and as such can undergo several main reaction mechanisms such as: hydrolysis at the acyl carbon (or "P=O" bond), which can be acid catalysed or base promoted, nucleophilic substitution at the acyl carbon, as well as alkylation reactions via S_N2 at the alkyl C adjacent to the ester O.

As discussed in section 4.1.2.1.1 of the main report, following oral administration to rats, TCPP was extensively metabolised prior to excretion in urine and faeces, with unchanged TCPP representing less than 2% of the administered dose. The metabolites identified were 0,0-[bis(1-chloro-2-propyl)]-0-(2-propionic acid)phosphate (> 50%), bis(1-chloro-2-propyl)monophosphoric acid (12%) and 1-chloro-2-propanol (not quantified) (Stauffer Chemical Co, 1984). A proposed metabolic pathway for TCPP is shown in **Figure D.1** below.

Figure D.1 A proposed metabolic pathway for TCPP

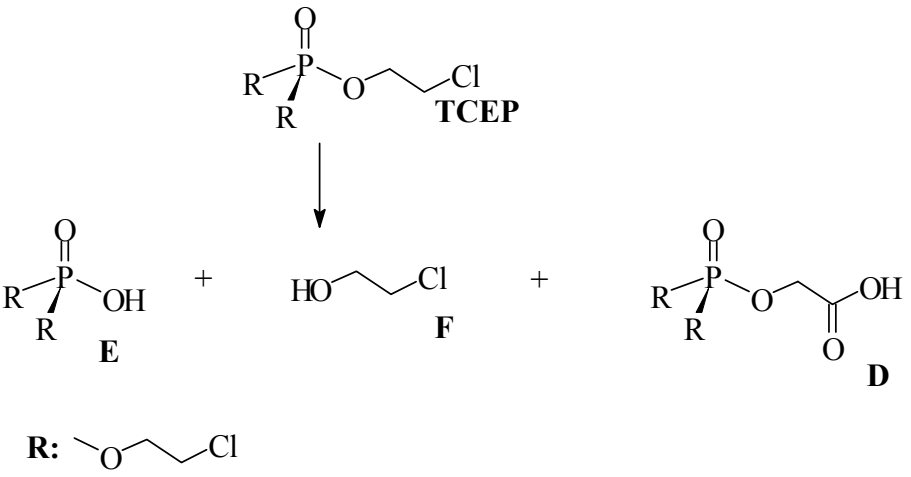


A: 0,0-[bis(1-chloro-2-propyl)]-0-(2-propionic acid)phosphate (>50%)
B: bis(1-chloro-2-propyl)monophosphoric acid (12%)
C: 1-chloro-2-propanol (not quantified)

From the metabolites identified it can be postulated that β-esterases catalyse the hydrolysis of TCPP to form metabolites B and C, while a second pathway mediated by cytochrome P450 enzymes results in aldehyde dehydrogenase (ALDH) oxidation reaction to form metabolite A.

The metabolism of TCEP has been investigated both *in vivo* and *in vitro* (BAUA, 2006). Following oral administration of ^{14}C-TCEP to rats and mice the following metabolites in urine were identified but not quantified: bis(2-chloroethyl)carboxymethylphosphate, bis(2-chloroethyl)hydrogen phosphate and bis (2-chloroethyl-2-hydroxyethyl-phosphate glucuronide (BAUA 2006). The structures and a proposed similar metabolic pathway to TCPP are presented in **Figure D.2** below. The presence of metabolites bis(2-chloroethyl)carboxymethylphosphate and bis(2-chloroethyl)hydrogen phosphate indicates that a similar metabolic pathway to TCPP may operate for TCEP: acyl-like hydrolysis at "P=O" bond cleaving a chloroalkyl chain and also metabolism via Cytochrome P450 enzymes.

Figure D.2 A proposed metabolic pathway for TCEP



D: Bis(2-chloroethyl)carboxymethyl phosphate
E: Bis(2-chloroethyl)hydrogen phosphate

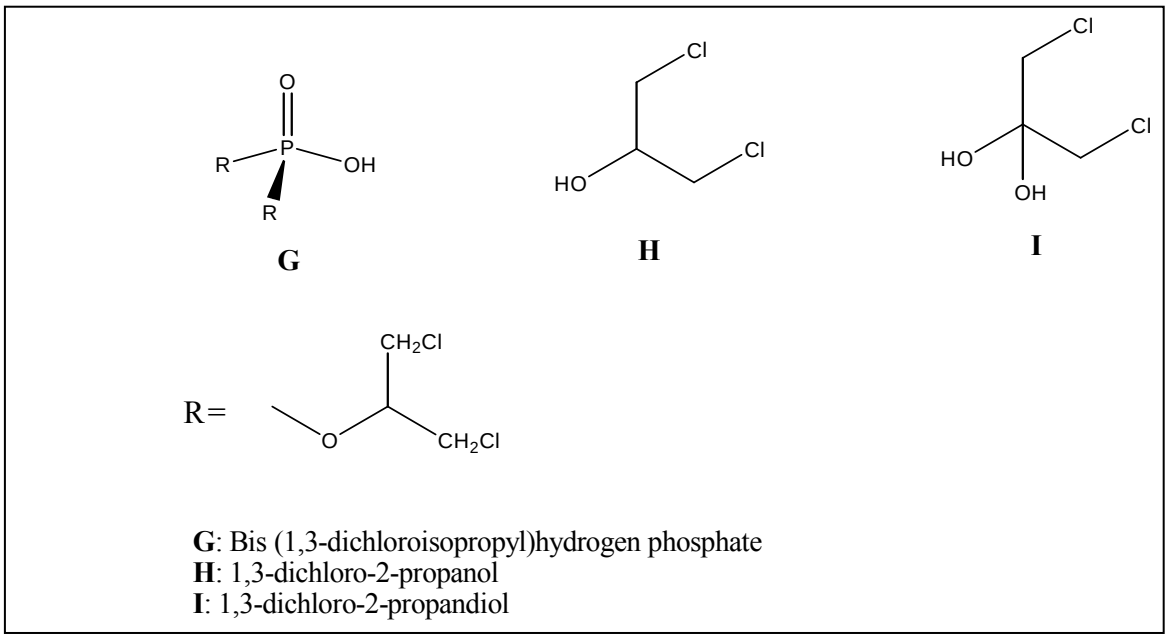
F: 2-chloroethanol

In *in vitro* metabolism studies of ^{14}C-TCEP in liver slices and liver microsomes, bis(2-chloroethyl)hydrogen phosphate and 2-chloroethanol were identified as the main metabolites (BAUA 2006) again supporting the hypothesis that a similar metabolic pathway to TCPP exists for TCEP.

No analogue of the TCEP metabolite bis (2-chloroethyl-2-hydroxyethyl-phosphate) glucuronide was identified in the *in vivo* metabolism study with TCPP, indicating there may be some differences in the metabolism of the two substances. However, as the metabolites of TCEP were not quantified it is not clear how significant this metabolite is.

The metabolism of TDCP has been investigated *in vitro* and also *in vivo* following intravenous administration (HSA/EA, 2008a). *In vitro*, mixed function oxidases (MFO) in microsomes of rat liver homogenate appear to play an important role in the metabolism of TDCP. The metabolite bis(1,3-dichloroisopropyl)hydrogen phosphate accounted for 75% of the MFO-metabolised TDCP (HSA/EA, 2008a). TDCP was also shown to be metabolised by glutathione-S-transferase present in the soluble fraction of rat liver, and it appears that TDCP is directly conjugated with glutathione. In a separate *in vitro* study, the metabolism of TDCP in the soluble fraction resulted in almost exclusively in one metabolite, which is possibly a γ-glutamylcysteinyl conjugation product of the parent TDCP. The following metabolites were also generated by the microsomal fraction of liver homogenate: bis(1,3-dichloro-2-propyl) phosphate (64 % of total metabolites), 1,3-dichloro-2-propanediol (20%), 1,3-dichloro-2-propanol (5.7 %) and an unknown metabolite (11 %). The structures are presented in **Figure D.3** below.

Figure D.3 TDCP metabolites



G: Bis (1,3-dichloroisopropyl)hydrogen phosphate
H: 1,3-dichloro-2-propanol
I: 1,3-dichloro-2-propandiol

Following i.v. administration, the metabolites isolated from rat urine were bis(1,3-dichloro-2-propyl) phosphate (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 presence of a glutathione conjugate of TDCP *in vitro* indicates a difference in the metabolism of TDCP, when compared with either TCPP or TCEP. However, the identification of bis(1,3-dichloro-2-propyl) phosphate, 1,3-dichloro-2-propanediol and 1,3-dichloro-2-propanol metabolites of TDCP points towards a similar acyl-like hydrolysis at "P=O" bond to that described for TCPP and TCEP, above. However, there does not appear to be an equivalent CYP 450 mediated reaction for TDCP.

An *in vitro* comparative metabolism study was carried out with TCPP, TDCP and TCEP (BASF Aktiengesellschaft, 2007). Two assays were performed: in the first, ^{14}C-TCPP, ^{14}C-TCEP and ^{14}C-TDCP were incubated in rat liver S9 fraction for four 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.

The mean metabolic turnover of 14C-TCPP in S9 fraction and liver slices was 89% and 61%, respectively. The results indicate that TCPP was metabolised to a hydroxylated metabolite by chlorine substitution in liver S9 fraction followed by glucuronic acid conjugation in liver slices. 11% and 39% of unmetabolised parent compound was detected in S9 fraction and liver slices, respectively.

TCEP was poorly metabolised, with a metabolic turnover of 9% and 5% in S9 fraction and liver slices, respectively. A metabolite derived from hydrolysis of one phosphoric ester groups was identified as the main metabolite. 91% and 95% of unmetabolised parent compound was detected in S9 fraction and liver slices, respectively. 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.

The study authors comment that the results of the study show that the biological behaviour of TCPP, TCEP and TDCP are not similar. However, it should be noted that the metabolites identified in this study are not completely consistent with those identified in other studies, particularly in the existing *in vivo* studies.

From the available information, it can be concluded that there is some similarity in the metabolic pathways of the three substances, although metabolism of the substances does not result in identical metabolites but rather analogous metabolites. The presence of conjugated metabolites (glurcuronide conjugate for TCEP and glutathione conjugate for TDCP) indicates that the metabolic pathways for the three substances are not identical, but, as neither conjugated metabolite was quantified, the overall impact of each metabolite on the toxicity of the substance is not known. Overall it can be concluded that there is sufficient similarity in the metabolism of the substances to support a read-across.

5. Carcinogenicity

As discussed in section 4.1.2.7 of the main report, it is accepted that TCPP is not genotoxic *in vivo*. However, there are no carcinogenicity data available for TCPP. Of the identified metabolites of TCPP, carcinogenicity data are available only for 1-chloro-2-propanol, which has been evaluated in 2-year carcinogenicity studies in both rats and mice. There was no evidence of a carcinogenic effect in either species (NTP 1998).

TCEP and TDCP both have an agreed classification of Carc. Cat 3 R40 "Limited evidence of a carcinogenic effect"[26].

TCEP administered orally to rats and mice for 2 years resulted in an increased incidence of neoplastic lesions (BAUA, 2006). In rats, the main target organ was the kidney, where there was an increase in the incidence of both proliferative lesions and adenomas of the renal tubule, which correlates with the distribution to this organ in the toxicokinetic studies. There was also an increased incidence of thyroid follicular cell neoplasms, which were possibly treatment related, and an increase in mononuclear cell leukaemia. In mice, the main target organ was the kidney, where there was a marginal increase in the incidence of renal tubule neoplasms in males at the highest dose (350 mg/kg). There was also an increase in Harderian gland adenomas in females. In an 18 month dietary study in mice, an increased incidence of renal tumours was observed, in addition to an increased incidence of tumours in the liver.

TCEP is not mutagenic *in vivo* and is therefore considered to be a non-genotoxic carcinogen (BAUA, 2006). A number of possible mechanisms were hypothesized in the TCEP risk assessment report for the formation of kidney tumours observed in the carcinogenicity studies, including biotransformation of TCEP metabolites in the kidney to nephrotoxic species. However, the mode of TCEP tumour formation has not been elucidated (BAUA 2006). Of the identified metabolites of TCEP, carcinogenicity data are only available for 2-chloroethanol. In 2-year dermal studies in rats and mice, no evidence of carcinogenicity in either species was found (NTP 1985). Therefore, it is not possible to attribute the tumours observed following TCEP administration to one particular metabolite.

TDCP is also considered to be a non-genotoxic carcinogen. In a 2 year oral carcinogenicity study in rats an increase in the incidence of renal cortical and hepatocellular adenomas in both sexes were observed, in addition to benign testicular cell tumours and Leydig cell tumours in

[26] 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.

males. The LOAEL of 5 mg/kg/day derived from the study was based on the observed hyperplasia of the convoluted tubule epithelium of the kidney (HSA/EA, 2008a). It is generally accepted that hyperplasia is a pre-neoplastic lesion, leading to tumour formation. However, it is not clear whether the hyperplasia observed following treatment with TDCP would progress to cancer or whether the kidney tumours observed with TDCP arise through a different mechanism. One possible mechanism of tumour formation involves the further metabolism of the glutathione conjugated metabolite by the brush border enzymes of the kidney to yield cytotoxic species. The resulting sustained cytotoxicity leads to compensatory tissue repair and cell proliferation, and the formation of renal tumours. However, this possible mechanism has not been confirmed for TDCP, although it is acknowledged that the absence of a similar glutathione conjugated metabolite for TCPP precludes this mechanism applying to TCPP. No specific mechanisms have been identified for the formation of any of the tumours observed in the study.

Of the identified metabolites of TDCP, carcinogenicity data are available for 1,3-dichloro-2-propanol. Following administration in the drinking water of rats for 104 weeks, a statistically significant increase in the incidence of hepatocellular adenomas and carcinomas, squamous cell papillomas and carcinomas of the tongue/oral cavity and thyroid follicular cell adenomas and carcinomas were noted (NTP, 2005). The available mutagenicity data is not sufficient to rule out a genotoxic mechanism for the induction of the tumours of the rat tongue, although it would appear that non-genotoxic mechanisms are responsible for the other tumours observed (COC, 2004). 1,3-dichloro-2-propanol is listed on Annex I to 67/548/EEC as Carc. Cat 2 R45. There are no carcinogenicity data available for the other identified metabolites. Therefore, it is not possible to attribute the tumours observed in the study to one particular metabolite.

There are sufficient data available to conclude that TCEP and TDCP are non-genotoxic carcinogens. Although the target organs for TCEP and TDCP differ, no specific mechanism of tumour formation has been elucidated for either substance. Therefore, whatever mechanisms exist for TCEP and TDCP may also exist for TCPP.

6. QSAR estimates

The carcinogenic potential of TCEP, TDCP and TCPP were estimated using a number of QSAR models – TOPKAT, Danish EPA QSAR database, OncoLogic™ and Derek for Windows (Patlewicz *et al.,* 2007). For TOPKAT, TCEP is in the NTP training set of the model and is predicted to be positive. TCPP and TDCP have conflicting species predictions. TDCP is predicted to be a carcinogen in male rat, but predictions in female rat and male mouse are outside the applicability domain of the model and therefore unreliable. TCPP is predicted to be a carcinogen in male species but not female species (Patlewicz *et al.,* 2007). Based on the conflicting species and sex predictions, it is considered that TOPKAT predictions for these substances are unreliable.

MCASE carcinogenicity predictions were extracted from the Danish EPA QSAR database (Patlewicz *et al.,* 2007). Overall, predictions generated indicate that TCEP is a carcinogen, although it should be noted that it is possible TCEP is the training set of the model (this could not be verified). TCPP is not predicted to be a carcinogen. TDCP is predicted to be a carcinogen, although a number of the predictions were outside the model domain.

OncoLogic™, which was run in the default mode and so did not make use of the available experimental data (e.g. mutagenicity, physical chemical properties) on the substances, predicted the final level of concern for TCEP as "low-moderate", but again it should be noted

that TCEP is in OncoLogic's training set. TDCP and TCPP both had predictions of "moderate" level of concern. (Patlewicz *et al.,* 2007).

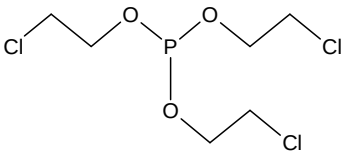
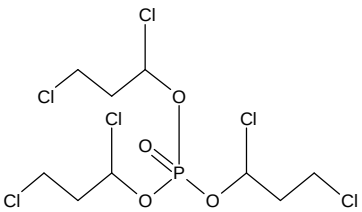
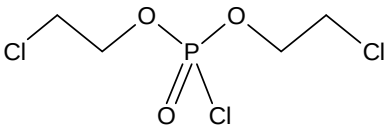
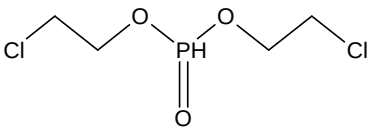
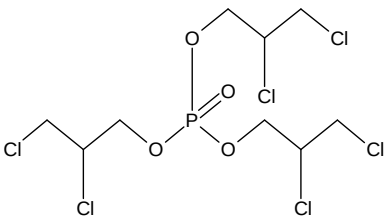
Derek for Windows produced "plausible" alerts for carcinogenicity (alkylating agent with $-CH_2Cl$), chromosomal damage (*in vitro*) and mutagenicity (*in vitro)* for all three substances, with little differentiation between the substances (Patlewicz *et al.,* 2007).

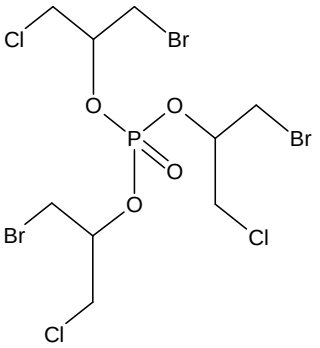
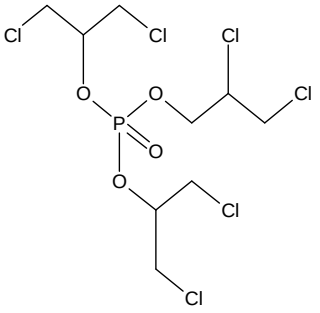
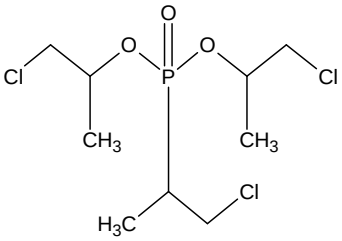
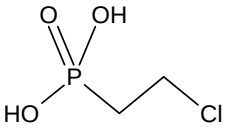
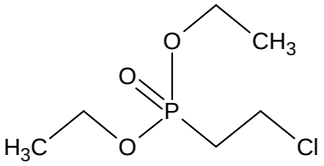
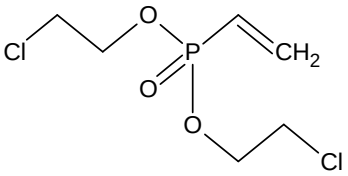
As there were some inconsistencies between the predictions generated by the different models, and also when the predictions were compared with the available experimental data for TCEP and TDCP, it is not possible to draw any definitive conclusions from these predictions with respect to the carcinogenicity of TCPP.

7. Comparison with other potential analogues

A search was conducted using Leadscope software to identify other potential structural analogues of TCPP, which could be used to support a read-across for this endpoint (Patlewicz *et al.,* 2007). **Table D.4** below summarises the closest structural analogues to TCPP (Tanimoto similarity of 40%). However, none of the identified substances had available carcinogenicity data and therefore cannot be used as analogues in a read-across for this endpoint.

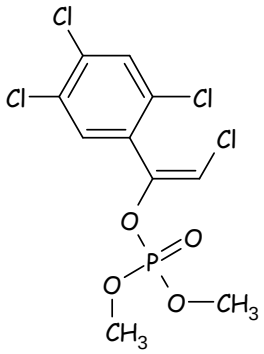
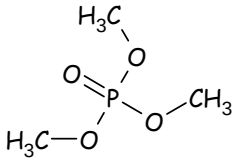
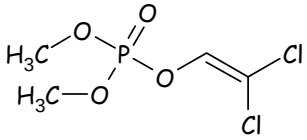
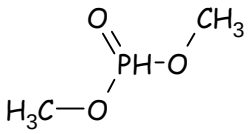
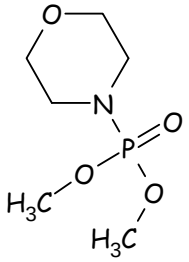
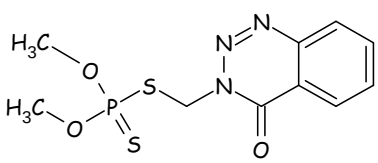
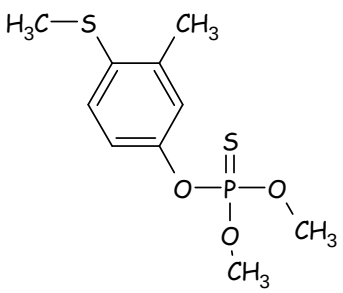
Table D.4 Structurally similar analogues of TCPP without carcinogenicity data identified by Leadscope

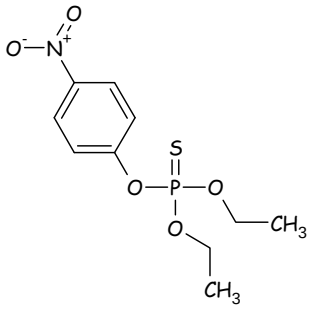
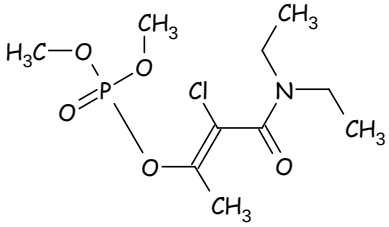
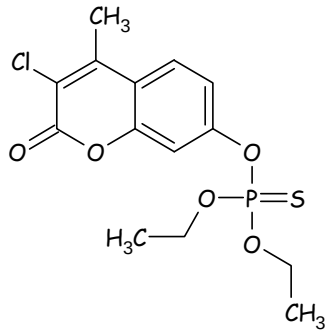
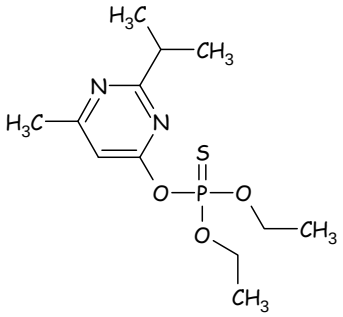
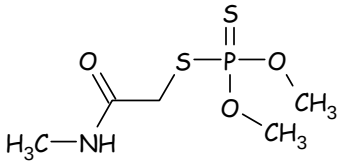
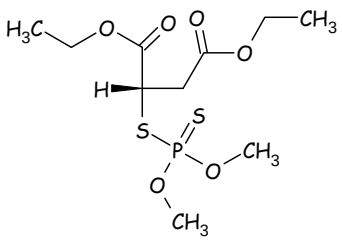
Structure	CAS	Name/SMILES	Studies available
	140-08-9	Tris(2-chloroethyl) phosphate/ ClCCOP(OCCCl)OCCCl	acute toxicity, irritation, multiple dose, RTECS mutation
	40120-74-9	Tris(1,3-dichloropropyl)phosphate ClCCC(Cl)OP(=O)(OC(Cl)CCCl)OC(Cl)CCCl	No
	6087-94-1	Bis(2-chloroethyl) chlorophosphate/ ClCCOP(Cl)(=O)OCCCl	No
	1070-42-4	Di-2-chloroethyl phosponate/ ClCCOP(=O)OCCCl	No
	78-43-3	Tris(2,3-dichloropropyl) phosphate ClC(COP(=O)(OCC(Cl)CCl)OCC(Cl)CCl)CCl	acute toxicity, multiple dose, RTECS mutation

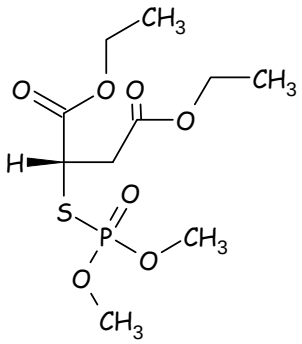
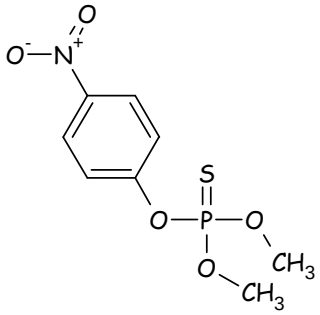
Structure	CAS	Name/SMILES	Studies available
	7328-28-1	Tris(1-bromo-3-chloroisopropyl) phosphate BrCC(OP(=O)(OC(CBr)CCl)OC(CCl)CBr)CCl	No
	68460-03-7	Phosphoric acid, bis[2-chloro-1-(chloromethyl)ethyl] 2,3-dichloropropyl ester ClCC(OP(=O)(OC(CCl)CCl)OCC(Cl)CCl)CCl	No
	7316-55-4	Phosphonic acid, (2-chloro-1-methylethyl)-, bis(2-chloro-1-methylethyl) ester (7Cl,8Cl,9Cl) ClCC(C)OP(=O)(OC(C)CCl)C(C)CCl	Acute toxicity
	16672-87-0	(2-Chloroethyl)phosphonic acid ClCCP(O)(O)=O	Acute toxicity, multiple dose
	10419-79-1	Diethyl (2-chloroethyl)phosphonate O=P(CCCl)(OCC)OCC	Acute toxicity
	115-98-0	Bis(2-chloroethyl) vinylphosphonate ClCCOP(=O)(OCCCl)C=C	Acute toxicity

In addition, a search was also conducted using Leadscope to identify the closest structural analogues to TCPP for which carcinogenicity data are available. The results are presented in **Table D.5**, below. (Patlewicz *et al* .,2007). However, none of the substances were considered to be sufficiently structurally similar to TCPP to be used as a valid analogue for read-across and therefore, these substances were not taken into account in the weight of evidence approach.

Table D.5 Structurally similar substances with carcinogenicity data identified using Leadscope

Structure	Name/SMILES	CAS	Sal	MR	FR	MM	FM
	2-Chloro-1-(2,4,5-trichlorophenyl)vinyl dimethyl phosphate; Tetrachlorvinphos Clc1cc(C(=C\Cl)/OP(=O)(OC)OC)c(Cl)cc1Cl	961-11-5	N	N	P	P	P
	Trimethyl phosphate O=P(OC)(OC)OC	512-56-1	P	P	N	N	P
	Phosphoric acid, 2,2-dichloroethenyl dimethyl ester; Dichlorvos O=P(O\C=C(/Cl)Cl)(OC)OC	62-73-7	P	N	N	N	N
	Dimethyl hydrogen phosphite O=P(OC)OC	868-85-9	P	CE	EE	NE	NE
	Dimethylmorpholinophosphoramidate COP(=O)(OC)N1CCOCC1	597-25-1	N	SE	SE	NE	NE
	Phosphorodithioic acid, O,O-dimethyl S-((4-oxo-1,2,3-benzotriazin-3(4H)-yl)methyl) ester; Azinphos-methyl COP(=S)(OC)SCN1\N=N/c2ccccc2C1=O	86-50-0	P	E	N	N	N
	Fenthion; O,O-Dimethyl O-4-methylthio-m-tolyl phosphorothioate; Phosphorothioic acid, O,O-dimethyl O-(4-(methylthio)-m-tolyl) ester Cc1cc(ccc1SC)OP(=S)(OC)OC	55-38-9	P	N	N	E	N

	Parathion; Phosphorothioic acid, O,O-diethyl O-(p-nitrophenyl) ester S=P(Oc1ccc(cc1)[N+]([O-])=O)(OCC)OCC	56-38-2	P	E	E	N	N
	Phosphamidon; Phosphoric acid, 2-chloro-3-(diethylamino)-1-methyl-3-oxo-1-propenyl dimethyl ester; Phosphoric acid, dimethyl ester, ester with 2-chloro-N,N-diethyl-3-hydroxycrotonamide O=P(OC(/C)=C(\Cl)C(=O)N(CC)CC)(OC)OC	13171-21-6	P	E	E	N	N
	Coumaphos; O-3-Chloro-4-methyl-7-coumarinyl O,O-diethyl phosphorothioate; Phosphorothioic acid, O-(3-chloro-4-methyl-2-oxo-2H-1-benzopyran-7-yl) O,O-diethyl ester CCOP(=S)(OCC)Oc1ccc2c(c1)OC(=O)C(/Cl)=C2/C	56-72-4	N	N	N	N	N
	Diazinon; Phosphorothioic acid, O,O-diethyl O-(2-isopropyl-6-methyl-4-pyrimidinyl) ester; O,O-Diethyl 2-isopropyl-6-methyl-4-pyrimidinylphosphorothioate Cc1cc(OP(=S)(OCC)OCC)nc(n1)C(C)C	333-41-5	N	N	N	N	N
	Dimethoate COP(=S)(OC)SCC(=O)NC	60-51-5	P	N	N	N	N
	Malathion COP(=S)(OC)S[C@@H](CC(=O)OCC)C(=O)OCC	121-75-5	N	N	N	N	N

	Malaoxon COP(=O)(OC)S[C@@H](CC(=O)OCC)C(=O)OCC	1634-78-2	N	N	N	N	N
	Methyl parathion; Phosphorothioic acid, O,O-dimethyl O-(p-nitrophenyl) ester S=P(Oc1ccc(cc1)[N+]([O-])=O)(OC)OC	298-00-0	N	N	N	N	N

Where N= Negative, P = Positive, MR = Male Rat, FR = Female Rat, MM = Male Mouse, FM = Female Mouse, CE = Clear Evidence of Carcinogenicity, EE = Equivocal Evidence, NE = No Evidence, SE = Some Evidence, NT = Not Tested

As there were no other structurally similar analogues identified for which carcinogenicity data are available, it is therefore concluded that TCEP and TDCP are the most appropriate analogues of TCPP for read-across.

8. Qualitative versus quantitative read-across

As discussed above, there are no carcinogenicity data for TCPP, and it is accepted that TCPP is not-genotoxic *in vivo*.

As described in section 4.1.2.6 of the main report, the study of longest duration for TCPP is a 90-day dietary study in rats. Increased liver weights were observed in males at 52 mg/kg and above and periportal hepatocyte swelling was noted at highest dose (1349 mg/kg in males and 1745 mg/kg in females). In addition, mild follicular cell hyperplasia was noted in females at 1745 mg/kg and in all dosed males. In the kidney, vacuolation in females at highest dose were also observed. A slightly excessive fatty infiltration indicative of mild bone marrow hypoplasia was noted in three high dose females. The selected LOAEL of 52 mg/kg/day is based on increased liver weights observed in males. As a reasonable worst case approach, in the absence of carcinogenicity data for TCPP, it cannot be excluded that the effects observed in this study with TCPP could progress to cancer via a non-genotoxic mechanism.

In addition, it is considered that there is sufficient information from the structures, physical-chemical properties, toxicokinetics and mutagenic profiles of TCEP, TDCP and TCPP to support a qualitative read-across for carcinogenicity.

However, based on the available data, there are some differences in the metabolism and target organs of the three substances, which indicate that a quantative read-across for carcinogenicity from either TDCP or TCEP to TCPP may not be appropriate.

When the study of longest duration for TCPP (90-day) is compared with studies of similar duration for TCEP, there is some difference in the potency and severity of the effects seen between the two substances. In a 16 week oral gavage study in rat with TCEP, the most relevant toxic effects observed were mortality at highest dose (350 mg/kg) and brain lesions in females at 175 mg/kg and above (BAUA, 2006). Although an increase in relative kidney and liver weights was observed, no corresponding histopathological effects were seen in these organs. The NOAEL identified was 88 mg/kg, based on neuronal effects. In a second 3 month dietary study in rats, an increased incidence of regenerative hyperplasia in renal cortex was observed in both sexes at the highest dose (506 mg/kg males and 586 mg/kg females). The NOAEL identified was 192 mg/kg/day (BAUA, 2006). Based on the above, there appears to be a difference in target organs and severity of effects between TCPP and TCEP.

No study of similar duration is available for TDCP, although in the 2-year carcinogenicity study, the liver and kidney were identified as target organ for TDCP (HSA/EA 2008a). The LOAEL of 5 mg/kg/day was based on hyperplasia observed in the kidney and testicular effects observed at this dose. There is greater than an order of magnitude difference in potency between the two substances, which is too large to be explained by differences in the study durations alone. Therefore, it is concluded that a direct read-across from TDCP is not possible.

Therefore, differences in the target organs, the severity of the effects observed and the potency of the three substances also indicates that a direct read-across to carcinogenicity data on TCEP or TDCP is not appropriate.

A summary of the available repeat dose toxicity data for TCEP, TDCP and TCPP is presented in **Table D.6** below.

9. Conclusion

As discussed above, TCPP, like TDCP and TCEP is not genotoxic *in vivo*. Based on the available repeat dose toxicity data for TCPP and a qualitative read-across from TDCP and TCEP, there is a potential concern for carcinogenicity for TCPP by a non-genotoxic mechanism. No quantitative read-across can be performed since there are no insights into an underlying mode of action for TCEP and TDCP which would make a prediction on a relatively potency of TCPP possible.

It is proposed that the effects observed in the 90-day study for TCPP are taken as a starting point for risk characterisation. If these effects were to progress to cancer, they would do so by a non-genotoxic mechanism. Therefore, it is proposed that the LOAEL, of 52 mg/kg/day, identified from the 90-day study with TCPP should be used as a basis for risk characterisation of the carcinogenicity endpoint.

Table D.6 Summary of the available repeat dose toxicity data for TCEP, TCPP and TDCP

Study type	TCEP	TCPP	TDCP
14-day (oral)			
Species	Rat	Rat	No study available
Dose	0,22,44,88,175, 350 mg/kg	417, 648, 1015, 1636 mg/kg (M) 382, 575, 904, 1517 mg/kg (F)	
NOAEL	350 mg/kg	1015 mg/kg	
Target organs/ effects:	-Increase kidney weight at ≥ 175 -Increase liver weight at 350	-Decrease bw gain	
14-day (oral)			
Species	Mouse		
Dose	0, 44, 88, 175, 350, 700 mg/kg		
NOAEL	175 mg/kg		
Target organs/ effects:	-ataxia and convulsive movements Days 1-3		

Study type	TCEP	TCPP	TDCP
28-day (oral)			
Species	No study available	Rat	No study available
Dose		0, 417,648,1015, 1636 mg/kg (M)	
		0,382,575,904,1517 mg/kg (F)	
NOAEL		10 mg/kg/day	
Target organs/ effects:		- Increase in liver weight high dose & liver histopathology in mid and high	
		- Decrease in ALAT at high	
90-day (oral)	(3mth dietary)		
Species	Rat	Rat	
Dose	0,26,65,192,506 mg/kg (M)	0,52,160,481, 1349 mg/kg (M)	
	0,30,75,215,586 mg/kg (F)	0,62,171,570,1745 mg/kg (F)	
NOAEL	192 mg/kg (regenerative hyperplasia in kidney)	52 mg/kg (LOAEL)	
	-Increase in kidney & liver wt at ≥ 192/215 mg/kg		
Target organs/ effects:	-Increase incidence of tubular hyperplasia at high dose	-Increase in liver weight in all treated males & liver histopath at high dose	
	- Decrease in gonad & brain wt at 2 highest doses	-Increase in kidney weight	
	-decreased heart wt at high dose	-Thyroid follicular cell hyperplasia	

Study type	TCEP	TCPP	TDCP
90-day (oral)	(3 mth dietary)		
Species	Mouse		
Dose	0,12,60,300 & 1500 mg/kg		
NOAEL	LOAEL 12 mg/kg		
Target organs/ effects:	- Decreased heart & testes wt at high dose - decreased kidney wt F at 1500 mg/kg -focal necrosis & vacuolation in liver -hypertrophy & hyperplasia of urinary tubule epithelium.		
16 weeks (oral)			
Species	Rat		
Dose	0,22,44,88,175, 350 mg/kg		
NOAEL	88 mg/kg		
Target organs/ effects:	-Increase kidney & liver wt (no histopath) at > 44 mg/kg (F) &350 mg/kg (M) - Increase in brain wt at 350 mg/kg - Neuronal necrosis hippocampus & thalamus at ≥ 175 mg/kg		

Study type	TCEP	TCPP	TDCP
16 weeks (oral)			
Species	Mouse		
Dose	0,44,88,175,350, 700 mg/kg		
NOAEL	350 mg/kg		
Target organs/ effects:	- Increase in liver wt F ≥ 175 mg/kg (no histopath) - Decrease in kidney wt M at ≥ 175 mg/kg - Histopath kidney proximal convoluted tubule - slight decrease in sperm count		
2-yr (oral)	(103 weeks)		
Species	Rat	No study available	Rat
Dose	0, 44,88 mg/kg		0,5,20,80 mg/kg
NOAEL	44 mg/kg (LOAEL kidney; NOAEL brain)		5 mg/kg (LOAEL)
Target organs/ effects:	- Increase in focal hyperplasia of renal tubule epithelium - degenerative lesions of brain stem & cerebrum		- Increase in kidney weight & hyperplasia in convoluted tubule of kidney - Increase in liver weight & liver histopath at mid & high dose - Increase in thyroid weight high dose female - Testis effects in all treated groups

Study type	TCEP	TCPP	TDCP
2-yr (oral)	(103 weeks)		
Species	Mouse		
Dose	0,175,350 mg/kg		
NOAEL	175 mg/kg (LOAEL kidney) No NOAEL for liver effects		
Target organs/ effects:	-Karyomegaly of tubule epithelium in kidney -Increased incidence of foci of cytologic alteration in liver at all doses (precursor of hepatocellular neoplasms)		

Study type	TCEP	TCPP	TDCP
2-year carcinogenicity			
Species	Rat		Rat
Route	Oral		Oral
Dose	0, 44, 88 mg/kg		0,5,20,80 mg/kg
NOAEL	None established		5 mg/kg (LOAEL)
Target organs/ effects:	- Increase in incidence of neoplastic lesions in kidney (proliferative lesions & adenomas of the renal tubule). - Increased incidence of thyroid follicular cell neoplasms (possibly treatment related) - Increase in mononuclear cell leukaemia		- Hyperplasia of convoluted tubule in all treated males & high dose females - Increase in renal cortical adenomas in mid & high dose at 24 mths -Increase in benign testicular cell tumours at mid & high dose - Increase in Leydig cell tumours in mid & high dose males. - Increase incidence of hepatocellular adenomas at high dose.

Study type	TCEP	TCPP	TDCP
2-year carcinogenicity			
Species	Mouse		
Route	Oral		
NOAEL	None established		
Dose	0,175, 350 mg/kg		
Target organs/ effects:	- marginal increase in incidence of renal tubule neoplasms at 350 mg/kg (M) - Increase in Harderian gland adenomas at 175 mg/kg (F)		
2-year carcinogenicity			
Species	Mouse		
Route	Oral (dietary)		
Dose	0, 12, 60, 300, 1500 mg/kg		
NOAEL	Kidney: ≥ 12 mg/kg (LOAEL) Liver : 60 mg/kg		
Target organs/ effects:	- Increase in incidence of tumours in liver and kidney		

European Commission

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