European Union Risk Assessment Report

PROPAN-1-OL

CAS No: 71-23-8
EINECS No: 200-746-9

RISK ASSESSMENT

GENERAL NOTE

This document contains two different reports:

-**Volume 82**, **Part I Environment** (publication EUR 22159 EN) – pages 2-103
- **Part II Human Health** (final approved version awaiting for publication) – pages 104-226

European Chemicals Bureau

Institute for Health and Consumer Protection

European Chemicals Bureau

Existing Substances

European Union Risk Assessment Report

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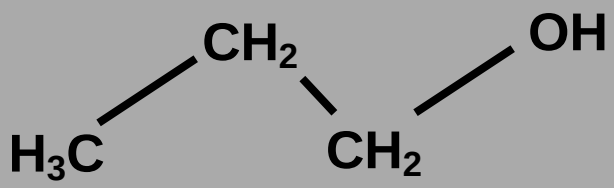
propan-1-ol
part I - environment



2nd Priority List

Volume: 82

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European Union Risk Assessment Report
propan-1-ol – part I - environment

CAS: 71-23-8
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The mission of the IHCP is to provide scientific support to the development and implementation of EU polices related to health and consumer protection. The IHCP carries out research to improve the understanding of potential health risks posed by chemical, physical and biological agents from various sources to which consumers are exposed.

The Toxicology and Chemical Substances Unit (TCS), commonly known as the European Chemicals Bureau (ECB), provides scientific and technical input and know-how to the conception, development, implementation and monitoring of EU policies on dangerous chemicals including the co-ordination of EU Risk Assessments. The aim of the legislative activity of the ECB is to ensure a high level of protection for workers, consumers and the environment against dangerous chemicals and to ensure the efficient functioning of the internal market on chemicals under the current Community legislation. It plays a major role in the implementation of REACH through development of technical guidance for industry and new chemicals agency and tools for chemical dossier registration (IUCLID5). The TCS Unit ensures the development of methodologies and software tools to support a systematic and harmonised assessment of chemicals addressed in a number of European directives and regulation on chemicals. The research and support activities of the TCS are executed in close co-operation with the relevant authorities of the EU Member States, Commission services (such as DG Environment and DG Enterprise), the chemical industry, the OECD and other international organisations.

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RISK ASSESSMENT

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RISK ASSESSMENT

Final Report, 2008

Germany

The first draft of the Comprehensive Risk Assessment Report of propan-1-ol, a substance chosen from the EU 2nd priority list in 1995 was distributed for the preliminary written procedure in June 2002.

The "in depth discussion" was at the Technical Meeting in March 2003 (TM I'03).

This document is a revised draft of the environmental part of the Risk Assessment Report which is intended to be discussed as "final written approval" at the Technical Meeting in September 2003 (TM III´03).

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Final report:	**2008**

Foreword

We are pleased to present this Risk Assessment Report which is the result of in-depth work carried out by experts in one Member State, working in co-operation with their counterparts in the other Member States, the Commission Services, Industry and public interest groups.
The Risk Assessment was 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 Health and Environmental Risks (SCHER) which gives its opinion to the European Commission on the quality of the risk assessment.
If a Risk Assessment Report concludes that measures to reduce the risks of exposure to the substances are needed, beyond any measures which may already be in place, the next step in the process is for the "Rapporteur" to develop a proposal for a strategy to limit those risks.
The Risk Assessment Report is also presented to the Organisation for Economic Co-operation and Development as a contribution to the Chapter 19, Agenda 21 goals for evaluating chemicals, agreed at the United Nations Conference on Environment and Development, held in Rio de Janeiro in 1992 and confirmed in the Johannesburg Declaration on Sustainable Development at the World Summit on Sustainable Development, held in Johannesburg, South Africa in 2002.
This Risk Assessment improves our knowledge about the risks to human health and the environment from exposure to chemicals. We hope you will agree that the results of this in-depth study and intensive co-operation will make a worthwhile contribution to the Community objective of reducing the overall risks from exposure to chemicals.

Roland Schenkel
Director General
DG Joint Research Centre

Mogens Peter Carl
Director General
DG Environment

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[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]

0 OVERALL RESULTS OF THE RISK ASSESSMENT

CAS No:	71-23-8
EINECS No:	200-746-9
IUPAC Name:	propan-1-ol

Overall results of the risk assessment:

Conclusion (i) There is 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.

Summary of conclusions:

Environment

From the intrinsic properties it is expected that propan-1-ol is of low concern for the environment. The environmental risk assessment was performed, using conservative estimates based on worst-case assumptions at the exposure and effects side. The risk assessment results in the following conclusion:

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.

Based on the currently available data, propan-1-ol represents no risk to the environment for the area of production, processing, formulation and use.

Human Health

Workers

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

For mutagenicity the base set data have to be completed, risk assessment concerning carcinogenicity will be delayed until the mutagenicity data are available.

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

Summary of conclusions:

There is a need for limiting the risks of propan-1-ol for several scenarios with short-term and repeated exposures. The most critical exposure route is inhalation, dermal contact being of minor importance. In detail concern is expressed for use of paints, use of cleaning formulations without LEV, short term exposures during use of printing inks.

The toxic effects leading to concern are respiratory depression according to stimulation of the trigeminus nerve, local effects in the airways after repeated exposure and reproductive toxicity concerning fertility as well as developmental toxicity. Risk reduction measures especially for the inhalative exposure situation have to be initiated.

Consumers

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

Mutagenicity

The minimum requirements in mutagenic testing are not met. An *in vitro* study on chromosome aberration in Chinese hamster cells is currently ongoing.

The producer has to be requested to make available existing studies.

Carcinogenicity

There is no valid carcinogenicity study available. The present data base gives no indication for carcinogenic effects. For performing the risk assessment on carcinogenicity, however, the completed data on mutagenicity have to be taken into account.

Humans exposed indirectly 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.

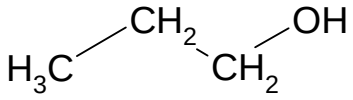
CONTENTS

TABLES

1 GENERAL SUBSTANCE INFORMATION

1.1 IDENTIFICATION OF THE SUBSTANCE

CAS No:	71-23-8
EINECS No:	200-746-9
IUPAC Name:	Propan-1-ol
Synonyms:	1-hydroxypropane, 1-propanol, ethylcarbinol, n-propanol, n-propyl alcohol, propanol-1, alcohol C_3
Empirical formula:	C_3H_8O
Molecular weight:	60.1 g/mol
Structural formula:	



1.2 PURITY/IMPURITIES, ADDITIVES

Purity:	> 99%
Impurities:	methanol
	ethanol
	C6 aldehydes
	propyl propionate
	2-methylvaleraldehyde
	≤ 0.2% w/w aldehyde
	< 0.1 w/w dipropyl ether
	≤ 0.1% w/w water
	≤ 0.003% w/w acetic acid

Table 1.1 Physico-chemical properties

Property	Result	References
Physical state	clear colourless liquid with characteristic odour	
Melting point	-126.5°C	CRC Handbook (1991/92)
Boiling point	97.1°C at 1,013 hPa	Hiaki et al. (1994)
Density	0.803 g/cm^3 at 20°C	Wilhoit and Zwolinski (1973)
Vapour pressure	19.4 hPa at 20°C	Hiaki et al. (1994)
Surface tension	67.1 mN/m at 25°C c=1 g/l	CRC Handbook (1991/92)
Partition coefficient	0.34 (shake flask method)	Hansch and Anderson (1967)
Water solubility	completely soluble	Yaws et al. (1990)
Flash point	22°C (corrected to the presence of iso-propanol) 23.5°C (99.9% pure)	CHEMSAFE DIN 51755, ISO 3679
Auto flammability	385°C	CHEMSAFE DIN 51794
Flammability	flammable	CHEMSAFE
Explosive properties	not explosive	due to structural reasons
Oxidising properties	no oxidising properties	due to structural reasons

Vapour pressure

The values given for the vapour pressure at 20°C vary between 19 and 20.3 hPa. In the safety data sheet of the BASF AG a value of 19.4°C is quoted, in the data sheet of the Hoechst AG the vapour pressure is quoted with 20 hPa. In both cases no other information is given. Also without any further information Sasol has quoted a value of 20 hPa at 20°C. For the risk assessment the value of 19.4 hPa at 20°C is recommended. This value is derived from the Antoine equation determined by Boublik T, Fried V and Hala E (1984).

Partition coefficient n-octanol/water

The values for the partition coefficient n-octanol/water are varying between 0.25 and 0.38. The safety data sheets of the BASF AG, Hoechst AG and Union Carbide are quoting values between 0.25 and 0.34 without further information. Furthermore the partition coefficients are calculated. The following values are found: 0.271 (according to Rekker with program PRO-LOGP, ver.2 from CompuDrug Ltd.), 0.38 (Abraham MH, Chadha HS, Whiting GS and Mitchell RC (1994)). Further undocumented values are quoted by Petrasol BV, Gorinchem and BASF AG (1989): Labor fuer Umweltanalytik by 0.25 and 0.271, respectively. Other values from literature are in the above mentioned range.

For risk assessment the value of 0.34 of Hansch C and Anderson SM is recommended. The authors have great experience in the field of measuring and calculating octanol/water partition coefficients. They used some kind of shake flask method (Hansch C and Anderson SM (1967)).

Flash point

The value of 23.5°C was determined for n-propanol with a purity of 99.9%. The tests were conducted according to DIN 51755 (Testing of mineral oils and other combustible liquids; determination of flash point by the closed tester according to Abel-Pensky) and ISO 3679 (Paints, varnishes, petroleum and related products - determination of flashpoint – rapid equilibrium method). The value of 23.5°C was corrected for commercial n-propanol to 22°C because of iso-propanol which is usually present as an impurity.

1.3 CLASSIFICATION

- (Classification according to Annex I)[4]

Highly flammable	R 11	Highly flammable
Irritant	R 41	Risk of serious damage to eyes
	R 67	Vapours may cause drowsiness and dizziness

- (Proposal of the rapporteur)

Flammable	R 10	Flammable
Irritant	R 41	Risk of serious damage to eyes

A value of 23.5°C was determined for the flash point of n-propanol with a purity of 99.9%. This value of 23.5°C was corrected for commercial n-propanol which usually contains iso-propanol as an impurity to 22°C.

The classification for liquid substances with a flash point between 21 and 55°C is “flammable”.

Therefore the legal classification according to Annex I for propan-1-ol which is at the moment “highly flammable” must be corrected. The original classification resulted from measurements of n-propanol contaminated with impurities (flash point < 21°C).

According to the data presented below and the criteria of Directive 93/21/EEC propan-1-ol has not to be classified as dangerous for the environment.

Propan-1-ol is classified according to water-hazard class 1 (slightly hazardous to water).

[4] The classification of the substance is established by Commission Directive 98/98/EC of 15 December 1998 adapting to technical progress for the 25th time Council Directive 67/548/EEC on the approximation of the laws, regulations and administrative provisions relating to the classification, packaging and labelling of dangerous substances (OJ L 355, 30.12.98, p.1).

2 GENERAL INFORMATION ON EXPOSURE

2.1 PRODUCTION

According to the information from the currently available IUCLID data sets there is one production site of propan-1-ol in the EU. The chemical is imported by 5 other companies from outside of the EU. There is no information on possible exports of propan-1-ol.

Based on the production and import quantities approximately 30,100 tonnes/annum of propa-1-ol are used in the EU.

Propan-1-ol is produced almost exclusively by the reaction of ethene with synthesis gas. Reaction is performed at 25-30 MPa and 140-180°C in the liquid phase in the presence of cobalt carbonyl hydrogen as catalyst. After the separation of the catalyst the raw mixture obtained can be hydrogenated in the gaseous phase on the nickel catalyst (0.2-0.3 MPa, 115°C) or on the copper catalyst (3-5 MPa, 130-160°C) and in the liquid phase (8 MPa, 115°C) on the nickel catalyst. By means of subsequent distillation the production of pure propan-1-ol is achieved (Weissermel and Arpe, 1988; Falbe et al., 1980).

2.2 PROCESSING/APPLICATION (CATEGORIES OF USE, AMOUNTS)

In Western Europe propan-1-ol is mainly used as solvent for the formulation of disinfectants, pharmaceutical products, cleaning agents, paints, coating materials, enamel and lacquer paints, printing inks and cosmetics (GDCh, 1997).

Propan-1-ol is processed chemically to intermediates such as propylamines, carboxylic acid esters and halogenated hydrocarbons, which in turn are needed for the synthesis of herbicides, aroma and perfume substances, cosmetics and pharmaceuticals (GDCh, 1997).

Of the propan-1-ol produced by BASF AG, 10% were processed to intermediates and 90% were used as solvent; application areas as solvent were paints, surface coatings and inks, cosmetics and pharmaceuticals, detergents and other (BASF, 1994).

Hoechst AG manufactured 5,000 tonnes of propan-1-ol in 1993; about 3,500 tonnes were processed to n-propylamines, 1,500 tonnes of propan-1-ol were sold. In 1995, the production of propan-1-ol was ceased; the requisite amounts of propan-1-ol for the production of the n-propylamine derivatives are supplied from Bay City, USA (Hoechst AG, 1994 and 1995b).

Based on the available information the following consumption's of propan-1-ol are estimated for Western EU (CEH, 1995):

- 55% were used as solvent, hereof:

approximately	20%	to cosmetics,
approximately	35%	pharmaceutics (disinfectants),
approximately	5%	cleaning/washing agents and
approximately	5%	to other.

- 45% were processed as an intermediate for the production of:

approximately	75%	to n-propylacetate,
approximately	20%	to propylchlorformiate and
approximately	5%	to reactive resins.

Because of the various direct applications of propan-1-ol in end products it has to be expected that the handled amount of propan-1-ol in Europe may increase through import. No further information is available on import or export, as well as on residual content of propan-1-ol in end products.

The use of propan-1-ol in cleaning agents, pesticides, thinners, paints, printing inks and solvents is described in SPIN – Substances in Preparations in the Nordic countries data base. The information contained in SPIN is listed in **Table 2.1**.

Table 2.1 Information on propan-1-ol in consumer products in the Nordic countries obtained from SPIN data base (July 2003)

Country	Year	Number of preparations	Quantity of propan-1-ol contained in preparations [tonnes]
FIN	2001	181	3,341
N	2001	110	2,675
DK	2001	208	1,925
S	2000	202	743

The main use categories of the preparations containing propan-1-ol are cleaning/washing agents (N), reprographic agents and solvents (DK), activators and dyestuffs (FIN), solvents and de-icing agents (S).There are also non-industrial sources of propan-1-ol: It is contained in landfill gas; it is formed from plants and animals through putrefaction and decomposition; alcohol-forming bacteria are involved here. The substance is contained in aromas of fruits and other foodstuffs. It is a natural component of alcoholic beverages that have been obtained through fermentation of plant raw materials (GDCh, 1997). The quantity of formed propan-1-ol and the resultant environmental concentration in the different compartments cannot be quantified. However, it is assumed that these are very low and can be neglected.

The following table shows the main, industrial and use categories and the mass balance of propan-1-ol for the EU.

Table 2.2 Use categories and mass balance of propan-1-ol

Main category (MC)	Industrial category (IC)	Use category (UC)	Mass balance [in % of use]
Non-dispersive use (3)	Chemical industry (3)	Intermediate (33)	45
Non-dispersive use (3)	Other (0)	Solvent (48)	3
Wide dispersive use (4)	Personal/domestic (5)*	Solvent (48)	33
Wide dispersive use (4)	Paint, lacquers and varnishes industry (14)	Solvent (48)	19

* Sum of quantity used as solvent for cosmetics, pharmaceutics (disinfectants) and cleaning/washing agents.

3 ENVIRONMENT

From the intrinsic properties it is expected that propan-1-ol is of low concern for the environment. Conservative estimates based on worst-case assumptions at the exposure and effects side were used. If the risk assessment performed in that way does not indicate any risk, no further work is considered to be necessary.

3.1 ENVIRONMENTAL EXPOSURE

3.1.1 General discussion

Release into the environment

During production, processing (use as an intermediate), formulation of products (containing propan-1-ol) and further use as a solvent, propan-1-ol is expected to enter the environment via the waste water and the exhaust air.

Degradation

Biodegradation

The biodegradability of propan-1-ol in water has been shown in a number of investigations under most varied conditions. However, no standardised tests for ready biodegradability are available. The most relevant test results for the risk assessment are presented below. Only tests are cited in which propan-1-ol was the only carbon source.

Vaishnav et al. (1987) and Babeu and Vaishnav (1987) found a BOD_5/THOD ratio of 60% with an acclimated microbial culture from a domestic sewage (1 ml/bottle).

A BOD_5/COD ratio of 73% was determined with industrial activated sludge (BASF, 1978).

Pitter (1976) achieved an elimination of propan-1-ol of 98.8% measured as COD after 20 days with adapted activated sludge in a concentration of 100 mg/l.

Only one study is available that used unadapted and unacclimated inoculum (Price et al., 1974). In this study BOD/TOD ratios of 64, 76, 81 and 75% were found after 5, 10, 15 and 20 days. As the pass level of 60% as well as the 10-day window criterion was reached in this study, propan-1-ol can be regarded as readily biodegradable.

There are no results from simulation tests for biodegradation in waste water treatment plants, in the aquatic compartment and in soil. Consequently, taking account of the above-mentioned study, the following rate constants may be considered for biodegradation in accordance with the TGD.

Table 3.1 Degradation constants of propan-1-ol in different compartments

Compartment	Degradation constant
Waste water treatment plant	$kbio_{WWTP}$ = 1 h^{-1}
Aquatic environment	$kbio_{SW}$ = 0.047 d^{-1}
Soil	$kbio_{SOIL}$ = 0.023 d^{-1}
Sediment	$kbio_{SED}$ = 0.0023 d^{-1}

(see Appendix A1 for calculation)

Photodegradation

Direct photolysis of propan-1-ol in the atmosphere is not to be expected. However, in the atmosphere gaseous propan-1-ol reacts with hydroxyl radicals which are formed photochemically. Wallington and Kurylo (1987) determined a rate constant ($kdeg_{air}$) of 5.34 · 10^{-12} cm^3 $molecule^{-1}$ s^{-1} for this reaction at 296 K. Using an atmospheric concentration of the OH-radicals amounting to 5 · 10^5 OH/cm^3, a half-life of 3 days is calculated for the photochemical degradation in the atmosphere.

An estimation of the half-life for the atmospheric reaction of propan-1-ol with hydroxyl radicals with the program AOP 1.65 yields a value of 77.3 h (24-hour day, 5 · 10^5 OH/cm^3). This estimated half-life is used for the further calculations (see Appendix A1 for calculation).

Hydrolysis and Photolysis

A direct hydrolysis or photolysis in water is not expected due to the molecular structure of propan-1-ol, i.e. there is no relevant absorption above a wavelength of 290 nm.

Distribution

On account of the vapour pressure of 19.4 hPa, propan-1-ol is expected to evaporate quickly from surfaces.

A Henry's law constant of 0.117 Pa m^3/mol at 20°C is calculated from the data on the vapour pressure and water solubility of propan-1-ol given in Section 1. There are some experimentally determined Henry's law constants available in the literature. These vary from 0.377 Pa m^3/mol to 0.779 Pa m^3/mol (Altschuh et al., 1999; Betterton, 1992; Welke et al., 1998). Checks showed that these measured values although higher than the calculated one have no impact on the distribution in the WWTP. Since data are partly cited from secondary literature only and originating mainly from 1963 to 1985, the calculated Henry's law constant is used further in the RAR. Based on above mentioned data propan-1-ol can be considered as moderately volatile from an aqueous solution. (see Appendices for the calculation).

No bioaccumulation potential is to be expected due to the measured log P_{ow} value of 0.34. Based on this value the K_{oc} is calculated as 4.291 l/kg and the partition coefficients can be calculated according to the organic carbon content in the individual environmental compartments.

Table 3.2 Partition coefficients of propan-1-ol

Compartment	Partition coefficient
Soil-water	Kp_{soil} = 0.086 l/kg
Sediment-water	Kp_{sed} = 0.215 l/kg
Suspended matter-water	Kp_{susp} = 0.429 l/kg
Sewage sludge-water	Kp_{sludge} = 1.588 l/kg

(see Appendices for the calculation)

The following theoretical distribution is the environment results for propan-1-ol using the distribution model according to Mackay (Level 1) and the physico-chemical properties given in Section 1.

Table 3.3 Percentage distribution of propan-1-ol

Compartment	Percentage
Air	3.87
Water	96.13
Soil	0.0
Sediment	0.0

Consequently, the hydrosphere is the target compartment for propan-1-ol in the environment.

Elimination in waste water treatment plants

Based on the physico-chemical properties of propan-1-ol and in consideration of the rate constant for biodegradation of 0.1 h^{-1}, the elimination in waste water treatment plants can be determined using the SIMPLETREAT model in accordance with the TGD as follows (see Appendices):

Table 3.4 Elimination in WWTPs

Evaporation to air (%)	0.1
Release (dissolved) to water (%)	12.6
Adsorption to sewage sludge (%)	0
Degradation (%)	87.3
Total elimination from water (%)	87.4

Accumulation

No investigations on bioaccumulation are available. The measured log P_{ow} of 0.34 does not provide any indication of a relevant bioaccumulation potential.

The calculated K_{oc} value of 4.29 l/kg (see Appendix A1 for the calculation) also does not indicate that a significant geoaccumulation potential is to be expected for propan-1-ol. The substance may be washed out from soil to groundwater by rainwater depending on the elimination in soil by degradation and distribution.

3.1.2 Aquatic compartment

Releases into the waste water occur during production, use as an intermediate and use in products. The exposure data submitted by the company for production of propan-1-ol is used for the calculation of environmental concentration of propan-1-ol in surface water.

Since no exposure data have been submitted by the companies for further processing of propan-1-ol, in accordance with the ESD (Emission Scenario Documents, TGD Chapter 7), releases into the waste water amounting to 0.7% of the processing quantity are considered.

The exposure scenario for the formulation and use of propan-1-ol in products is based on the A and B Tables of the TGD.

3.1.2.1 Determination of the $Clocal_{water}$ / generic approach with regard to production and use as an intermediate

A generic exposure scenario for the entry of propan-1-ol into the waste water during production is not used because the producer has submitted the necessary exposure information.

Taking into consideration a maximum processing quantity at one site of 5,000 tonnes/annum (typical quantity for a company) a $Clocal_{water}$ of approximately 2.84 µg/l results for use of propan-1-ol as an intermediate at one processing site (see Appendices for the calculation, river flow rate = 60 $m^3 s^{-1}$).

3.1.2.2 Determination of the $Clocal_{water}$ / site-specific approach with regard to production

Using the currently available information on the individual manufacturer, site specific exposure calculation can be performed for this site (BASF AG, 1996).

Recently provided monitoring data (364 individual measurements in waste water effluent from 2000, 90 percentile) result in a $Clocal_{water}$ of 0.14 µg/l for the producer of propan-1-ol in the EU (see Appendices for the calculation).

3.1.2.3 Determination of the $Clocal_{water}$ / generic approach: use

Propan-1-ol is used as a solvent for disinfectants, pharmaceutical products, cleaning agents, paints, coating materials, enamel and lacquer paints, printing inks and cosmetics. The quantitative distribution of the application areas described in Section 2.2 is used for the exposure assessment.

A total amount of ca. 16,600 tonnes/annum is used as a solvent in the EU. Since it can be assumed that there are a large number of formulators and users involved, the 10% rule is applied to the calculation of $Clocal_{water}$ for these life cycles. The following quantities are considered for the calculation of the $Clocal_{water}$.

Table 3.5 Use quantities of propan-1-ol as solvent within EU

Application	Distribution	Quantity
cosmetics	20%	
pharmaceutics (disinfectants)	35%	
cleaning/washing agents	5%	
for IC 5 (personal/domestic)	60%	9,960 tonnes/annum
for IC 14 (lacquer, paints)	35%	5,810 tonnes/annum
for IC 0 (other)	5%	830 tonnes/annum

Use as a solvent in cosmetics, pharmaceutics (disinfectants) and cleaning/washing agents

If the substance is used as a solvent in cosmetics, pharmaceutics (disinfectants) and cleaning/washing agents, exposure is to be expected during the formulation of the products in the relevant companies. In addition, releases are expected during use of the products.

For the release estimations based on the use of propan-1-ol as a solvent in cosmetics, pharmaceutics (disinfectants) and cleaning/washing agents it is assumed that these 3 use categories are summarised for the formulation in companies and for the use as products in private households. This assumption is the conservative "worst case" and is used for the exposure calculation on the basis of default values.

The product quantity is estimated according to different fields of application (input B Tables).

Table 3.6 Estimation of product quantities containing propan-1-ol

application	quantity of used propan-1-ol	concentration in products	quantity of products
cosmetics	3,320 tonnes/annum	10%	33,200 tonnes/annum
pharmaceutics (disinfectants)	5,810 tonne/annum	25%	23,240 tonnes/annum
cleaning/washing agents	830 tonne/annum	15%	5,530 tonnes/annum
total quantity	9,960 tonne/annum	-	Approximately 61,970 tonnes/annum

In the case of use as a solvent in different products it is assumed that the total quantity used is released either to household waste water or to the atmosphere as a result of evaporation.

Use as a solvent in paints and lacquers

If the substance is used as a solvent in paints and lacquers exposure is to be expected during the formulation of the final products in the relevant companies. In addition, releases are expected during use of the paints and lacquers.

For the release estimations based on the use of propan-1-ol as a solvent in paints and lacquers a content of 50 % of the substance in the products is used for the derivation of the fraction of main source (input B Table) for the formulation of the products. The use of propan-1-ol in solvent based products specified as quick-drying paints and lacquers is assumed.

In the risk assessment the "worst case" exposure scenarios for both, paints for private and industrial use are calculated side by side (based on the used quantity of 5,810 tonnes/annum). With these two calculations one can identify the exposure scenario, leading to the maximum $Clocal_{water}$. For the continental and regional exposure calculation only the private use is taken

into consideration (based on the maximum release of propan-1-ol to the environment). There is no information to be able to assign the total volume to one of these two uses.

Use as a solvent in other non specified products

If the substance is used as a solvent for different non specified products exposure is to be expected during the formulation of the final products in the relevant companies. In addition, releases are expected during use of the solvent.

For the release estimations based on the use of propan-1-ol as a solvent a content of approximately 100% of the substance in the products are used for the derivation of the fraction of main source (input B Table) for the formulation (manufacture) of the products. The use of propan-1-ol as solvent is located in industrial area.

The results of the calculations of the $Clocal_{water}$ are summarised in the following table. Since it can be assumed that there are a large number of formulators and users involved, the 10% rule is applied to the calculation of $Clocal_{water}$ in these solvent scenarios.

Table 3.7 Results of calculations of $Clocal_{water}$ according to TGD for use of propan-1-ol as solvent

Types of use	**Solvent in household chemicals (pharmaceutics, disinfectants, cosmetics and cleaning / washing agents)**		**Solvent in paints and lacquers**			**Solvent not specified**	
Tonnage [a)] (t/a)	1,000	1,000	600	600	600	100	100
Main category	non-dispersive use (3)	wide dispersive use	non-dispersive use (3)	wide dispersive use	non-dispersive use	non-dispersive use (3)	non-dispersive use (3)
Industrial category Use category	5 (personal/domestic) 48 (solvents)	5 (personal/domestic) 48 (solvents)	14 (paints) 48 (solvents)	14 (paints) 48 (solvents)	14 (paints) 48 (solvents)	0 (other) 48 (solvents)	0 (other) 48 (solvents)
Life cycle step	formulation	private use	formulation	private use	processing	formulation	processing
Number of days	300 (B-table 2.1)	365 (B-table 4.1)	300 (B-table 2.10)	300 (B-table 4.5)	300 (B-table 3.13)	200 (B-table 2.8)	32 (B-table 3.14)
Release factor to water	0.003 (A-table 2.1)	0.6 (A-table 4.1)	0.02 (A-table 2.1)	0.04 (A-table 4.5)	0.02 (A-table 3.15)	0.02 (A-table 2.1)	0.01 (A-table 3.16)
Fraction of main source	0.4 (B-table 2.3)	0.002 (B-table 4.1)	1 (B-table 2.10)	0.0004 (B-table 4.5)	0.15 (B-table 3.13)	1 (B-table 2.8)	0.8 (B-table 3.14)
total emission to waste water (t/a)	3	600	12	24	12	2	1
Size of STP (m^3/d)	2,000	2,000	2,000	2,000	2,000	2,000	2,000
Dilution in receive. water	10	10	10	10	10	10	10
$Clocal_{effl.}$ (mg/l)	0.252	0.207	2.52	$2.02 \cdot 10^{-3}$	0.38	0.63	1.58
$Clocal_{water}$ (µg/l)	25.2	20.7	252	0.202	37.8	63	158

a) Tonnages result from application of 10% rule.
See Appendices for the calculation.

3.1.2.4 Data on occurrence in the hydrosphere

No measured values relating to the occurrence of propan-1-ol in the hydrosphere are available.

3.1.2.5 Sediment

Data on the occurrence in sediment do not exist for propan-1-ol. According to the known physico-chemical properties, there is no indication that propan-1-ol accumulates in sediment.

3.1.3 Atmosphere

In the case of the production of propan-1-ol in the EU, the release into the atmosphere is estimated as being 0.8 tonnes/annum (BASF AG, 1996). No further information is available with regard to the release into the atmosphere during the processing, formulation and use of the substance.

Since no exposure data have been submitted by the companies for further processing and formulation of propan-1-ol, the releases into the atmosphere are calculated in accordance with the TGD (A and B Tables in Chapter 3 Appendix I).

Using the SIMPLETREAT model, with regard to propan-1-ol, release from industrial waste water treatment plants as a result of evaporation into the air is estimated as approximately 0.1% of the quantity of the substance entering the waste water treatment plant. Consequently, an additional release into the atmosphere results for the individual production and processing and formulation sites. The same release route is also to be expected for use of the substance.

Based on the site specific exposure data of the one production site in the EU the resultant air concentration of 0.74 µg/m^3 and deposition quantity of 0.89 µg/m^2/day are calculated (see Appendices for the calculation).

For the further processing of approximately 5,000 tonnes/annum propan-1-ol at one site (typical quantity for a company) the resultant air concentration of 116 µg/m^3 and deposition quantity of 137 µg/m^2/day are calculated based on the A and B Tables of the TGD (see Appendices for the calculation).

By taking into consideration the current formulation quantities, the exposure tables in Chapter 3, Appendix I of the TGD and the SIMPLETREAT model, it is possible to calculate the releases into the atmosphere and the resultant deposition quantities according to the physico-chemical properties of the substance and the quantities of it which are used. The results of the calculations are summarised in the following table.

Please note, that releases resulting from use of propan-1-ol in household chemicals are expected mainly to waste water. Although environmental exposure is possible this route is neglected in the assessment.

Table 3.8 Results of calculations of $Clocal_{air}$ and $DEPtotal_{ann.}$ according to TGD for use of propan-1-ol as solvent

Types of use	**Solvent in household chemicals (pharmaceutics, disinfectants, cosmetics and cleaning / washing agents)**		**Solvent in paints and lacquers**			**Solvent not specified**	
Tonnage [a)] (t/a)	1,000	1,000	600	600	600	100	100
Main category	non-dispersive use (3)	wide dispersive use	non-dispersive use (3)	wide dispersive use	non-dispersive use	non-dispersive use (3)	non-dispersive use (3)
Industrial category Use category	5 (personal/domestic) 48 (solvents)	5 (personal/domestic) 48 (solvents)	14 (paints) 48 (solvents)	14 (paints) 48 (solvents)	14 (paints) 48 (solvents)	0 (other) 48 (solvents)	0 (other) 48 (solvents)
Life cycle step	formulation	private use	formulation	private use	processing	formulation	processing
Number of days	300 (B-table 2.1)		300 (B-table 2.10)	300 (B-table 4.5)	300 (B-table 3.13)	200 (B-table 2.8)	32 (B-table 3.14)
Release factor to air	0.025(A-table 2.1)		0.025 (A-table 2.1)	0.95 (A-table 4.5)	0.9 (A-table 3.15)	0.025 (A-table 2.1)	0.01 (A-table 3.16)
Fraction of main source	0.4 (B-table 2.1)		1 (B-table 2.10)	0.0004 (B-table 4.5)	0.15 (B-table 3.13)	1 (B-table 2.8)	0.8 (B-table 3.14)
Direct emission to air (t/a)	25		15	5,520	570	2.5	1
Annual deposition (µg/m²d)	11		16	0.25	89	2.7	0.88
Clocalair (µg/m³)	9.3		14	0.21	75	3.5	7.0

a) Tonnages result from application of 10% rule.
See Appendix A4 for the calculation

3.1.4 Terrestrial compartment

Propan-1-ol is expected to enter the soil as a result of deposition from the atmosphere. In this regard, the point sources of the production and the use of the substance as an intermediate as well as a solvent involving the highest amount of air pollution are considered (see Section 3.1.3).

The release of propan-1-ol to the soil according to these scenarios are summarised in the following table.

Table 3.9 Calculation of $Clocal_{soil}$

Type of use	Route of exposure	$PEClocal_{soil\text{-}porew}$ in µg/l	$Clocal_{soil}$ in µg/kg
Production	deposition	3.15	0.08
Processing of 5 000 t/a at one site (typical quantity for a company)	deposition	69.1	12.9
Formulation of household chemicals	deposition	8.04	1.03
Processing of paints in paint shops	deposition	45.8	8.35
Formulation of solvents (not specified)	deposition	4.04	0.26

See Appendices for the calculation.

Based on the SIMPLETREAT model (see Section 3.1.1) there is no adsorption of propan-1-ol at sewage sludge to be expected and the release to soil with sewage sludge application in agriculture is not considered in the risk assessment.

3.1.5 Secondary poisoning

Since there is no indication of propan-1-ol possessing a bioaccumulation potential, a risk characterisation for exposure via the food chain is not necessary.

3.1.6 Other non industrial emissions of propan-1-ol

Propan-1-ol is contained in landfill gas; it is formed from plants and animals through putrefaction and decomposition; alcohol-forming bacteria are involved here. The substance is contained in aromas of fruits and other foodstuffs. It is a natural component of alcoholic beverages that have been obtained through fermentation of plant raw materials (GDCh, 1997).

The quantity of formed propan-1-ol and the resultant environmental concentration in the different compartments cannot be quantified.

3.1.7 Regional exposure consideration

One production site and a processing site with a capacity of 5,000 tonnes/annum propan-1-ol (typical quantity for a company) are located in the considered region. The emission from the processing of the remaining approximately 8,500 tonnes/annum propan-1-ol is located in the continent.

In the determination of a regional background concentration all releases, from point and diffuse sources of the formulation and use of propan-1-ol, are considered. Two third (67%) of the total exposure quantity are taken into account for the continental model and one third (33%) of it for the defined regional EU standard model (densely populated area of 200 · 200 km with 20 million inhabitants). This conservative assumption is used for the first exposure assessment.

No direct release into the soil was identified. Diffuse release only occurs as a result of dispersal processes. Release is therefore to be expected as a result of deposition from the air (see Section 3.1.4).

Information is available according to which approximately 55% of the production volume is used as a solvent. Releases into the hydrosphere (see Section 3.1.2.3) and the atmosphere (see Section 3.1.3) are to be expected here.

Since not all of the previously mentioned releases arising from use of the substance enter the hydrosphere directly, but instead primarily via the waste water which is possibly purified in municipal waste water treatment plants, a 70% connection to waste water treatment plants, in which 67.4% of the substance is biodegraded and 0.1% volatilised, is assumed for this scenario. The remaining 30% of the water is discharged directly into the hydrosphere.

The individual environmental releases are summarised in the following table.

Table 3.10 Releases of propan-1-ol from different sources

field of application	Ratio reg./cont.	release to WWTPs (t/a)	direct release to the hydrosphere (t/a)	release into the atmosphere (t/a)
production	100/0	9.8	-	0.8
use as intermediate approx. 13,500 t/a	37/63	94.5	-	337.5
formulation of household chemicals	33/67	30	-	249
private use of household chemicals	33/67	4,186	1,794	-
formulation of paints	33/67	17.4	-	145.3
private use of paints	33/67	162.4	69.6	5,520
processing of paints*	(33/67)	(116)	(-)	(5,229)
formulation of solvents (not specified)	33/67	16.6	-	20.8
use of solvents (not specified)	33/67	8.3	-	8.3
total	-	4,525	1,864	6,282

* Not considered for the regional PEC, since the more release-relevant life-cycle stage “private use of paints” is already involved

In the calculation for the continental and regional model the individual releases are as follows.

Table 3.11 Input data for calculation of regional and continental PECs

	release (tonnes/annum) continental model	release (tonnes/annum) regional model
to air	4,174	2,107
to soil	-	-
to hydrosphere (direct)	1,249	615
to WWTPs	3,016	1,509

The figures given in the tables above were included exactly as they were estimated in the previous sections in order to ensure comprehensibility. The exactitude of the figures is not, however, intended as an indication of the absolute correctness.

The regional PECs resulting from the SimpleBox 2.0 calculations are (further details are presented in the Appendix A6):

$PECregional_{water}$ = 8.59 µg/l
$PECregional_{soil}$ = 0.525 µg/kg
$PECregional_{air}$ = 94.5 ng/m^3

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

3.2.1 Aquatic compartment

Available effect data

The propan-1-ol short-term toxicity studies to fish are summarised in the following table.

Table 3.12 Short-term toxicity to vertebrates

Test organism	Criterion	Duration [h]	Test system	Result [mg/l]	Reference
Oncorhynchus mykiss (Salmo gairdneri)	Mortality	48	static	LC_{50} = 3,200 (n)	Slooff et al. 1983
Pimephales promelas	Mortality	48	static	LC_{50} = 5,000 (n)	Slooff et al. 1983
Oryzias latipes	Mortality	48	static	LC_{50} = 5,900 (n)	Slooff et al. 1983
Leuciscus idus melanotus	Mortality	48	static	LC_{50} = 4,320 (n)	Juhnke/ Lüdemann 1978
Leuciscus idus melanotus	Mortality	48	static	LC_{50} = 4,560 (n)	Juhnke/ Lüdemann 1978
Pimephales promelas	Mortality	96	flow-through	LC_{50} = 4,480 (m)	Brooke et al. 1984
Pimephales promelas	Mortality	96	flow-through	LC_{50} = 4,630 (m)	Brooke et al. 1984
Alburnus alburnus	Mortality	96	flow-through	LC_{50} = 3,800 (n)	Bengtsson et al. 1984

Table 3.12 continued overleaf

Table 3.12 continued Short-term toxicity to vertebrates

Test organism	Criterion	Duration [h]	Test system	Result [mg/l]	Reference
Oryzias latipes	Mortality	48	static	LC_{50} > 1,000 (n) (T: 10°C, 20°C) LC_{50} = 640 (n) (T: 30°C)	Tsuji et al. 1986
Ambystoma mexicanum	Mortality	48	static	LC_{50} = 4,000 (n)	Slooff/ Baerselman 1980
Xenopus laevis	Mortality	48	static	LC_{50} = 4,000 (n)	Slooff/ Baerselman 1980

n nominal;
m measured

The reported effect values for fish show similar sensitivity of the tested species without considerable influence of the exposure duration (48 or 96 hours). Most tests were performed in open static systems without analytical monitoring of the test substance concentration. As propan-1-ol is moderately volatile (Henry's law constant 0.117 Pa m³/mol) it cannot be excluded that a decrease in test substance concentration may have occurred and the real effect values are therefore lower. However, from the two tests performed in flow-through systems with analytical monitoring, that found effect values in the same order of magnitude like the other available tests, it can be concluded that volatilisation will not have influenced the test results to a great extent.

Generally the observed effects reveal no serious toxicity of propan-1-ol to fish and amphibians. The lowest reported LC_{50}-value for fish was 640 mg/l. However, as this value was found at a temperature of 30°C it is not used for the further risk assessment. Instead, the value of 3,200 mg/l found for *Oncorhynchus mykiss* is interpreted as lowest available effect value for fish.

The following table shows the propan-1-ol short-term toxicity studies for invertebrates.

Table 3.13 Short-term toxicity to invertebrates

Test organism	Criterion	Duration [h]	Result [mg/l]	Reference
Daphnia magna	Immobilisation	24 48	EC_{50} = 4,262 EC_{50} = 3,644	Kühn et al.1989
Daphnia magna	Immobilisation	24	EC_{50} = 4,450	Bringmann/ Kühn 1977a
Artemia salina	Mortality	24	LC_{50} = 4,200	Price 1974
Daphnia magna IRCHA	Immobilisation	24	EC_{50} = 4,415	Bringmann/ Kühn 1982
Gammarus pulex	Mortality	48	EC_{50} = 1,000	Slooff 1983
Corixa punctata	Mortality	48	EC_{50} = 2,000	Slooff 1983
Ischnura elegans	Mortality	48	LC_{50} = 4,200	Slooff 1983
Nemoura cinerea	Mortality	48	LC_{50} = 1,520	Slooff 1983
Erpobdella octoculata	Mortality	48	LC_{50} = 1,400	Slooff 1983
Nitocra spinipes	Mortality	96	LC_{50} = 2,300	Bengtsson et al. 1984

All tests were performed without analytical monitoring of the test substance concentration. As discussed above, no significant decrease in test substance concentration due to volatilisation is expected. The lowest effect value of 1,000 mg/l was found for *Gammarus pulex*.

The following table shows the toxicity of propan-1-ol to different algae species. The results are given as nominal concentrations.

Table 3.14 Toxicity to algae

Test organism	Criterion	Duration [h]	Result [mg/l]	Reference
Selenastrum capricornutum	Growth	96	NOEC = 2,000	Slooff et al. 1983
Scenedesmus pannonicus	Growth	48	NOEC = 2,900	Slooff et al. 1983
Chlorella pyrenoidosa	Growth	48	NOEC = 1,150	Slooff et al. 1983
Microcystis aeruginosa	Cell multiplication inhibition	192	TTC = 255	Bringmann 1975
Scenedesmus quadricauda	Cell multiplication inhibition	192	TTC = 3,100	Bringmann/ Kühn 1977b

All tests were performed in open systems without analytical monitoring of the test substance concentration. Again, as discussed above it is not expected that the concentration of propan-1-ol significantly decreases due to volatilisation. Only NOEC-values or toxic threshold concentrations are available. The lowest TTC of 255 mg/l was found in a test with the blue-green algae *Microcystis aeruginosa*. However, due to the long exposure period of 8 days it can be assumed the growth of the algal culture is no longer in the exponential phase and therefore the test is regarded as not valid. The same is true for the 192-hour test with *Scenedesmus quadricauda*. Therefore, the lowest valid effect value for algae is the NOEC of 1,150 mg/l found for *Chlorella pyrenoidosa*.

The propan-1-ol microbial toxicity studies are shown in the following table.

Table 3.15 Toxicity to microorganisms

Test organism	Criterion	Duration [h]	Result [mg/l]	Reference
Activated sludge	Resp. inhibition	12	EC_{50} = 9,600 (closed system)	Blum/Speece 1991
Nitrosomonas sp.	Inhibition of ammonia consumption	12	EC_{50} = 980 (closed system)	Blum/Speece 1991
Methanogens	Inhibition of gas production	12	EC_{50} = 34,000 (closed system)	Blum/Speece 1991
Activated sludge (municipal)	Resp. inhibition	3	IC_{50} > 1,000	Klecka et al. 1985
Pseudomonas putida	Resp. inhibition	0.5	EC_{10} = 11,421	Robra 1979
Pseudomonas putida	Cell multiplication	16	TTC = 2,700	Bringmann/ Kühn 1977b
Entosiphon sulcatum	Cell multiplication	72	TTC = 38	Bringmann 1978
Uronema parduczi	Cell multiplication	20	TTC = 568	Bringmann/ Kühn 1980
Chilomonas paramaecium	Cell multiplication	48	TTC = 175	Bringmann et al. 1980

Determination of $PNEC_{aqua}$

The lowest available effect value of 1,000 mg/l was obtained with the invertebrate species *Gammarus pulex*. As no long-term tests with invertebrates and fish are available, an assessment factor of 1,000 has normally to be applied for the PNEC derivation. However, according to the TGD the usage of a lower assessment factor of 100 is justified because a) data from a wide selection of species covering additional taxonomic groups other than those represented by the base-set species (e.g. amphibians) are available (which additionally cross three trophic levels) and b) the mode of action law can be implemented (non-polar narcosis).

Therefore: $PNEC_{aqua}$ = 1,000 mg/l / 100 = 10 mg/l

Determination of $PNEC_{microorganisms}$

The most sensitive species to propan-1-ol was the protozoan *Entosiphon sulcatum*. However, the TTC of 38 mg/l found with this species is not used for the derivation of the $PNEC_{microorganisms}$ as *Entosiphon sulcatum* is not a typical representative of sewage treatment plants. The application of the following assessment factors according to the TGD to the other lowest available effect values gives the following $PNEC_{microorganisms}$:

Table 3.16 Derivation of $PNEC_{microorganisms}$

Chilomonas paramaecium	TTC = 175 mg/l	AF 1	PNEC = 175 mg/l
Activated sludge	IC50 = 9,600 mg/l	AF 100	PNEC = 96 mg/l
Nitrosomonas sp	IC50 = 980 mg/l	AF 10	PNEC = 98 mg/l
Pseudomonas putida	TTC = 2,700 mg/l	AF 1	PNEC = 2,700 mg/l

The PNEC derived from the effect values of activated sludge and *Nitrosomonas* are in the same order. The lowest derived value is 96 mg/l.

Therefore: $PNEC_{microorganisms}$ = 96 mg/l

Sediment

No risk assessment is required for this compartment since there are no indications of adsorption of the substance to sediments and neither measured concentrations of propan-1-ol for sediments nor experimental investigations with sediment organisms are available.

3.2.2 Atmosphere

No ecotoxicological data are available for this environmental compartment.

3.2.3 Terrestrial compartment

No ecotoxicological data are available for terrestrial organisms. In approximation, the aquatic PNEC can be used for the purpose of a risk assessment for the terrestrial compartment and compared with the concentration determined for the soil pore water:

$$PNEC_{soil} = 10 \text{ mg/l (soil pore water)}$$

3.2.4 Secondary Poisoning

Since propan-1-ol does not possess a bioaccumulation potential and is neither classified as "toxic" nor "harmful", it is not necessary for a PNEC to be derived.

3.3 RISK CHARACTERISATION

3.3.1 Aquatic compartment

Waste water treatment plants

The highest discharge concentration for waste water treatment plants is calculated as 2.52 mg/l for the formulation of solvents in paints and lacquers. A value of 1.58 mg/l was derived for the industrial processing of solvents (not specified); all other $Clocal_{eff}$ are in the µg/l range. Generic models are used for the calculation of the $Clocal_{effl.}$since no specific information is available.

Taking into consideration a $PNEC_{microorganisms}$ of 96 mg/l, maximum $Clocal_{effl.}$/PNEC ratios of 0.03 and ~0.02 result for these life cycle steps of propan-1-ol. Since the $Clocal_{effl.}$/PNEC ratios < 1, there is no risk to the microorganism population in the WWTP. **Conclusion (ii)**.

Aquatic environments

The PEC/PNEC ratios for all of the areas of production, processing and use are summarised in the following table (PECregional= 8.59 µg/l; $PNEC_{aqua}$= 10 mg/l).

Table 3.17 PEC/PNEC ratios for aquatic compartment

Company/area of use	PEC_{local} [µg/l]	$PEC/PNEC_{aqua}$
• Production at Site – A • Use as intermediate at Site - B (typical quantity for a company)	8.73 11.4	< 0.01 < 0.01
Formulation of household chemicals (used as a solvent in cosmetics, pharmaceutics, disinfectants and cleaning/washing agents)	33.8	< 0.01
Use of household chemicals (used as a solvent in cosmetics, pharmaceutics, disinfectants and cleaning/washing agents)	29.3	< 0.01
Formulation of paints	261	0.03
Use of paints in the private domain	8.79	< 0.01
Processing of paints in paint shops (e.g. car painting)	46.4	< 0.01
Formulation of solvents (for industrial use)	71.6	< 0.01
Processing of solvents (for industrial use)	166	0.02

Based on the conservative approaches for the exposure assessment all PEC/PNEC ratios < 1. On the currently available data there is no risk to aquatic organisms. **Conclusion (ii)**.

Sediment

No data on the occurrence in sediment or investigations of the effect on benthic organisms are available in connection with propan-1-ol. According to the available physico-chemical properties of the substance, there is no indication that propan-1-ol accumulates in sediment. Consequently, there is no need for a risk consideration for this compartment. **Conclusion (ii)**.

3.3.2 Atmosphere

Due to the atmospheric half-life ($t_{1/2}$ = approximately 77 hours), abiotic effects on the atmosphere, such as global warming and ozone depletion, are not to be expected in connection with propan-1-ol. The highest calculated air concentration is around 75 µg/m^3 for the processing of paints in paint shops (propan-1-ol is used as a solvent). Since no data are available on the ecotoxicological effect of the substance in connection with this environmental compartment, it is not possible to undertake a quantitative assessment of this environmental compartment. On the basis of the available information on the substance, the performance of tests is not considered necessary. **Conclusion (ii)**.

3.3.3 Terrestrial compartment

Releases into the terrestrial compartment as a result of deposition from the atmosphere are to be expected. The highest deposition rates result from the processing of propan-1-ol (generic approach) and the processing of paints in paint shops (propan-1-ol is used as a solvent). Soil concentrations for propan-1-ol amounting to

processing (generic)	13 µg/kg and 70 µg/l soil pore water
processing (paints)	8.4 µg/kg and 47 µg/l soil pore water.

Since no ecotoxicological data are available for terrestrial organisms, in approximation, the aquatic PNEC (10,000 µg/l) is considered for the purpose of the risk assessment of the terrestrial compartment and compared with the concentration determined for the soil pore water. With these data a maximum PEC/PNEC ratio of < 0.01 is calculated. Therefore, there is no indication of a risk to the terrestrial environmental compartment at present. **Conclusion (ii)**.

3.3.4 Secondary poisoning

Since there is no indication that propan-1-ol possesses a bioaccumulation potential, a risk characterisation for exposure via the food chain is not necessary.

4 HUMAN HEALTH

4.1 HUMAN HEALTH (TOXICITY)

4.1.1 Exposure assessment

4.1.1.1 General discussion

4.1.1.2 Occupational exposure

4.1.1.3 Consumer exposure

4.1.1.4 Indirect exposure via the environment

In accordance with the TGD, the indirect exposure of man to propan-1-ol via the environment, e.g. via food, drinking water and air, must be determined. In the form of a worst-case scenario, the most significant point source (in this case: propan-1-ol as solvent; processing of paints) is considered for calculation purposes. This result is then compared with a second calculation which is based on the regional background concentrations (see Section 3.1.7).

The results of these calculations with the corresponding input values are summarised in the Appendices. It is necessary to note, however, that the calculation model applied is as yet only provisional. It requires revision as soon as further information is available.

Table 4.1 Input parameter for calculation of indirect exposure

Parameters	Local scenario	Regional scenario
Annual average PEC in surface water[1] [mg/l]	0.040	$8.59 \cdot 10^{-3}$
Annual average PEC in air[1] [mg/m³]	0.062	$9.45 \cdot 10^{-5}$
PEC in grassland [mg/kg]	0.014	-
PEC in agricultural soil [mg/kg]	-	$2.91 \cdot 10^{-4}$
PEC in porewater of agricultural soil [mg/l]	0.046	$1.50 \cdot 10^{-3}$
PEC in porewater of grassland [mg/l]	0.071	-
PEC in groundwater under agricultural soil [mg/l]	0.046	-

1) For the estimation of indirect exposure via the environment, the local concentrations calculated for the emission period have to be averaged over the whole year.

The resultant daily doses for the substance are as follows:

- DOSEtot = 0.036 $mg/kg_{body\ weight}$ day (local scenario)
- DOSEtot = $3.119 \cdot 10^{-4}$ $mg/kg_{body\ weight}$ day (regional background concentrations)

The calculated uptake quantities result via the following routes.

Table 4.2 Route percentages of indirect exposure

Uptake route	% of total uptake	
	local	regional
drinking water	3.61	78.7
fish	0.07	1.76
plant shoot	58.8	10.5
root	0.67	2.53
meat	< 0.01	< 0.01
milk	0.02	0.01
air	36.8	6.49

Drinking water is the most significant route of uptake when using the regional approach. However, the local model indicates consumption of leaf crops and inhalation as main routes for indirect exposure.

5 RESULTS

5.1 ENVIRONMENT

From the intrinsic properties it is expected that propan-1-ol is of low concern for the environment. Therefore, a targeted environmental risk assessment was performed. Using conservative estimates based on worst-case assumptions at the exposure and effects side. The targeted risk assessment results in the following conclusion:

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.

Based on the currently available data, propan-1-ol represents no risk to the environment for the area of production, processing, formulation and use (see Section 3.3).

Although the exposure calculation is based on conservative “worst case” assumptions the calculated environmental concentrations remain clearly under the predicted no effect concentrations.

5.2 HUMAN HEALTH

5.2.1.1 Workers

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

For mutagenicity the base set data have to be completed, risk assessment concerning carcinogenicity will be delayed until the mutagenicity data are available.

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

There is a need for limiting the risks of propan-1-ol for several scenarios with short-term and repeated exposures. The most critical exposure route is inhalation, dermal contact being of minor importance. In detail concern is expressed for use of paints, use of cleaning formulations without LEV, short term exposures during use of printing inks.

The toxic effects leading to concern are respiratory depression according to stimulation of the trigeminus nerve, local effects in the airways after repeated exposure and reproductive toxicity concerning fertility as well as developmental toxicity. Risk reduction measures especially for the inhalative exposure situation have to be initiated.

5.2.1.2 Consumers

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

Mutagenicity

The minimum requirements in mutagenic testing are not met. An *in vitro* study on chromosome aberration in Chinese hamster cells is currently ongoing.

The producer has to be requested to make available existing studies.

Carcinogenicity

There is no valid carcinogenicity study available. The present data base gives no indication for carcinogenic effects. For performing the risk assessment on carcinogenicity, however, the completed data on mutagenicity have to be taken into account.

5.2.1.3 Humans exposed indirectly 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.

6 REFERENCES

Abraham MH, Chadha HS, Whiting GS and Mitchell RC (1994). J.Pharm.Sci. **83**, (8), 1085-1100.

Ahrens W and Jöckel K-H (1996). Stoffbelastung in der Papierindustrie. Schriftenreihe der Bundesanstalt für Arbeitsschutz – GA 48, S.68-71, Wirtschaftsverlag NW, Bremerhaven.

Alarie Y (1981). Dose response analysis in animal studies: Prediction of human responses. Env. Health Perspect **42**, 9-13.

Ariel (2000). Ariel Insight 2.0: For survival in a world of expanding chemical regulations, April 2000.

Altschuh J, Brüggemann R, Santl H, Eichinger G and Piringer OG (1999). Henry's law constants for a diverse set of organic chemicals: experimental dtermination and comparison of estimation methods. Chemosphere **39**, 1871-1887

Auty RM and Branch RA (1976). The elimination of ethyl, n-propyl, n-butyl and iso-amyl alcohols by the isolated perfused rat liver. J. Pharmacol. Exp. Ther. **197**, 669-674

Babeu L and Vaishnav DD (1987). Prediction of biodegradability for selected organic chemicals. J. Ind. Microbiol. **2**, 107-115.

Bariljak IP and Kozachuk CJ (1988). Untersuchung der cytogenetischen Wirkung einer Reihe einwertiger Alkohole auf Zellen des Knochenmarks von Ratten. Citologija i genetika **22**, 49-52.

BASF AG (1974). Unpublished report XXIV 522.

BASF AG (1978). Bestimmung der biologischen Abbaubarkeit von n-Propanol - Prüfergebnis, BASF AG DUU/OM - Z 570, 16.3.1978, unpublished.

BASF AG (1980). Unpublished results; 78/647.

BASF AG (1989). Labor fuer Umweltanalytik; unveroeffentlichte Untersuchung, 09.01.1989.

BASF AG (1994). Unpublished information from 19.01.1994.

BASF AG (1995). Safety data sheet "n-Propanol", 23.05.1995.

BASF AG (1996). Unpublished information from 19.04.1996.

BASF AG (1999). Unpublished information from 18.10.1999.

BAU (1994). Neue Stoffe am Arbeitsplatz: Ein Bewertungskonzept, Amtliche Mitteilungen der Bundesanstalt für Arbeitsschutz, Sonderdruck März 1994, Dortmund, Germany.

Baumann W and Muth A (1997). Farben und Lacke 1 - Daten und Fakten zum Umweltschutz, S. 143-146, Springer Verlag, Berlin.

Baumann W and Herberg-Liedtke B (1991). Druckereichemikalien - Daten und Fakten zum Umweltschutz, Springer Verlag, Berlin.

Beaugé F, Clément M, Nordmann J and Nordmann R (1979). Comparative effects of ethanol, n-propanol and isopropanol on lipid disposal by rat liver. Chem. Biol. Interact **26**, 155-166.

Bengtsson B-E, Renberg L and Tarkpea M (1984). Molecular structure and aquatic toxicity - an example with C1-C13 aliphatic alcohols. Chemosphere **13**,. 613-622.

Betterton EA (1992). Henry's law constants of soluble and moderately soluble organic gases: effects on aqueous phase chemistry. **In**: Gaseous pollutants, Wiley Series: Advances in Environmental Science and Technology, Vol. 24, Nriagu JO (ed.), John Wiley & Sons, New York.

BGAA (1997). Altstoffe – Exposition am Arbeitsplatz – 1-Propanol **In**: Berufsgenossenschaftlicher Arbeitskreis Altstoffe (BGAA) Report 1/99, Hauptverband der gewerblichen Berufsgenossenschaften, Sankt Augustin.

Bilzer N and Penners B-M (1985). Zur Frage der Abbau- und Ausscheidungsgeschwindigkeit der Begleitstoffe Propanol-1 und Isobutanol nach Genuß von Whisky der Marke "Chivas Regal". Blutalkohol **22**, 140-145.

Bilzer N, Schmutte P, Jehs M and Penners B-M (1990). Kinetik aliphatischer Alkohole (Methanol, Propanol-1 und Isobutanol) bei Anwesenheit von Äthanol im menschlichen Körper. Blutalkohol **27**, 385-409.

Blum DJW and Speece RE (1991). A database of chemical toxicity to environmental bacteria and its use in interspecies comparison and correlations. Res. J. Water Poll. Contr. Fed. **63** (3), 198-207.

Bonte W, Rüdell E, Sprung R, Frauenrath C, Blanke E, Kupilas G, Wochnik J and Zäh G (1881a). Experimentelle Untersuchungen zum Nachweis geringer Dosen höherer aliphatischer Alkohole im Urin von Versuchsteilnehmern. Blutalkohol **18**, 399-411.

Bonte W, Sprung R, Rüdell E, Frauenrath C, Blanke E, Kupilas G, Wochnik J and Zäh G (1981b). Experimentelle Untersuchungen zum Nachweis geringer Dosen höherer aliphatischer Alkohole im Urin von Versuchsteilnehmern. Blutalkohol **18**, 412-426.

Bos PMJ, Zwart A, Reuzel PGJ and Bragt PC (1992). Evaluation of the sensory irritation test for the assessment of occupational health risk. Crit. Rev. Toxicol. **21** (6), 423-450.

Boublik T, Fried V and Hala E (1984). The vapour pressure of pure substances. 2nd ed., Elsevier, Amsterdam.

Bremmer HJ and van Veen MP (2000). Factsheet algemeen. RIVM report 612810 009.

Bringmann G (1975). Bestimmung der biologischen Schadwirkung wassergefährdender Stoffe aus der Hemmung der Zellvermehrung der Blaualge Microcystis. Gesundheitsingenieur **96**, 238-241.

Bringmann G (1978). Bestimmung der biologischen Schadwirkung wassergefährdender Stoffe gegen Protozoen. Mitt. I. Bakterienfressende Flagellaten. Z. Wasser Abwasser Forsch. **11**, 210-215.

Bringmann G and Kühn R (1977a). Befunde der Schadwirkung wassergefährdender Stoffe gegen Daphnia magna. Z. Wasser Abwasser Forsch. **10**, 161-166.

Bringmann G and Kühn R (1977b). Grenzwerte der Schadwirkung wassergefährdender Stoffe gegen Bakterien (Pseudomonas putida) und Grünalgen (Scenedesmus quadricauda) im Zellvermehrungshemmtest. Z. Wasser Abwasser Forsch. **10**, 87-98.

Bringmann G and Kühn R (1980). Bestimmung der biologischen Schadwirkung wassergefährdender Stoffe gegen Protozoen. Mitt. II. Bakterienfressende Ciliaten. Z. Wasser Abwasser Forsch. **13**, 26-31.

Bringmann G, Kühn R and Winter A (1980). Bestimmung der biologischen Schadwirkung wassergefährdender Stoffe gegen Protozoen. Mitt. III. Saprozoische Flagellaten. Z. Wasser Abwasser Forsch. **13**, 170-173.

Bringmann G and Kühn R (1982). Ergebnisse der Schadwirkung wassergefährdender Stoffe gegen Daphnia magna in einem weiterentwickelten standardisierten Testverfahren. Z. Wasser Abwasser Forsch. **15**, 1-6.

Brooke LT, Call DJ, Geiger DL and Northcott CE (1984). Acute toxicities of organic chemicals to fathead minnows (Pimephales promelas). Center for Laker Superior Environmental Studies, University of Wisconsin-Superior. **1** (3), 5-16, 65-68.

Bushy Run Research Center (1979). Evaluation of the dermal carcinogenic potential of n-propanol.

Bushy Run Research Center (1980). Evaluation of the dermal cancerogenic potential of n-propanol. Project Report 42-96; March 14, 1980. **In**: EUCLID Data Sheet Union Carbide Benelux N V; 07.12.1995.

Bushy Run Research Center (1992). n-Propyl Alcohol (n-Propanol): Nine-Day-Vapor Inhalation in Rats. Project Report 54-87, March 5.

Carlson GP (1993). Formation of fatty acid propyl esters in liver, lung and pancreas of rats administered propan-1-ol. Res. Comm. Chem. Pathol. Pharmacol. **81** (1), 121-124.

CEH (1995). Oxochemicals. **In**: Chemical Economics Handbook, S. 682.7002S, 682.7002T, 684.7001C; SRI International.

CHEMSAFE: national database for safety data of the Physikalisch-technische Bundesanstalt Braunschweig, established by expert judgement

Cometto-Muniz JE and Cain WS (1990). Thresholds for odor and pungency. Physiol. and Behavior **48**, 719-725.

CRC handbook of chemistry and physics, 72nd ed., 1991-1992, CRC Press.

Crebelli R, Conti G and Carere A (1989). A comparative study on ethanol and acetaldehyde as inducers of chromosome malsegregation in aspergillus nidulans. Mutation Res. **215**, 187-195.

Delogu B (2000). Understanding the Precautionary Principle. Presentation at the International Conference on Chemical Control Regulations as representative of EU DG SANCO, 10/12 May 2000, Documentation ChemCon 2000, Austrian Federal Economic Chamber, Salzburg, Austria.

Dürwald W and Degen W (1956). Eine tödliche Vergiftung mit n-Propylakohol. Arch. Toxikol. **16**, 84-88.

ECB4/TR2/98: Technical Recommendation "The use of the 10% rule in emission estimations", ECB, Joint Research Centre, 1998.

EHC (1990). International Programme on Chemical Safety (IPCW), Environmental Health Criteria 102, propan-1-ol, World Health Organization, Geneva, 98 pages.

Ehrig T, Bohren KM, Wermuth B and von Wartburg JP (1988). Degradation of aliphatic alcohols by human liver alcohol dehydrogenase: Effect of ethanol and pharmacokinetic implications. Alcoholism: Clin. Exp. Res. **12**, 789-794.

Fahelbum IMS and James SP (1979). Absorption, distribution and metabolism of propyl anthranilate. Toxicol. **12**, 75-87.

Falbe J et al. (1980). Propanol. **In**: Ullmanns Enzyklopädie der technischen Chemie, 4. Auflage, Bd.19, 443-451, Verlag Chemie, Weinheim, Deerfield Beach, Basel.

Fiserova-Bergerova V (1985). Toxicokinetics of organic solvents. Scand. J. Work Environ. Health **1**, 7-21.

Fiserova-Bergerova V and Diaz ML (1986). Determination and prediction of tissue-gas partition coefficients. Int. Arch. Occup. Environ. Health **58**, 75-87.

Gad SC, Dunn BJ, Dobbs DW, Reilly C and Walsh RD (1986). Development and validation of an alternative dermal sensitization test: The mouse ear swelling test (MEST). Toxicol. Appl. Pharmacol. **84**, 93-114.

GDCh (1997). BUA report 190, 1-propanol, S. Hirzel Wissenschaftliche Verlagsgesellschaft

Gerner, Muhl, Rühl, Teich and Waßmann (1997). Beschichtungsarbeiten, BAU BG Hamburg.

Grant KA and Samson HH (1984). n-Propanol induced microcephaly in neonatal rat. Neurobehav. Toxicol. Teratol. **6**, 165-169.

Gulati A, Nath C, Shanker K, Srimal RC, Dhawan KN and Bhargava KP (1985). Effect of alcohols on the permeability of blood-brain barrier. Pharmacol. Res. Comm. **17**, 85-93.

Guo Z, Sparks LE and Bero MR (1995). Air exchange rate measurements in an IAQ test house. Engeneering Solutions to Indoor Air Quality Problems, Research Triangle Park, 498-510.

Haddock NF and Wilkin JK (1982). Cutaneous reactions to lower aliphatic alcohols before and during disulfiram therapy. Arch. Dermatol. **118**, 157-159.

Halarnkar PP and Blomquist GJ (1989). Comparative aspects of propionate metabolism. Comp. Biochem. Physiol. **92** (B), 227-231.

Hansch C and Anderson SM (1967). J. Org. Chem. **32**, 2583.

Harke P (1998). Disinfectants – Uses. **In**: Ullmann's Encylopedia of Industrial Chemistry, Sixth Ed., Edition Release, Wiley VCH, Weinheim.

Hayes S, Hayes C, Duncan D, Bennett V and Blushke J (1990). Stimulation of mutations suppressing the loss of replication control by small alcohols. Mutation. Res. **231**, 151-163.

Hein PM, Magerl H and Schulz E (1989). Detection of alcohols in saliva. J. Clin. Chem. Clin. Biochem. **27**, 231.

Heydenreich A (1966). Chemisch-toxische Schäden der Augen (Vergiftungen, Berufskrankheiten) Monatsblätter für Augenheilkunde **149**, 145-165.

Hiaki T, Takahashi K, Tsuji T, Hongo M and Kojima K (1994). J. Chem. Eng. Data **39**, 602-604.

Hillbom ME, Franssila K and Forsander OA (1974). Effects of chronic ingestion of some lower aliphatic alcohols in rats. Res. Comm. Pathol. Pharmacol. **9**, 177-180.

Hilscher H, Geissler E, Lohs K and Gibel W (1969). Untersuchungen zur Toxizität und Mutagenität einzelner Fuselöl-Komponenten an E.coli. Acta. Biol. Med. Germ. **23**, 843-852.

Hoechst AG (1994). Unpublished information from 14.06.1994.

Hoechst AG (1995a). Safety data sheet "n-Propanol", 20.07.1995.

Hoechst AG (1995b). Unpublished information from 15.12.1995.

Hoechst AG (1997). Unpublished information from 20.03.1997.

Hude von der et al. (1987). Genotoxicity of three-carbon compounds evaluated in the SCE test in vitro. Environ. Mutag. 1987 **9**, 401-410.

Hude von der et al. (1988). Evaluation of the SOS chromotest. Mutation Res. 1988 **203**, 81-94.

Iffland R, Balling P, Oehmichen M, Lieder F and Norpoth Th (1989). Methanol, Isopropanol, n-propanol - endogene Bildung unter Äthanoleinfluß? Blutalkohol. **26**, 87-97.

INRS (2000). Results of occupational exposure measurements to 1-propanol, COLCHIC database, No. 38/2000.

Juhnke I and Lüdemann D (1978). Ergebnisse der Untersuchung von 200 chemischen Verbindungen auf akute Fischtoxizität mit dem Goldorfentest. Z. Wasser Abwasser Forsch. **5**, 161-164.

Kalberlah F et al. (1999). Zeitextrapolation und Interspeziesextrapolation bei lokal wirksamen Stoffen mit begrenzter Datenlage, Endbericht des Forschungsvorhabens F1719 der BauA, Bundesanstalt für Arbeitsschutz und Arbeitsmedizin, Schriftenreihe -Forschung- Fb 862.

Kalberlah F and Schneider K (1998). Quantifizierung von Extrapolationsfaktoren, Endbericht des Forschungsvorhabens Nr. 116 06 113 des Umweltbundesamtes, Bundesanstalt für Arbeitsschutz und Arbeitsmedizin, Schriftenreihe -Forschung- Fb 796.

Kamil IA, Smith JN and Wiliams RT (1953). Studies in detoxication. 46. The metabolism of aliphatic alcohols. The glucuronic acid conjugation of acyclic aliphatic alcohols. Biochem J. **53**, 129-137.

Kane LE, Dombroke R and Alarie Y (1980). Evaluation of sensory irritation from some common industrial solvents. Am. Ind. Hyg. Ass. **41**, 451-455.

Kaneko T, Wang P-Y and Sato A (1994). Partition coefficients of some acetate esters and alcohols in water, blood, olive oil, and rat tissues. Occupat. Environ. Med. **51**, 68-72.

Kennedy GL and Graepel GJ (1991). Acute toxicity in the rat following either oral or inhalation exposure. Toxicol. Letters **56**, 317-326.

Khudoley et al. (1987). The study of mutagenic activity of carcinogens and other chemical agents with Salmonella typhimurium assays: Testing of 126 compounds. Arch. F. Geschwulstforschung **57**, 453-462.

Klecka GM, Land LP and Bodner KM (1985). Evaluation of the OECD activated sludge respiration inhibition test. Chemosphere **14**, 1239-1251.

Kristiansen U, Hansen L, Nielsen GD and Holst E (1986). Sensory irritation and pulmonary irritation of cumene and n-propanol: Mechanisms of receptor activation and desentitization. Acta. Pharmacol. et Toxicol. **59**, 60-72.

Kühn R, Pattard M, Pernak K-D and Winter A (1989). Results of the harmful effects of selected water pollutants (anilines, phenols, aliphatic compounds) to Daphnia magna. Water Res. **23**, 495-499.

Kühnholz B, Wehner HD and Bonte W (1984). In-vitro-Untersuchungen zur Löslichkeit aliphatischer Alkohole in Körpergeweben. Blutalkohol. **21**, 308-318.

Lasne et al. (1984). The in vitro micronucleus for detection of cytogenetic effects induced by mutagen-arcinogens:comperison with in vitro sister-chromatid exchange assay. Mutation Res. **130**, 273-282.

Ludwig E and Hausen BM (1977). Sensitivity to isopropyl alcohol. Contact Dermatitis; **3**, 240-244.

Maickel RP and Nash JF Jr (1985). Differing effects of short-chain alcohols on body temperature and coordinated muscular activity in mice. Neuropharmacology; **24**, 83-89.

Morgan ET, Koop DR and Coon MJ (1982). Catalytic activity of cytochrome P-450 isozyme 3a isolated from liver microsomes of ethanol-treated rabbits. J. Biol. Chem. **257**, 13951-13957.

Morris JB and Cavanagh DG (1987). Metabolism and deposition of propanol and acetone vapors in the upper respiratory tract of the hamster. Fund. Appl. Toxicol. **9**, 34-40.

Munch JC (1972). Aliphatic alcohols and alkyl esters: narcotic and lethal potencies to tadpoles and rabbits. Ind. Med. **41**, 31-33.

Nelson BK, Brightwell WS, Taylor and Burg JR (1985). Comparison of behavioral teratogenic effects of ethanol and N-propanol administered by inhalation to rats. Neurobehavioral Toxicol. and Teratol. **7**, 779-783.

Nelson BK, Brightwell WS, MacKenzie-Taylor DR, Khan A, Burg JR and Weigel WW (1988). Teratogenicity of n-propanol and isopropanol administered at high inhalation concentration to rats. Fd. Chem. Toxicol. **26**, 247-254.

Nelson BK, Brightwell WS, Taylor BJ, Khan A, Burg JR, Krieg EF, JR and Massari VJ (1989). Behavioral teratology investigation of propan-1-ol administered by inhalation to rats. Neurotoxicol. and Teratol. **11**, 153-159.

Nelson BK, Brightwell WS and Krieg EF (1989). Developmental toxicology of industrial alcohols: a summary of 13 alcohols administered by inhalation to rats. Teratol. **39**, 471.

Nelson BK, Brightwell WS and Krieg EF (1990). Developmental toxicology of industrial alcohols: a summary of 13 alcohols administered by inhalation to rats. Toxicol. Ind.l Health **6**, 373-387.

Nelson BK, Brightwell WS and Krieg EF (1996). Developmental toxicology of industrial alcohols: a summary of 13 alcohols administered by inhalation to rats. Int. J. Occup. Med. Immunol. Toxicol. **5**, 29-42.

Nielsen GD and Bakbo JC (1985). Exposure limits for irritants. Ann. Am. Conf. Ind. Hyg. **12**, 119-133.

NO_NL (1999). Guidelines for quantitative risk characterisation of non-threshold carcinogens in the framework of existing chemicals following Council Regulation (EEC) 793/93, Draft 09.08.99, Commission Working Group on the Technical Meetings for Risk Assessment for Existing Substances, NO_NL/01/99.

Nordman R (1980). Metabolism of some higher alcohols. INSERM (Les Colloques de l`INSERM: Alcohol and the gastrointestinal tract) **95**, 187-205.

Obe G and Ristow H (1977). Acetaldehyde, but not ethanol, induces sister chromatid exchanges in Chinese hamster cells in vitro. Mutation Res. **56**, 211-213.

Pedersen LM (1987). Biological Studies in human exposure to and poisoning with organic solvents: Kinetics, haemotology, and serum chemistry. Phamacol. Toxicol. **61** (III), 1-38.

Peschel O, Bauer MF, Gilg T, v Meyer L (1992). Veränderung von Begleitstoffanalysen durch percutane Resorption propanolhaltiger Antiseptika. Blutalkohol. **29**, 172-184.

Petrasol B.V. Gorinchem, HSDB (Hazardous Substances Data Bank), on-line via STN.

Pitter P (1976). Determination of biological degradability of organic substances. Water Res. **10**, 231-235.

Price KS, Waggy GT and Conway RA (1974). Brine shrimp bioassay and seawater BOD of petrochemicals. J. Water Pollut. Contr. Fed. **46**, 63-77.

Rietbrock N and Abshagen U (1971). Pharmakokinetik und Stoffwechsel aliphatischer Alkohole. Arzneimittelforsch **21**, 1309-1319.

Robra KH (1979). Akute Bakterientoxizität: Auswertung von Ringversuchen mit einer Reinkultur im Vergleich zu Untersuchungen an Mischpopulationen. Vom Wasser **53**, 267-282.

Rotter ML, Koller W and Neumann R (1991). The influence of cosmetic additives on the acceptability of alcohol-based hand disinfectants. J. Hospital Inf. **18**, 57-63.

Savini EC (1968). Estimation of the LD50 in Mol/kg. Proceedings of the European Society for the Study of Drug. Toxicity **9**, 276-278.

Schaper M (1993). Development of a database for sensory irritants and its use in establishing occupational exposure limits. Am. Ind. Hyg. Assoc. **54**, 488-495.

Scheuplein RJ and Blank IH (1973). Mechanism of percutaneous absorption. IV. Penetration of nonelectrolytes (alcohols) from aqueous solutions and from pure liquids. J. Invest. Dermatol. **60**, 286-296.

Schmutte P, Bilzer N and Penners BM (1988). Zur Nüchternkinetik der Begleitalkohole Methanol und Propanol-1. Blutalkohol. **25**, 137-142.

Shehata M and Saad S (1978). The effect of aliphatic alcohols on certein vitamins of the B-complex group in the liver of the rat. Po. J. Pharmacol. Pharm. **30**, 35-39.

Siegel IA, Izutsu KT and Watson E (1981). Mechanisms of non-electrolyte penetration across dog and rabbit oral mucosa in vitro. Arch. Oral Biol. **26**, 357-361.

Sinclair J, Lambrecht L and Smith EL (1990). Hepatic alcohol dehydrogenase activity in chick hepatocytes towards the major alcohols present in commercial alcoholic beverages: Comparison with activities in rat and human liver. Comp. Biochem. Physiol. **96** (B), 677-682.

Slooff W (1983). Benthic macroinvertebrates and water quality assessment: some toxical considerations. Aquatic Toxicol. **4**, 73-82.

Slooff W and Baerselman R (1980). Comparison of the usefulness of the mexican Axolotl (Ambystoma mexicanum) and the Clawed Toad (Xenopus laevis) in toxicological bioassays. Bull. Environ. Contam. Toxicol. **24**, 439-443.

Slooff W, Canton JH and Hermens JLM (1983). Comparison of the susceptibility of 22 freshwater species to 15 chemical compounds. I. (Sub)acute toxicity tests. Aquatic Toxicol. **4**, 113-128.

Smyth HF, Carpenter CP, Weil CS and Pozzani UC (1954). Range-finding toxicity data, List V. Arch. Ind. Hyg. Occup. Med. 1954 **10**, 61-68.

Stolzenberg SJ and Hine CH (1979). Mutagenicity of halogenated and oxygenated three-carbon compounds. J. Toxicol. Environ. Health **5**, 1149-1158.

Stoye D, Funke W, Hoppe L, Hasselkus J, Curtis L, Hoehne K, Zech H-J, Heiling P and Yamabe M (1998). Paints and Coatings – Production Technology. **In**: Ullmann's Encylopedia of Industrial Chemistry, Sixth Ed., Edition Release, Wiley VCH, Weinheim.

Taylor JM, Jenner PN and Jones WI (1964). A comparison of the toxicity of some allyl, propenyl, and propyl compounds in the rat. Toxicol. Appl. Pharmacol. **6**, 378-387.

Union Carbide Corporation (1991). Safety data sheet "n-Propanol".

Union Carbide Corporation (1992). N-propyl alcohol (n-propanol): acute inhalation toxicity in rats. Bushy Run Research Center; Project Report 54-48 (BRRC No. 90-13-40281).

Vaishnav DD, Boethling RS and Babeu L (1987). Quantitative structure-biodegradability relationships for alcohols, ketones and alicyclic compounds. Chemosphere 16/4, 695-703.

van Veen MP, Fortezza F, Bloemens HJTh and Kliest JJ (1999). Indoor air exposure to volatile compounds emitted by paints: Model and experiment. J. Expo. Anal. Epidem. **9**, 569-574.

Wakabayashi KA, Kayo A and Popinigis J (1991). Effects of alkyl alcohols and related chemicals in rat liver structure and function. Acta. Pathol. Jap. **41**, 405-413.

Wallington TJ and Kurylo MJ (1987). The gas phase reaction of hydroxyl radicals with a series of aliphatic alcohols over the temperature range 240 - 440 K. Intern. J. Chem. Kinet. **19**, 1015-1023.

Wehner HD and Schieffer MC (1989). Eliminationseigenschaften des Begleitstoffes n-Propanol. Blutalkohol. **26**, 28-41.

Weissermel K and Arpe H-J (1988). Synthesen mit Kohlenmonoxid. In: Industrielle organische Chemie, bedeutende Vor- und Zwischenprodukte. 3. Auflage, 133-145, VCH Verlagsgesellschaft mbH, Weinheim.

Welke B, Ettlinger K and Riederer M (1998). Sorption of Volatile Organic Chemicals in Plant Surfaces. Environ. Sci. Technol. **32**, 1099-1104.

Wilhoit RC and Zwolinski BJ (1973). J. Phys. Chem. Reference Data 2 (Suppl.1) 1-1, 1-5, 1-66, 1-68 bis 1-72, 1-76, 1-389 bis 1-408.

Wilkin JK and Fortner G (1985a). Cutaneous vascular sensitivity to lower aliphatic alcohols and aldehydes in Orientals. Clin. Exp. Res. **9**, 522-525.

Wilkin JK and Fortner G (1985b). Ethnic contact urticaria to alcohol. Contact Dermatitis **12**, 118-120.

Yaws CL, Yang H-C, Hopper JR and Hansen KC (1990). Chemical Enginieering **7**, 116.

ABBREVIATIONS

ADI	Acceptable Daily Intake
AF	Assessment Factor
Ann	Annex
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
BOD	Biochemical Oxygen Demand
bw	body weight / *Bw, bw*
C	Corrosive (Symbols and indications of danger for dangerous substances and preparations according to Annex II of Directive 67/548/EEC)
°C	degrees Celsius (centigrade)
C_{50}	median immobilisation concentration or median inhibitory concentration 1 / *explained by a footnote if necessary*
CA	Chromosome Aberration
CA	Competent Authority
CAS	Chemical Abstract Services
CEC	Commission of the European Communities
CEN	European Standards Organisation / European Committee for Normalisation
CEPE	European Committee for Paints and Inks
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	Day(s)
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
DT_{50}	Degradation half-life or period required for 50 percent dissipation / degradation
DT_{50lab}	Period required for 50 percent dissipation under laboratory conditions (define method of estimation)
DT_{90}	Period required for 90 percent dissipation / degradation
$DT_{90field}$	Period required for 90 percent dissipation under field conditions (define method of estimation)
E	Explosive (Symbols and indications of danger for dangerous substances and preparations according to Annex II of Directive 67/548/EEC)
EASE	Estimation and Assessment of Substance Exposure Physico-chemical properties [Model]
EbC_{50}	Effect Concentration measured as 50% reduction in biomass growth in algae tests
EC	European Communities
EC_{10}	Effect Concentration measured as 10% effect
EC_{50}	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 II of Directive 67/548/EEC)
FAO	Food and Agriculture Organisation of the United Nations
FELS	Fish Early Life Stage
foc	Organic carbon factor (compartment depending)
G	Gram(s)
GLP	Good Laboratory Practice
h	hour(s)
ha	Hectares/*h*
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 tonnes/annum)
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
kg	kilogram(s)
Koc	organic carbon normalised distribution coefficient
Kow	octanol/water partition coefficient
Kp	solids-water partition coefficient
kPa	kilo Pascals
l	litre(s)
$L(E)C_{50}$	median Lethal (Effect) Concentration
LAEL	Lowest Adverse Effect Level
LC_{50}	median Lethal Concentration
LD_{50}	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
log	logarithm to the basis 10
m	Meter
MAC	Maximum Allowable Concentration
MATC	Maximum Acceptable Toxic Concentration
MC	Main Category
mg	Milligram(s)
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 II 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	Oxidising (Symbols and indications of danger for dangerous substances and preparations according to Annex II of Directive 67/548/EEC)
OC	Organic Carbon content
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
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
$PNEC_{water}$	Predicted No Effect Concentration in Water
POP	Persistent Organic Pollutant
PPE	Personal Protective Equipment
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 IV 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 II of Directive 67/548/EEC)
TDI	Tolerable Daily Intake
TG	Test Guideline
TGD	Technical Guidance Document
TNsG	Technical Notes for Guidance (for Biocides)
TNO	The Netherlands Organisation for Applied Scientific Research
ThOD	Theoritical Oxygen Demand
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
µg	microgram(s)
vB	very Bioaccumulative
VOC	Volatile Organic Compound
vP	very Persistent
vPvB	very Persistent and very Bioaccumulative
v/v	volume per volume ratio
w	gram weight
w/w	weight per weight ratio
WHO	World Health Organisation
WWTP	Waste Water Treatment Plant
Xn	Harmful (Symbols and indications of danger for dangerous substances and preparations according to Annex II of Directive 67/548/EEC)
Xi	Irritant (Symbols and indications of danger for dangerous substances and preparations according to Annex II of Directive 67/548/EEC)

Appendix A Distribution and fate

Distribution and Fate

$d := Tag$

Substance: **Propanol CAS Nr. 71-23-8**

melting point:	$MP := 146.65 \cdot K$
vapour pressure:	$VP := 1940 \cdot Pa$
water solubility:	$SOL := 1000000 \cdot mg \cdot l^{-1}$
part. coefficient octanol/water:	$LOGP_{OW} := 0.34$
molecular weight:	$MOLW := 0.06009 \cdot kg \cdot mol^{-1}$
gas constant:	$R := 8.3143 \cdot Pa \cdot m^3 \cdot mol^{-1} \cdot K^{-1}$
temperature:	$T := 293 \cdot K$
conc. of suspended matter in the river:	$SUSP_{water} := 15 \cdot mg \cdot l^{-1}$
density of the solid phase:	$RHO_{solid} := 2500 \cdot kg \cdot m^{-3}$
volume fraction water in susp. matter:	$Fwater_{susp} := 0.9$
volume fraction solids in susp.matter:	$Fsolid_{susp} := 0.1$
volume fraction of water in sediment:	$Fwater_{sed} := 0.8$
volume fraction of solids in sediment:	$Fsolid_{sed} := 0.2$
volume fraction of air in soil:	$Fair_{soil} := 0.2$
volume fraction of water in soil:	$Fwater_{soil} := 0.2$
volume fraction of solids in soil:	$Fsolid_{soil} := 0.6$
aerobic fraction of the sediment comp.:	$Faer_{sed} := 0.1$
product of CONjunge and $SURF_{air}$:	$product := 10^{-4} \cdot Pa$

distribution air/water: Henry-constant

$$HENRY := \frac{VP \cdot MOLW}{SOL} \qquad HENRY = 0.117 \circ Pa \cdot m^3 \cdot mol^{-1}$$

$$\log\left(\frac{HENRY}{Pa \cdot m^3 \cdot mol^{-1}}\right) = -0.933$$

$$K_{air\_water} := \frac{HENRY}{R \cdot T} \qquad K_{air\_water} = 4.785 \bullet 10^{-5}$$

solid/water-partition ceefficient Kp_{comp} and total compartment/water-partition coefficient K_{comp_water}

$a := 0.39$ (a,b from TGD, p. 539) alcohols

$b := 0.5$

$K_{OC} := 10^{a \cdot LOGP_{OW} + b} \cdot l \cdot kg^{-1}$ $K_{OC} = 4.291 \cdot l \cdot kg^{-1}$

Suspended matter

$Kp_{susp} := 0.1 \cdot K_{OC}$ $Kp_{susp} = 0.429 \cdot l \cdot kg^{-1}$

$K_{susp\_water} := Fwater_{susp} + Fsolid_{susp} \cdot Kp_{susp} \cdot RHO_{solid}$ $K_{susp\_water} = 1.007$

factor for the calculation of $Clocal_{water}$:

$faktor := 1 + Kp_{susp} \cdot SUSP_{water}$ $faktor = 1$

Sediment

$Kp_{sed} := 0.05 \cdot K_{OC}$ $Kp_{sed} = 0.215 \cdot l \cdot kg^{-1}$

$K_{sed\_water} := Fwater_{sed} + Fsolid_{sed} \cdot Kp_{sed} \cdot RHO_{solid}$ $K_{sed\_water} = 0.907$

Soil

$Kp_{soil} := 0.02 \cdot K_{OC}$ $Kp_{soil} = 0.086 \cdot l \cdot kg^{-1}$

$K_{soil\_water} := Fair_{soil} \cdot K_{air\_water} + Fwater_{soil} + Fsolid_{soil} \cdot Kp_{soil} \cdot RHO_{solid}$

$K_{soil\_water} = 0.329$

Sludge (activated sludge)

$K_{p\_sludge} := 0.37 \cdot K_{OC}$ $K_{p\_sludge} = 1.588 \cdot l \cdot kg^{-1}$

Raw sewage

$K_{p\_sewage} := 0.30 \cdot K_{OC}$ $K_{p\_sewage} = 1.287 \cdot l \cdot kg^{-1}$

Elimination in STPs

rate constant in STP: k = 1 h^{-1} elimination P = f (k, logpow, logH) =0.678

fraction directed to surface water $Fstp_{water}$ =0.322

biodegradation in different compartments

surface water

$kbio_{water} := 0.047 \cdot d^{-1}$ (cTGD, table 5)

soil

$DT50bio_{soil} := 30 \cdot d$ (cTGD, table 6)

$$kbio_{soil} := \frac{\ln(2)}{DT50bio_{soil}} \qquad kbio_{soil} = 0.023 \cdot d^{-1}$$

sediment

$$kbio_{sed} := \frac{\ln(2)}{DT50bio_{soil}} \cdot Faer_{sed} \qquad kbio_{sed} = 2.31 \cdot 10^{-3} \cdot d^{-1}$$

degradation in surface waters

$khydr_{water} := 0 \cdot h^{-1}$

$kphoto_{water} := 0 \cdot h^{-1}$

$kdeg_{water} := khydr_{water} + kphoto_{water} + kbio_{water}$

$kdeg_{water} = 0.047 \cdot d^{-1}$

Atmosphere

calculation of CONjunge * SURFaer for the OPS-model

$$VPL := \frac{VP}{\exp\left[6.79 \cdot \left(1 - \frac{MP}{285 \cdot K}\right)\right]} \qquad VP := wenn(MP > 285 \cdot K, VPL, VP) \qquad VP = 1.94 \cdot 10^{3} \cdot Pa$$

$$Fass_{aer} := \frac{product}{VP + product} \qquad Fass_{aer} = 5.155 \cdot 10^{-8}$$

degradation in the atmosphere

$kdeg_{air}$ = 0.00896 h^{-1} (see AOP-calculation)

SimpleTreat 3.0 (debugged version, 7 Feb 97)

today is 20. Jun 07

input

Characterization of the chemical

Name compound = Propanol

Physico-chemical properties

Molecular weight =	60	[	1E+02]	g mol^{-1}	0,06	kg mol^{-1}
K_{ow} =	2,19E+00	[	1E+03]	(-)	2,188	(-)
Vapour pressure =	1,94E+03	[	1E+00]	Pa	1940	Pa
Solubility =	1,00E+06	[	1E+02]	mg L^{-1}	16639	mol m^{-3}
K_a =		[	1E-20]	(-)	1E-20	(-)
K_b =		[	1E-20]	(-)	1E-20	(-)
Henry constant (H) =	1,17E-01	[	**1E-01**]	Pa m^3 mol^{-1}	0,117	Pa m^3 mol^{-1}
K_p (raw sewage) =		[	**7E-01**]	L kg_{dwt}^{-1}	0,656	L kg_{dwt}^{-1}
K_p (activated sludge) =		[	**8E-01**]	L kg_{dwt}^{-1}	0,81	L kg_{dwt}^{-1}

Emission scenario

T air =		[	15]	centigrade	288	Kelvin
T water =		[	15]	centigrade	288	Kelvin
Windspeed =		[	3]	m s^{-1}	3	m s^{-1}
Sewage flow =		[	200]	L $person^{-1} d^{-1}$	0,2	m^3 $person^{-1} d^{-1}$
Number inhabitants =		[	1E+04]	person	10000	person
Sludge loading rate (table 1) =		[	0,15]	kg_{BOD} kg_{dwt}^{-1} d^{-1}	0,15	kg_{BOD} kg_{dwt}^{-1} d^{-1}
Bubble or surface aeration: b/s		[	s]	(-)	s	(-)
Emission rate chemical =		[	1]	kg d^{-1}	1	kg d^{-1}

table 2: chosen operation parameters

Sludge loading rate =	0,15	kg_{BOD} kg_{dwt}^{-1} d^{-1}
with primary sedimentation		
HRT =	6,9	hr
SRT =	9,2	d
without primary sedimentation		
HRT =	10,8	hr
SRT =	9,2	d

table 1:

Sludge loading rate (SLR) related to hydraulic retentiontime (HRT) and sludge retention time (SRT). HRT with primary sedimentation (PS) and without; PS has no influence on SR

SLR (kg_{BOD} kg_{dwt}^{-1} d^{-1})	HRT (PS) (hr)	HRT (hr)	SRT (d)	nitrification (-)
0.04 (low)	25,9	40,5	37,0	yes
0.06 (low)	17,3	27	24,1	yes
0.1 (low)	10,4	16,2	14,1	yes
0.15 (medium)	6,9	10,8	9,2	yes
0.2 (medium)	5,2	8,1	6,8	no
0.3 (high)	3,5	5,4	4,5	no
0.6 high)	1,7	2,7	2,2	no

table 3:

Concentration in raw sewage calculated from the default emission rate of the chemical

C total raw sewage=	5,0E-01	mg l^{-1}
Dissolved =	100	%
C dissolved =	5,0E-01	mg l^{-1}
C in solids =	3,3E-01	mg kg^{-1}

Biodegradation in activated sludge

Temperature dependence (y/n) [n] (-) n

Method 1: estimated from OECD/EU standardized biodegradability tests (USES 2.0)

Assumption: degradation according to first order kinetics with respect to the concentration in the aqueous phase of activated sludge, implying that the chemical adsorbed to solids is not available for biodegradation.

The following values are recommended:

Readily biodegradable, fulfilling 10 d window criterion: range is 1 to 3 hr^{-1} (TGD-EU: 1 hr^{-1})

Readily biodegradable, not fulfilling 10 d window criterion: range is 0.3 to 1 hr^{-1} (TGD-EU: 0.3 hr^{-1})

Inherently biodegradable in MITI II and within 10 d in the Zahn-Wellens (window = 3 d): range is 0.1 to 0.3 hr^{-1}

Inherently biodegradable: range is 0 .01 to 0.1 hr^{-1} (TGD-EU: 0.1 hr^{-1})

k biodeg1 = 1 [0] hr^{-1} 3E-04 s^{-1} ,T water = 15 C

Method 2: chemical is biodegradable in activated sludge batch test (draft ISO test)

Assumption: degradation according to first order kinetics with respect to the concentration in the slurry phase, implying that biodegradation occurs both in the aqueous and the solids phase of activated sludge.

k biodeg2 = [0] hr^{-1} 0 s^{-1} ,T water = 15 C

Method 3: chemical is biodegradable in activated sludge, Monod Kinetics

Assumption: biodegradation in the aqueous phase of activated sludge, however, the rate constant is a function of the influent concentration, δ_{max} and Ks and the sludge retention time. Default values for δ_{max} and Ks pertain to readily biodegradable chemicals.

δ_{max} =		[	2]	d^{-1}	2	d^{-1}
K_s =		[	0,5]	mg L^{-1}	0,5	mg L^{-1}

output of SimpleTreat 3.0 (debugged version, 7 Feb 97)								today is #########			
	report of	**Propanol**									
including primary sedimentation											
Elimination in the primary settler											
	volatilization	0,0									
	via primary sludge	0,0									
	total	0,0	%								
						without primary sedimentation					
Elimination in the aerator						Elimination in the aerator					
	stripping	0,0					stripping	0,0			
	biodegradation	87,3					biodegradation	91,5			
	total	87,3	%				total	91,5	%		
Elimination in the solids liquid separator						Elimination in the solids liquid separator					
	volatilization	0,0					volatilization	0,0			
	via surplus sludge	0,0					via surplus sludge	0,0			
	total	0,0	%				total	0,0	%		
Total elimination from waste water		87,4	%			Total elimination from waste water		91,5	%		
Total emission via effluent		12,6	%------V			Total emission via effluent		8,5	%------V		
			12,64	% dissolved					8,46	% dissolved	
			0,00	% associated					0,00	% associated	
	balance	100,0	%				balance	100,0	%		
Summary of distribution						Summary of distribution					
	to air	0,1					to air	0,0			
	to water	12,6					to water	8,5			
	via primary sludge	0,0					via surplus sludge	0,0			
	via surplus sludge	0,0					degraded	91,5			
	degraded	87,3					total	100,0	%		
	total	100,0	%								
Concentrations						Concentrations					
	in air	9,79E-09	g m^{-3}				in air	4,13E-09	g m^{-3}		
	in combined sludge	2,62E-01	mg kg^{-1}				in surplus sludge	3,48E-02	mg kg^{-1}		
	|	in primary sludge:	3,28E-01	mg kg^{-1}			in effluent (total)	4,23E-02	mg l^{-1}		
	|	in surplus sludge:	5,14E-02	mg kg^{-1}			|	dissolved	4,23E-02	mg l^{-1}	
	in effluent (total)	0,06319	mg l^{-1}				|	associated	1,04E-06	mg l^{-1}	
	|	dissolved	6,32E-02	mg l^{-1}			in solids effluent	3,48E-02	mg kg^{-1}		
	|	associated	1,54E-06	mg l^{-1}							
	in solids effluent	5,14E-02	mg kg^{-1}								
Operation of the plant equipped with a primary settler						Operation of the plant without primary settler					
Sludge loading rate =	0,15	kg BOD (kg dw)$^{-1}$ d^{-1}				Sludge loading rate =	0,15	kg BOD (kg dw)$^{-1}$ d^{-1}			
HRT =	6,9	h				HRT =	10,8	h			
SRT =	9,20	d				SRT =	9,20	d			
Aeration mode =	surface aeration					Aeration mode =	surface aeration				
Primary sludge =	6,00E+02	kg dry weight d^{-1}				Surplus sludge =	3,31E+02	kg dry weight d^{-1}			
Surplus sludge =	1,90E+02	kg dry weight d^{-1}				Total wastewater =	2,00E+03	m^3 d^{-1}			
Total sludge =	7,90E+02	kg dry weight d^{-1}									
Total wastewater =	2,00E+03	m^3 d^{-1}									

Appendix B $Clocal_{water}$ Calculation for Processing, Formulation and Use

Estimation of $Clocal_{water}$ at formulation of household chemicals

(pharmaceutics, disinfectants, cosmetics and cleaning/washing agents)
status: formulation, IC:5 UC:48

$\mu g := 10^{-9} \cdot kg$

$d := 86400 \cdot s$

$a := 365 \cdot d$

chemical : Propanol CAS-Nr.: 71-23-8

Total annual tonnage of chemical: $TONNAGE := 1000 \cdot tonne \cdot a^{-1}$

Release factor (TGD,table A2.1): $f_{emission} := 0.003$

Fraction of main source (B-table:2.1, 10% rule): $Fmainsource := 0.4$

Waste water flow of wwtp: $EFFLUENT_{stp} := 2000 \cdot m^3 \cdot d^{-1}$

Duration of emission (B-table:2.1): $Temission := 300 \cdot d \cdot a^{-1}$

Fraction of emission directed to water:
(SimpleTreat; k:1 h^{-1}; logPow:0.34 ; logH:-0.93) $Fstp_{water} := 12.6 \cdot \%$

Dilution factor (TGD): $DILUTION := 10$

Factor (1+Kp * SUSPwater): $FACTOR := 1$

Emission per day:

$$Elocal_{water} := \frac{TONNAGE \cdot Fmainsource \cdot f_{emission}}{Temission} \qquad Elocal_{water} = 4 \circ kg \cdot d^{-1}$$

Influent concentration:

$$Clocal_{inf} := \frac{Elocal_{water}}{EFFLUENT_{stp}} \qquad Clocal_{inf} = 2 \circ mg \cdot l^{-1}$$

Effluent concentration:

$$Clocal_{eff} := Clocal_{inf} \cdot Fstp_{water} \qquad Clocal_{eff} = 0.252 \circ mg \cdot l^{-1}$$

Concentration in surface water:

$$Clocal_{water} := \frac{Clocal_{eff}}{FACTOR \cdot DILUTION} \qquad Clocal_{water} = 25.2 \circ \mu g \cdot l^{-1}$$

Total release for the regional model (without elimination in STPs):

$$RELEASE := TONNAGE \cdot f_{emission} \qquad RELEASE = 3 \circ tonne \cdot a^{-1}$$

Annual average local concentration in water:

$$Clocal_{water\_ann} := Clocal_{water} \cdot \frac{Temission}{365 \cdot d \cdot a^{-1}} \qquad Clocal_{water\_ann} = 0.021 \circ mg \cdot l^{-1}$$

Estimation of $Clocal_{water}$ at formulation of solvent (IC = other)

status: TGD,A+B table, IC - 0

$\mu g := 10^{-9} \cdot kg$

$d := 86400 \cdot s$

$a := 365 \cdot d$

chemical : Propanol CAS-Nr.: 71-23-8

Total annual tonnage of chemical: $TONNAGE := 100 \cdot tonne \cdot a^{-1}$

Release factor (A 2.1): $f_{emission} := 0.02$

Fraction of main source (B 2.8): $Fmainsource := 1$

Waste water flow of wwtp: $EFFLUENT_{stp} := 2000 \cdot m^3 \cdot d^{-1}$

Duration of emission (B 2.8): $Temission := 200 \cdot d \cdot a^{-1}$

Fraction of emission directed to water:
(SimpleTreat; k:1h^{-1}; logPow:0.34 ; logH:-0.93) $Fstp_{water} := 12.6 \cdot \%$

Dilution factor (TGD): $DILUTION := 10$

Factor (1+Kp * SUSPwater): $FACTOR := 1$

Emission per day:

$$Elocal_{water} := \frac{TONNAGE \cdot Fmainsource \cdot f_{emission}}{Temission} \qquad Elocal_{water} = 10 \cdot kg \cdot d^{-1}$$

Influent concentration:

$$Clocal_{inf} := \frac{Elocal_{water}}{EFFLUENT_{stp}} \qquad Clocal_{inf} = 5 \cdot mg \cdot l^{-1}$$

Effluent concentration:

$$Clocal_{eff} := Clocal_{inf} \cdot Fstp_{water} \qquad Clocal_{eff} = 0.63 \cdot mg \cdot l^{-1}$$

Concentration in surface water:

$$Clocal_{water} := \frac{Clocal_{eff}}{FACTOR \cdot DILUTION} \qquad Clocal_{water} = 63 \cdot \mu g \cdot l^{-1}$$

Total release for the regional model (without elimination in STPs):

$$RELEASE := TONNAGE \cdot f_{emission} \qquad RELEASE = 2 \cdot tonne \cdot a^{-1}$$

Annual average local concentration in water:

$$Clocal_{water\_ann} := Clocal_{water} \cdot \frac{Temission}{365 \cdot d \cdot a^{-1}} \qquad Clocal_{water\_ann} = 34.5 \cdot \mu g \cdot l^{-1}$$

Estimation of Clocal$_{water}$ at formulation of chemicals for paints, laques and varnishes (used as a solvent) status: TGD,A+B table, IC:14,UC:48

$\mu g := 10^{-9} \cdot kg$

$d := 86400 \cdot s$

$a := 365 \cdot d$

chemical : Propanol CAS-Nr.: 71-23-8

Total annual tonnage of chemical: $TONNAGE := 600 \cdot tonne \cdot a^{-1}$

Release factor (A 2.1): $f_{emission} := 0.02$

Fraction of main source (B 2.10): $Fmainsource := 1$

Waste water flow of wwtp: $EFFLUENT_{stp} := 2000 \cdot m^3 \cdot d^{-1}$

Duration of emission (B 2.10): $Temission := 300 \cdot d \cdot a^{-1}$

Fraction of emission directed to water: (SimpleTreat; k:1h^{-1}; logPow:0.34 ; logH:-0.93) $Fstp_{water} := 12.6 \cdot \%$

Dilution factor (TGD): $DILUTION := 10$

Factor (1+Kp * SUSPwater): $FACTOR := 1$

Emission per day:

$$Elocal_{water} := \frac{TONNAGE \cdot Fmainsource \cdot f_{emission}}{Temission} \qquad Elocal_{water} = 40 \cdot kg \cdot d^{-1}$$

Influent concentration:

$$Clocal_{inf} := \frac{Elocal_{water}}{EFFLUENT_{stp}} \qquad Clocal_{inf} = 20 \cdot mg \cdot l^{-1}$$

Effluent concentration:

$$Clocal_{eff} := Clocal_{inf} \cdot Fstp_{water} \qquad Clocal_{eff} = 2.52 \cdot mg \cdot l^{-1}$$

Concentration in surface water:

$$Clocal_{water} := \frac{Clocal_{eff}}{FACTOR \cdot DILUTION} \qquad Clocal_{water} = 252 \cdot \mu g \cdot l^{-1}$$

Total release for the regional model (without elimination in STPs):

$$RELEASE := TONNAGE \cdot f_{emission} \qquad RELEASE = 12 \cdot tonne \cdot a^{-1}$$

Annual average local concentration in water:

$$Clocal_{water\_ann} := Clocal_{water} \cdot \frac{Temission}{365 \cdot d \cdot a^{-1}} \qquad Clocal_{water\_ann} = 207.1 \cdot \mu g \cdot l^{-1}$$

Calculation of PEC_{local} for aquatic compartment during processing of Propanol generic scenario (without specific wwtp)

status: TGD,ESD, IC-3

$d := 86400 \cdot s$

$a := 365 \cdot d$

$\mu g := 10^{-9} \cdot kg$

chemical: **Propanol**

Processing volume:	$T := 5000 \cdot tonne \cdot a^{-1}$
Emission factor for processing; (TGD, tab. A3.3):	$f := 0.7 \cdot \%$
Fraction of the main source - processing (on-site)	$fmainsource := 1$
Duration of emission for processing; (on-site):	$Temission := 300 \cdot d \cdot a^{-1}$
Fraction of emission directed to water: (Simple-Treat, k:1h-1; logH:-0,93 ; $logK_{ow}$:0,34)	$Fstp_{water} := 12.6 \cdot \%$
River flow rate for processing (TGD):	$FLOW := 60 \cdot m^3 \cdot s^{-1}$
Factor (1 + K_p * SUSPwater):	$FACTOR := 1$

Release $RELEASE := T \cdot f$

$RELEASE = 95.89 \cdot kg \cdot d^{-1}$

processing

Emission per day: $Elocal_{water} := \frac{T \cdot f}{Temission}$

$Elocal_{water} = 116.67 \cdot kg \cdot d^{-1}$

Concentration in surface water:

$Clocal_{water} := \frac{Elocal_{water} \cdot Fstp_{water}}{FLOW \cdot FACTOR}$

$Clocal_{water} = 2.84 \cdot \mu g \cdot l^{-1}$

Estimation of Clocal$_{water}$ of chemicals for processing of solvents (IC = 0)
status: TGD,A+B table, IC - 0, MC:3

$\mu g := 10^{-9} \cdot kg$

$d := 86400 \cdot s$

$a := 365 \cdot d$

chemical : Propanol CAS-Nr.: 71-23-8

Total annual tonnage of chemical: $TONNAGE := 100 \cdot tonne \cdot a^{-1}$

Release factor (A 3.16): $f_{emission} := 0.01$

Fraction of main source (B 3.14): $Fmainsource := 0.8$

Waste water flow of wwtp: $EFFLUENT_{stp} := 2000 \cdot m^3 \cdot d^{-1}$

Duration of emission (B 3.14): $Temission := 32 \cdot d \cdot a^{-1}$

Fraction of emission directed to water:
(SimpleTreat; k:1h^{-1}; logPow:0.34 ; logH:-0.93) $Fstp_{water} := 12.6 \cdot \%$

Dilution factor (TGD): $DILUTION := 10$

Factor (1+Kp * SUSPwater): $FACTOR := 1$

Emission per day:

$$Elocal_{water} := \frac{TONNAGE \cdot Fmainsource \cdot f_{emission}}{Temission} \qquad Elocal_{water} = 25 \cdot kg \cdot d^{-1}$$

Influent concentration:

$$Clocal_{inf} := \frac{Elocal_{water}}{EFFLUENT_{stp}} \qquad Clocal_{inf} = 12.5 \cdot mg \cdot l^{-1}$$

Effluent concentration:

$$Clocal_{eff} := Clocal_{inf} \cdot Fstp_{water} \qquad Clocal_{eff} = 1.58 \cdot mg \cdot l^{-1}$$

Concentration in surface water:

$$Clocal_{water} := \frac{Clocal_{eff}}{FACTOR \cdot DILUTION} \qquad Clocal_{water} = 157.5 \cdot \mu g \cdot l^{-1}$$

Total release for the regional model (without elimination in STPs):

$$RELEASE := TONNAGE \cdot f_{emission} \qquad RELEASE = 1 \cdot tonne \cdot a^{-1}$$

Annual average local concentration in water:

$$Clocal_{water\_ann} := Clocal_{water} \cdot \frac{Temission}{365 \cdot d \cdot a^{-1}} \qquad Clocal_{water\_ann} = 13.8 \cdot \mu g \cdot l^{-1}$$

Estimation of $Clocal_{water}$ of chemicals for paints, laques and varnishes at processing in paint shops **status: TGD,A+B table, IC-14 UC:48**

$\mu g := 10^{-9} \cdot kg$

$d := 86400 \cdot s$

$a := 365 \cdot d$

chemical : Propanol CAS-Nr.: 71-23-8

Total annual tonnage of chemical: $TONNAGE := 600 \cdot tonne \cdot a^{-1}$

Release factor (A 3.15): $f_{emission} := 0.02$

Fraction of main source (B 3.13): $Fmainsource := 0.15$

Waste water flow of wwtp: $EFFLUENT_{stp} := 2000 \cdot m^3 \cdot d^{-1}$

Duration of emission (B 3.13): $Temission := 300 \cdot d \cdot a^{-1}$

Fraction of emission directed to water: (SimpleTreat; k:1h^{-1}; logPow:0.34 ; logH:-0.93) $Fstp_{water} := 12.6 \cdot \%$

Dilution factor (TGD): $DILUTION := 10$

Factor (1+Kp * SUSPwater): $FACTOR := 1$

Emission per day:

$$Elocal_{water} := \frac{TONNAGE \cdot Fmainsource \cdot f_{emission}}{Temission} \qquad Elocal_{water} = 6 \circ kg \cdot d^{-1}$$

Influent concentration:

$$Clocal_{inf} := \frac{Elocal_{water}}{EFFLUENT_{stp}} \qquad Clocal_{inf} = 3 \circ mg \cdot l^{-1}$$

Effluent concentration:

$$Clocal_{eff} := Clocal_{inf} \cdot Fstp_{water} \qquad Clocal_{eff} = 0.38 \circ mg \cdot l^{-1}$$

Concentration in surface water:

$$Clocal_{water} := \frac{Clocal_{eff}}{FACTOR \cdot DILUTION} \qquad Clocal_{water} = 37.8 \circ \mu g \cdot l^{-1}$$

Total release for the regional model (without elimination in STPs):

$RELEASE := TONNAGE \cdot f_{emission}$ $\quad RELEASE = 12 \circ tonne \cdot a^{-1}$

Annual average local concentration in water:

$$Clocal_{water\_ann} := Clocal_{water} \cdot \frac{Temission}{365 \cdot d \cdot a^{-1}} \qquad Clocal_{water\_ann} = 31.1 \circ \mu g \cdot l^{-1}$$

Estimation of $Clocal_{water}$ at use of household chemicals

(pharmaceutics, disinfectants, cosmetics and cleaning/washing agents)
status: private use, IC:5 UC:48

chemical : Propanol CAS-Nr.: 71-23-8

$d := 24 \cdot h$

$a := 365 \cdot d$

$\mu g := 10^{-9} \cdot kg$

Tonnage of chemical:	$TONNAGE := 1000000 kg \cdot a^{-1}$
Fraction of main source (B 4.1):	$F_{mainsource} := 0.002$
Release factor (A 4.1):	$f_{emission} := 0.6$
Duration of emission (B 4.1):	$T_{emission} := 365 \cdot d \cdot a^{-1}$
Fraction of emission directed to water: (SimpleTreat; k:1h^{-1}; logPow:0.34 ; logH:-0.93)	$Fstp_{water} := 12.6 \cdot \%$
Waste water flow of wwtp:	$EFFLUENT_{stp} := 2000 \cdot m^3 \cdot d^{-1}$
Dilution factor (TGD):	$DILUTION := 10$
Factor (1+K_p * SUSPwater):	$FACTOR := 1$

Emission per day:

$$Elocal_{water} := \frac{TONNAGE \cdot F_{mainsource} \cdot f_{emission}}{T_{emission}}$$

$Elocal_{water} = 3.288 \cdot kg \cdot d^{-1}$

Influent concentration:

$$Clocal_{inf} := \frac{Elocal_{water}}{EFFLUENT_{stp}}$$

$Clocal_{inf} = 1.644 \cdot mg \cdot l^{-1}$

Effluent concentration:

$Clocal_{eff} := Clocal_{inf} \cdot Fstp_{water}$

$Clocal_{eff} = 207.123 \cdot \mu g \cdot l^{-1}$

Concentration in receiving water:

$$Clocal_{water} := \frac{Clocal_{eff}}{DILUTION \cdot FACTOR}$$

$Clocal_{water} = 20.7 \cdot \mu g \cdot l^{-1}$

Emission for $PEC_{regional}$ (without wwtp):

$RELEASE := TONNAGE \cdot f_{emission}$

$RELEASE = 600 \cdot tonne \cdot a^{-1}$

annual average local concentration in surface water:

$Clocal_{water\_ann} := Clocal_{water}$

$Clocal_{water\_ann} = 20.7 \cdot \mu g \cdot l^{-1}$

Estimation of Clocal$_{water}$ of chemicals for paints, laques and varnishes at privat use **status: TGD,A+B table, IC-14, UC:48**

$\mu g := 10^{-9} \cdot kg$

$d := 86400 \cdot s$

$a := 365 \cdot d$

chemical : Propanol CAS-Nr.: 71-23-8

Total annual tonnage of chemical: $TONNAGE := 600 \cdot tonne \cdot a^{-1}$

Release factor (A 4.5): $f_{emission} := 0.04$

Fraction of main source (B 4.5): $Fmainsource := 0.0004$

Waste water flow of wwtp: $EFFLUENT_{stp} := 2000 \cdot m^3 \cdot d^{-1}$

Duration of emission (B 4.5): $Temission := 300 \cdot d \cdot a^{-1}$

Fraction of emission directed to water: (SimpleTreat; k:1h^{-1}; logPow:0.34 ; logH:-0.93) $Fstp_{water} := 12.6 \cdot \%$

Dilution factor (TGD): $DILUTION := 10$

Factor (1+Kp * SUSPwater): $FACTOR := 1$

Emission per day:

$$Elocal_{water} := \frac{TONNAGE \cdot Fmainsource \cdot f_{emission}}{Temission} \qquad Elocal_{water} = 0.03 \cdot kg \cdot d^{-1}$$

Influent concentration:

$$Clocal_{inf} := \frac{Elocal_{water}}{EFFLUENT_{stp}} \qquad Clocal_{inf} = 0.016 \cdot mg \cdot l^{-1}$$

Effluent concentration:

$$Clocal_{eff} := Clocal_{inf} \cdot Fstp_{water} \qquad Clocal_{eff} = 2.016 \cdot 10^{-3} \cdot mg \cdot l^{-1}$$

Concentration in surface water:

$$Clocal_{water} := \frac{Clocal_{eff}}{FACTOR \cdot DILUTION} \qquad Clocal_{water} = 0.2 \cdot \mu g \cdot l^{-1}$$

Total release for the regional model (without elimination in STPs):

$RELEASE := TONNAGE \cdot f_{emission}$ $RELEASE = 24 \cdot tonne \cdot a^{-1}$

Annual average local concentration in water:

$$Clocal_{water\_ann} := Clocal_{water} \cdot \frac{Temission}{365 \cdot d \cdot a^{-1}} \qquad Clocal_{water\_ann} = 0.2 \cdot \mu g \cdot l^{-1}$$

Appendix C $Clocal_{air}$ calculation for processing, formulation and use

Atmosphere (OPS-model)

Calculation of Clocal $_{air}$ and PEC local $_{air}$

chemical : Propanol CAS-Nr.: 71-23-8

stage of life cycle:formulation household chemical (pharmaceutics, disinfectants, cosmetics and cleaning/washing agents) IC:5 UC:48 MC:3

$d := 86400 \cdot s$

$a := 365 \cdot d$

$mg := 1 \cdot 10^{-6} \cdot kg$

tonnage for specific scenario:

$TONNAGE := 1000 \cdot tonne \cdot a^{-1}$

release factor (table A-2.1):

$f_{emission} := 0.025$

fraction of main source (table B-2.1):

$Fmainsource := 0.4$

days of use per year (table B-2.1):

$Temission := 300 \cdot d \cdot a^{-1}$

release during life cycle to air:

$RELEASE := TONNAGE \cdot f_{emission}$

$RELEASE = 25 \circ tonne \cdot a^{-1}$

local emission during episode to air:

$$Elocal_{air} := \frac{Fmainsource \cdot RELEASE}{Temission}$$

$Elocal_{air} = 33.333 \circ kg \cdot d^{-1}$

concentration in air at source strength of 1kg/d

$Cstd_{air} := 2.78 \cdot 10^{-4} \cdot mg \cdot m^{-3} \cdot kg^{-1} \cdot d$

fraction of the emission to air from STP

$Fstp_{air} := 0.1 \cdot \%$

local emission rate to water during emission episode

$Elocal_{water} := 4 \cdot kg \cdot d^{-1}$

local emission to air from STP during emission episode

$Estp_{air} := Fstp_{air} \cdot Elocal_{water}$

$Estp_{air} = 4 \bullet 10^{-3} \circ kg \cdot d^{-1}$

local concentation in air during emission episode:

$Clocal_{air} := wenn\left(Elocal_{air} > Estp_{air}, Elocal_{air} \cdot Cstd_{air}, Estp_{air} \cdot Cstd_{air}\right)$

$Clocal_{air} = 9.267 \bullet 10^{-3} \circ mg \cdot m^{-3}$

annual average concentration in air, 100m from point source

$$Clocal_{air\_ann} := Clocal_{air} \cdot \frac{Temission}{365 \cdot d \cdot a^{-1}}$$

$Clocal_{air\_ann} = 7.616 \bullet 10^{-3} \circ mg \cdot m^{-3}$

regional concentration in air

$PECregional_{air} := 9.45 \cdot 10^{-5} \cdot mg \cdot m^{-3}$

annual average predicted environmental concentration in air

$PEClocal_{air\_ann} := Clocal_{air\_ann} + PECregional_{air}$

$PEClocal_{air\_ann} = 7.711 \bullet 10^{-3} \circ mg \cdot m^{-3}$

Calculation of the deposition rate

standard deposition flux of aerosol-bound compounds at a source strength of 1kg/d

$$DEPstd_{aer} := 1 \cdot 10^{-2} \cdot mg \cdot m^{-2} \cdot d^{-1} \cdot kg^{-1} \cdot d$$

fraction of the chemical bound to aerosol (see: Distribution and Fate)

$$Fass_{aer} := 5.155 \cdot 10^{-8}$$

deposition flux of gaseous compounds as a function of Henry`s Law coefficient,at a source strength of 1kg/d

logH<-2	5*10 $^{-4}$ mg*m^{-2}*d^{-1}
-2<logH<2	4*10^{-4} mg*m^{-2}*d^{-1}
logH>2	3*10^{-4} mg*m^{-2}*d^{-1}

$$DEPstd_{gas} := 4 \cdot 10^{-4} \cdot mg \cdot m^{-2} \cdot d^{-1} \cdot kg^{-1} \cdot d$$

total deposition flux during emission episode

$$DEPtotal := \left(Elocal_{air} + Estp_{air}\right) \cdot \left[Fass_{aer} \cdot DEPstd_{aer} + \left(1 - Fass_{aer}\right) \cdot DEPstd_{gas}\right]$$

$$DEPtotal = 0.013 \, mg \cdot m^{-2} \cdot d^{-1}$$

annual average total depostion flux

$$DEPtotal_{ann} := DEPtotal \cdot \frac{Temission}{365 \cdot d \cdot a^{-1}}$$

$$DEPtotal_{ann} = 0.011 \, mg \cdot m^{-2} \cdot d^{-1}$$

Atmosphere (OPS-model)

Calculation of Clocal $_{air}$ and PEC local $_{air}$

chemical : Propanol CAS-Nr.: 71-23-8

stage of life cycle: formulation of solvent not specified
IC:0 UC:48 MC:3

$d := 86400 \cdot s$

$a := 365 \cdot d$

$mg := 1 \cdot 10^{-6} \cdot kg$

tonnage for specific scenario:

$TONNAGE := 100 \cdot tonne \cdot a^{-1}$

release factor (A 2.1):

$f_{emission} := 0.025$

fraction of main source (table B 2.8):

$Fmainsource := 1$

days of use per year (table B 2.8):

$Temission := 200 \cdot d \cdot a^{-1}$

release during life cycle to air:

$RELEASE := TONNAGE \cdot f_{emission}$

$RELEASE = 2.5 \cdot tonne \cdot a^{-1}$

local emission during episode to air:

$$Elocal_{air} := \frac{Fmainsource \cdot RELEASE}{Temission}$$

$Elocal_{air} = 12.5 \cdot kg \cdot d^{-1}$

concentration in air at source strength of 1kg/d

$Cstd_{air} := 2.78 \cdot 10^{-4} \cdot mg \cdot m^{-3} \cdot kg^{-1} \cdot d$

fraction of the emission to air from STP (App.II)

$Fstp_{air} := 0.1 \cdot \%$

local emission rate to water during emission episode

$Elocal_{water} := 10 \cdot kg \cdot d^{-1}$

local emission to air from STP during emission episode

$Estp_{air} := Fstp_{air} \cdot Elocal_{water}$

$Estp_{air} = 0.01 \cdot kg \cdot d^{-1}$

local concentation in air during emission episode:

$Clocal_{air} := wenn\left(Elocal_{air} > Estp_{air}, Elocal_{air} \cdot Cstd_{air}, Estp_{air} \cdot Cstd_{air}\right)$

$Clocal_{air} = 3.475 \cdot 10^{-3} \cdot mg \cdot m^{-3}$

annual average concentration in air, 100m from point source

$$Clocal_{air\_ann} := Clocal_{air} \cdot \frac{Temission}{365 \cdot d \cdot a^{-1}}$$

$Clocal_{air\_ann} = 1.904 \cdot 10^{-3} \cdot mg \cdot m^{-3}$

regional concentration in air

$PECregional_{air} := 9.45 \cdot 10^{-5} \cdot mg \cdot m^{-3}$

annual average predicted environmental concentration in air

$PEClocal_{air\_ann} := Clocal_{air\_ann} + PECregional_{air}$

$PEClocal_{air\_ann} = 1.999 \cdot 10^{-3} \cdot mg \cdot m^{-3}$

Calculation of the deposition rate

standard deposition flux of aerosol-bound compounds at a source strength of 1kg/d

$$DEPstd_{aer} := 1 \cdot 10^{-2} \cdot mg \cdot m^{-2} \cdot d^{-1} \cdot kg^{-1} \cdot d$$

fraction of the chemical bound to aerosol (see: Distribution and Fate)

$$Fass_{aer} := 5.155 \cdot 10^{-8}$$

deposition flux of gaseous compounds as a function of Henry`s Law coefficient,at a source strength of 1kg/d

logH<-2	5*10 $^{-4}$ mg*m^{-2}*d^{-1}
-2<logH<2	4*10^{-4} mg*m^{-2}*d^{-1}
logH>2	3*10^{-4} mg*m^{-2}*d^{-1}

$$DEPstd_{gas} := 4 \cdot 10^{-4} \cdot mg \cdot m^{-2} \cdot d^{-1} \cdot kg^{-1} \cdot d$$

total deposition flux during emission episode

$$DEPtotal := \left(Elocal_{air} + Estp_{air}\right) \cdot \left[Fass_{aer} \cdot DEPstd_{aer} + \left(1 - Fass_{aer}\right) \cdot DEPstd_{gas}\right]$$

$$DEPtotal = 5.004 \cdot 10^{-3} \ mg \cdot m^{-2} \cdot d^{-1}$$

annual average total depostion flux

$$DEPtotal_{ann} := DEPtotal \cdot \frac{Temission}{365 \cdot d \cdot a^{-1}}$$

$$DEPtotal_{ann} = 2.742 \cdot 10^{-3} \ mg \cdot m^{-2} \cdot d^{-1}$$

Atmosphere (OPS-model)

Calculation of Clocal $_{air}$ and PEC local $_{air}$

chemical : Propanol CAS-Nr.: 71-23-8

stage of life cycle: formulation of paints
IC:5,6 UC:48 MC:Ic

$d := 86400 \cdot s$

$a := 365 \cdot d$

$mg := 1 \cdot 10^{-6} \cdot kg$

tonnage for specific scenario:

$TONNAGE := 600 \cdot tonne \cdot a^{-1}$

release factor (A 2.1):

$f_{emission} := 0.025$

fraction of main source (table B 2.10):

$Fmainsource := 1$

days of use per year (table B 2.10):

$Temission := 300 \cdot d \cdot a^{-1}$

release during life cycle to air:

$RELEASE := TONNAGE \cdot f_{emission}$

$RELEASE = 15 \cdot tonne \cdot a^{-1}$

local emission during episode to air:

$$Elocal_{air} := \frac{Fmainsource \cdot RELEASE}{Temission}$$

$Elocal_{air} = 50 \cdot kg \cdot d^{-1}$

concentration in air at source strength of 1kg/d

$Cstd_{air} := 2.78 \cdot 10^{-4} \cdot mg \cdot m^{-3} \cdot kg^{-1} \cdot d$

fraction of the emission to air from STP (App.II)

$Fstp_{air} := 0.1 \cdot \%$

local emission rate to water during emission episode

$Elocal_{water} := 40 \cdot kg \cdot d^{-1}$

local emission to air from STP during emission episode

$Estp_{air} := Fstp_{air} \cdot Elocal_{water}$

$Estp_{air} = 0.04 \cdot kg \cdot d^{-1}$

local concentation in air during emission episode:

$Clocal_{air} := wenn\left(Elocal_{air} > Estp_{air}, Elocal_{air} \cdot Cstd_{air}, Estp_{air} \cdot Cstd_{air}\right)$

$Clocal_{air} = 0.014 \cdot mg \cdot m^{-3}$

annual average concentration in air, 100m from point source

$$Clocal_{air\_ann} := Clocal_{air} \cdot \frac{Temission}{365 \cdot d \cdot a^{-1}}$$

$Clocal_{air\_ann} = 0.011 \cdot mg \cdot m^{-3}$

regional concentration in air

$PECregional_{air} := 9.45 \cdot 10^{-5} \cdot mg \cdot m^{-3}$

annual average predicted environmental concentration in air

$PEClocal_{air\_ann} := Clocal_{air\_ann} + PECregional_{air}$

$PEClocal_{air\_ann} = 0.012 \cdot mg \cdot m^{-3}$

Calculation of the deposition rate

standard deposition flux of aerosol-bound compounds at a source strength of 1kg/d

$$DEPstd_{aer} := 1 \cdot 10^{-2} \cdot mg \cdot m^{-2} \cdot d^{-1} \cdot kg^{-1} \cdot d$$

fraction of the chemical bound to aerosol (see: Distribution and Fate)

$$Fass_{aer} := 5.155 \cdot 10^{-8}$$

deposition flux of gaseous compounds as a function of Henry`s Law coefficient,at a source strength of 1kg/d

logH<-2	5*10 $^{-4}$ mg*m^{-2}*d^{-1}
-2<logH<2	4*10^{-4} mg*m^{-2}*d^{-1}
logH>2	3*10^{-4} mg*m^{-2}*d^{-1}

$$DEPstd_{gas} := 4 \cdot 10^{-4} \cdot mg \cdot m^{-2} \cdot d^{-1} \cdot kg^{-1} \cdot d$$

total deposition flux during emission episode

$$DEPtotal := \left(Elocal_{air} + Estp_{air}\right) \cdot \left[Fass_{aer} \cdot DEPstd_{aer} + \left(1 - Fass_{aer}\right) \cdot DEPstd_{gas}\right]$$

$$DEPtotal = 0.02 \circ mg \cdot m^{-2} \cdot d^{-1}$$

annual average total depostion flux

$$DEPtotal_{ann} := DEPtotal \cdot \frac{Temission}{365 \cdot d \cdot a^{-1}}$$

$$DEPtotal_{ann} = 0.016 \circ mg \cdot m^{-2} \cdot d^{-1}$$

Atmosphere (OPS-model)

Calculation of Clocal $_{air}$ and PEC local $_{air}$

chemical : Propanol CAS-Nr.: 71-23-8

stage of life cycle: processing
site identification: generic scenario

$d := 86400 \cdot s$

$a := 365 \cdot d$

$mg := 1 \cdot 10^{-6} \cdot kg$

tonnage for scenario:

$TONNAGE := 5000 \cdot tonne \cdot a^{-1}$

release factor (A 3.3, MC = 3):

$f_{emission} := 0.025$

fraction of main source:

$Fmainsource := 1$

days of use per year:

$Temission := 300 \cdot d \cdot a^{-1}$

release during life cycle to air:

$RELEASE := TONNAGE \cdot f_{emission}$

$RELEASE = 125 \cdot tonne \cdot a^{-1}$

local emission during episode to air:

$$Elocal_{air} := \frac{Fmainsource \cdot RELEASE}{Temission}$$

$Elocal_{air} = 416.667 \cdot kg \cdot d^{-1}$

concentration in air at source strength of 1kg/d

$Cstd_{air} := 2.78 \cdot 10^{-4} \cdot mg \cdot m^{-3} \cdot kg^{-1} \cdot d$

fraction of the emission to air from STP

$Fstp_{air} := 0.1 \cdot \%$

local emission rate to water during emission episode

$Elocal_{water} := 116.67 \cdot kg \cdot d^{-1}$

local emission to air from STP during emission episode

$Estp_{air} := Fstp_{air} \cdot Elocal_{water}$

$Estp_{air} = 0.117 \cdot kg \cdot d^{-1}$

local concentation in air during emission episode:

$Clocal_{air} := wenn\left(Elocal_{air} > Estp_{air}, Elocal_{air} \cdot Cstd_{air}, Estp_{air} \cdot Cstd_{air}\right)$

$Clocal_{air} = 0.116 \cdot mg \cdot m^{-3}$

annual average concentration in air, 100m from point source

$$Clocal_{air\_ann} := Clocal_{air} \cdot \frac{Temission}{365 \cdot d \cdot a^{-1}}$$

$Clocal_{air\_ann} = 0.095 \cdot mg \cdot m^{-3}$

regional concentration in air

$PECregional_{air} := 9.45 \cdot 10^{-5} \cdot mg \cdot m^{-3}$

annual average predicted environmental concentration in air

$PEClocal_{air\_ann} := Clocal_{air\_ann} + PECregional_{air}$

$PEClocal_{air\_ann} = 0.095 \cdot mg \cdot m^{-3}$

Atmosphere (OPS-model)

Calculation of Clocal $_{air}$ and PEC local $_{air}$

chemical : Propanol CAS-Nr.: 71-23-8

stage of life cycle: processing of solvent not specified
IC:0 UC:48 MC:3

$d := 86400 \cdot s$

$a := 365 \cdot d$

$mg := 1 \cdot 10^{-6} \cdot kg$

tonnage for specific scenario:

$TONNAGE := 100 \cdot tonne \cdot a^{-1}$

release factor (A 3.16):

$f_{emission} := 0.01$

fraction of main source (table B 3.14):

$Fmainsource := 0.8$

days of use per year (table B 3.14):

$Temission := 32 \cdot d \cdot a^{-1}$

release during life cycle to air:

$RELEASE := TONNAGE \cdot f_{emission}$

$RELEASE = 1 \cdot tonne \cdot a^{-1}$

local emission during episode to air:

$$Elocal_{air} := \frac{Fmainsource \cdot RELEASE}{Temission}$$

$Elocal_{air} = 25 \cdot kg \cdot d^{-1}$

concentration in air at source strength of 1kg/d

$Cstd_{air} := 2.78 \cdot 10^{-4} \cdot mg \cdot m^{-3} \cdot kg^{-1} \cdot d$

fraction of the emission to air from STP (App.II)

$Fstp_{air} := 0.1 \cdot \%$

local emission rate to water during emission episode

$Elocal_{water} := 25 \cdot kg \cdot d^{-1}$

local emission to air from STP during emission episode

$Estp_{air} := Fstp_{air} \cdot Elocal_{water}$

$Estp_{air} = 0.025 \cdot kg \cdot d^{-1}$

local concentation in air during emission episode:

$Clocal_{air} := wenn\left(Elocal_{air} > Estp_{air}, Elocal_{air} \cdot Cstd_{air}, Estp_{air} \cdot Cstd_{air}\right)$

$Clocal_{air} = 6.95 \cdot 10^{-3} \cdot mg \cdot m^{-3}$

annual average concentration in air, 100m from point source

$$Clocal_{air\_ann} := Clocal_{air} \cdot \frac{Temission}{365 \cdot d \cdot a^{-1}}$$

$Clocal_{air\_ann} = 6.093 \cdot 10^{-4} \cdot mg \cdot m^{-3}$

regional concentration in air

$PECregional_{air} := 9.45 \cdot 10^{-5} \cdot mg \cdot m^{-3}$

annual average predicted environmental concentration in air

$PEClocal_{air\_ann} := Clocal_{air\_ann} + PECregional_{air}$

$PEClocal_{air\_ann} = 7.038 \cdot 10^{-4} \cdot mg \cdot m^{-3}$

Calculation of the deposition rate

standard deposition flux of aerosol-bound compounds at a source strength of 1kg/d

$$DEPstd_{aer} := 1 \cdot 10^{-2} \cdot mg \cdot m^{-2} \cdot d^{-1} \cdot kg^{-1} \cdot d$$

fraction of the chemical bound to aerosol (see: Distribution and Fate)

$$Fass_{aer} := 5.155 \cdot 10^{-8}$$

deposition flux of gaseous compounds as a function of Henry`s Law coefficient,at a source strength of 1kg/d

logH<-2	5*10 $^{-4}$ mg*m^{-2}*d^{-1}
-2<logH<2	4*10^{-4} mg*m^{-2}*d^{-1}
logH>2	3*10^{-4} mg*m^{-2}*d^{-1}

$$DEPstd_{gas} := 4 \cdot 10^{-4} \cdot mg \cdot m^{-2} \cdot d^{-1} \cdot kg^{-1} \cdot d$$

total deposition flux during emission episode

$$DEPtotal := \left(Elocal_{air} + Estp_{air}\right) \cdot \left[Fass_{aer} \cdot DEPstd_{aer} + \left(1 - Fass_{aer}\right) \cdot DEPstd_{gas}\right]$$

$$DEPtotal = 0.01 \, mg \cdot m^{-2} \cdot d^{-1}$$

annual average total depostion flux

$$DEPtotal_{ann} := DEPtotal \cdot \frac{Temission}{365 \cdot d \cdot a^{-1}}$$

$$DEPtotal_{ann} = 8.776 \cdot 10^{-4} \, mg \cdot m^{-2} \cdot d^{-1}$$

Atmosphere (OPS-model)

Calculation of Clocal $_{air}$ and PEC local $_{air}$

chemical : Propanol CAS-Nr.: 71-23-8

stage of life cycle: processing of paints in paint shops

$d := 86400 \cdot s$

$a := 365 \cdot d$

$mg := 1 \cdot 10^{-6} \cdot kg$

tonnage for specific scenario:

$TONNAGE := 600 \cdot tonne \cdot a^{-1}$

release factor (table A 3.15):

$f_{emission} := 0.9$

fraction of main source (table B 3.13):

$Fmainsource := 0.15$

days of use per year (table B 3.13):

$Temission := 300 \cdot d \cdot a^{-1}$

release during life cycle to air:

$RELEASE := TONNAGE \cdot f_{emission}$

$RELEASE = 540 \cdot tonne \cdot a^{-1}$

local emission during episode to air:

$$Elocal_{air} := \frac{Fmainsource \cdot RELEASE}{Temission}$$

$Elocal_{air} = 270 \cdot kg \cdot d^{-1}$

concentration in air at source strength of 1kg/d

$Cstd_{air} := 2.78 \cdot 10^{-4} \cdot mg \cdot m^{-3} \cdot kg^{-1} \cdot d$

fraction of the emission to air from STP (App.II)

$Fstp_{air} := 0.1 \cdot \%$

local emission rate to water during emission episode

$Elocal_{water} := 6 \cdot kg \cdot d^{-1}$

local emission to air from STP during emission episode

$Estp_{air} := Fstp_{air} \cdot Elocal_{water}$

$Estp_{air} = 6 \cdot 10^{-3} \cdot kg \cdot d^{-1}$

local concentation in air during emission episode:

$$Clocal_{air} := wenn\left(Elocal_{air} > Estp_{air}, Elocal_{air} \cdot Cstd_{air}, Estp_{air} \cdot Cstd_{air}\right)$$

$Clocal_{air} = 0.075 \cdot mg \cdot m^{-3}$

annual average concentration in air, 100m from point source

$$Clocal_{air\_ann} := Clocal_{air} \cdot \frac{Temission}{365 \cdot d \cdot a^{-1}}$$

$Clocal_{air\_ann} = 0.062 \cdot mg \cdot m^{-3}$

regional concentration in air

$PECregional_{air} := 9.45 \cdot 10^{-5} \cdot mg \cdot m^{-3}$

annual average predicted environmental concentration in air

$PEClocal_{air\_ann} := Clocal_{air\_ann} + PECregional_{air}$

$PEClocal_{air\_ann} = 0.062 \cdot mg \cdot m^{-3}$

Calculation of the deposition rate

standard deposition flux of aerosol-bound compounds at a source strength of 1kg/d

$$DEPstd_{aer} := 1 \cdot 10^{-2} \cdot mg \cdot m^{-2} \cdot d^{-1} \cdot kg^{-1} \cdot d$$

fraction of the chemical bound to aerosol (see: Distribution and Fate)

$$Fass_{aer} := 5.155 \cdot 10^{-8}$$

deposition flux of gaseous compounds as a function of Henry`s Law coefficient,at a source strength of 1kg/d

logH<-2	5*10 $^{-4}$ mg*m^{-2}*d^{-1}
-2<logH<2	4*10^{-4} mg*m^{-2}*d^{-1}
logH>2	3*10^{-4} mg*m^{-2}*d^{-1}

$$DEPstd_{gas} := 4 \cdot 10^{-4} \cdot mg \cdot m^{-2} \cdot d^{-1} \cdot kg^{-1} \cdot d$$

total deposition flux during emission episode

$$DEPtotal := \left(Elocal_{air} + Estp_{air}\right) \cdot \left[Fass_{aer} \cdot DEPstd_{aer} + \left(1 - Fass_{aer}\right) \cdot DEPstd_{gas}\right]$$

$$DEPtotal = 0.108 \, mg \cdot m^{-2} \cdot d^{-1}$$

annual average total depostion flux

$$DEPtotal_{ann} := DEPtotal \cdot \frac{Temission}{365 \cdot d \cdot a^{-1}}$$

$$DEPtotal_{ann} = 0.089 \, mg \cdot m^{-2} \cdot d^{-1}$$

Atmosphere (OPS-model)

Calculation of Clocal $_{air}$ and PEC local $_{air}$

chemical : Propanol CAS-Nr.: 71-23-8

stage of life cycle: privat use of paints

$d := 86400 \cdot s$

$a := 365 \cdot d$

$mg := 1 \cdot 10^{-6} \cdot kg$

tonnage for specific scenario:

$TONNAGE := 600 \cdot tonne \cdot a^{-1}$

release factor (table A 4.5):

$f_{emission} := 0.95$

fraction of main source (table B 4.5):

$Fmainsource := 0.0004$

days of use per year (table B 4.5):

$Temission := 300 \cdot d \cdot a^{-1}$

release during life cycle to air:

$RELEASE := TONNAGE \cdot f_{emission}$

$RELEASE = 570 \cdot tonne \cdot a^{-1}$

local emission during episode to air:

$$Elocal_{air} := \frac{Fmainsource \cdot RELEASE}{Temission}$$

$Elocal_{air} = 0.76 \cdot kg \cdot d^{-1}$

concentration in air at source strength of 1kg/d

$Cstd_{air} := 2.78 \cdot 10^{-4} \cdot mg \cdot m^{-3} \cdot kg^{-1} \cdot d$

fraction of the emission to air from STP (App.II)

$Fstp_{air} := 0.1 \cdot \%$

local emission rate to water during emission episode

$Elocal_{water} := 0.03 \cdot kg \cdot d^{-1}$

local emission to air from STP during emission episode

$Estp_{air} := Fstp_{air} \cdot Elocal_{water}$

$Estp_{air} = 3 \cdot 10^{-5} \cdot kg \cdot d^{-1}$

local concentation in air during emission episode:

$Clocal_{air} := wenn\left(Elocal_{air} > Estp_{air}, Elocal_{air} \cdot Cstd_{air}, Estp_{air} \cdot Cstd_{air}\right)$

$Clocal_{air} = 2.113 \cdot 10^{-4} \cdot mg \cdot m^{-3}$

annual average concentration in air, 100m from point source

$$Clocal_{air\_ann} := Clocal_{air} \cdot \frac{Temission}{365 \cdot d \cdot a^{-1}}$$

$Clocal_{air\_ann} = 1.737 \cdot 10^{-4} \cdot mg \cdot m^{-3}$

regional concentration in air

$PECregional_{air} := 9.45 \cdot 10^{-5} \cdot mg \cdot m^{-3}$

annual average predicted environmental concentration in air

$PEClocal_{air\_ann} := Clocal_{air\_ann} + PECregional_{air}$

$PEClocal_{air\_ann} = 2.682 \cdot 10^{-4} \cdot mg \cdot m^{-3}$

Calculation of the deposition rate

standard deposition flux of aerosol-bound compounds at a source strength of 1kg/d

$$DEPstd_{aer} := 1 \cdot 10^{-2} \cdot mg \cdot m^{-2} \cdot d^{-1} \cdot kg^{-1} \cdot d$$

fraction of the chemical bound to aerosol (see: Distribution and Fate)

$$Fass_{aer} := 5.155 \cdot 10^{-8}$$

deposition flux of gaseous compounds as a function of Henry`s Law coefficient,at a source strength of 1kg/d

logH<-2	5*10 $^{-4}$ mg*m^{-2}*d^{-1}
-2<logH<2	4*10^{-4} mg*m^{-2}*d^{-1}
logH>2	3*10^{-4} mg*m^{-2}*d^{-1}

$$DEPstd_{gas} := 4 \cdot 10^{-4} \cdot mg \cdot m^{-2} \cdot d^{-1} \cdot kg^{-1} \cdot d$$

total deposition flux during emission episode

$$DEPtotal := \left(Elocal_{air} + Estp_{air}\right) \cdot \left[Fass_{aer} \cdot DEPstd_{aer} + \left(1 - Fass_{aer}\right) \cdot DEPstd_{gas}\right]$$

$$DEPtotal = 3.04 \cdot 10^{-4} \ mg \cdot m^{-2} \cdot d^{-1}$$

annual average total depostion flux

$$DEPtotal_{ann} := DEPtotal \cdot \frac{Temission}{365 \cdot d \cdot a^{-1}}$$

$$DEPtotal_{ann} = 2.49873 \cdot 10^{-4} \ mg \cdot m^{-2} \cdot d^{-1}$$

Appendix D Exposure of soil

Exposure of Soil

chemical : Propanol CAS-Nr.:71-23-8
formulation of household chemicals

$d := 86400 \cdot s$

$ppm := mg \cdot kg^{-1}$

$a := 365 \cdot d$

$i := 1..3$

annual average total deposition flux:

$DEPtotal_{ann} := 0.011 \cdot mg \cdot m^{-2} \cdot d^{-1}$

soil-water partitioning coefficient:

$K_{soil\_water} := 0.329$

concentration in dry sewage sludge:

$C_{sludge} := 0 \cdot mg \cdot kg^{-1}$

air-water partitioning coefficient:

$K_{air\_water} := 0.000048$

rate constant for for removal from top soil:

$kbio_{soil} := 0.023 \cdot d^{-1}$

PECregional:

$PECregional_{natural\_soil} := 5.25 \cdot 10^{-4} \cdot mg \cdot kg^{-1}$

Defaults:

mixing depth of soil:

$DEPTHsoil_i :=$

0.2·m
0.2·m
0.1·m

bulk density of soil:

$RHO_{soil} := 1700 \cdot kg \cdot m^{-3}$

average time for exposure:

$T_i :=$

30·d
180·d
180·d

partial mass transfer coefficient at air-side of the air-soil interface:

$kasl_{air} := 120 \cdot m \cdot d^{-1}$

partial mass transfer coefficient at soilair-side of the air-soil interface:

$kasl_{soilair} := 0.48 \cdot m \cdot d^{-1}$

partial mass transfer coefficient at soilwater-side of the air-soil interface:

$kasl_{soilwater} := 4.8 \cdot 10^{-5} \cdot m \cdot d^{-1}$

fraction of rain water that infiltrates into soil:

$Finf_{soil} := 0.25$

rate of wet precipitation:

$RAINrate := 1.92 \cdot 10^{-3} \cdot m \cdot d^{-1}$

dry sludge application rate:

$$APPLsludge_i :=$$

$0.5 \cdot kg \cdot m^{-2} \cdot a^{-1}$
$0.5 \cdot kg \cdot m^{-2} \cdot a^{-1}$
$0.1 \cdot kg \cdot m^{-2} \cdot a^{-1}$

Calculation:

aerial deposition flux per kg of soil:

$$D_{air_i} := \frac{DEPtotal_{ann}}{DEPTHsoil_i \cdot RHO_{soil}}$$

rate constant for valatilisation from soil:

$$k_{volat_i} := \left[\left(\frac{1}{kasl_{air} \cdot K_{air\_water}} + \frac{1}{kasl_{soilair} \cdot K_{air\_water} + kasl_{soilwater}} \right) \cdot K_{soil\_water} \cdot DEPTHsoil_i \right]^{-1}$$

rate constant for leaching from soil layer:

$$k_{leach_i} := \frac{Finf_{soil} \cdot RAINrate}{K_{soil\_water} \cdot DEPTHsoil_i}$$

removal from top soil:

$$k_i := k_{volat_i} + k_{leach_i} + kbio_{soil}$$

concentration in soil

concentration in soil due to 10 years of continuous deposition:

$$Cdep_{soil\_10_i} := \frac{D_{air_i}}{k_i} \cdot \left(1 - \exp\left(-365 \cdot d \cdot 10 \cdot k_i\right)\right)$$

concentration just after the first year of sludge application:

$$Csludge_{soil\_1_i} := \frac{C_{sludge} \cdot APPLsludge_i \cdot a}{DEPTHsoil_i \cdot RHO_{soil}}$$

initial concentration in soil after 10 applications of sludge:

$$Csludge_{soil\_10_i} := Csludge_{soil\_1_i} \cdot \left[1 + \left[\sum_{n=1}^{9} \left(\exp\left(-365 \cdot d \cdot k_i\right)^n \right) \right] \right]$$

sum of the concentrations due to both processes:

$$C_{soil\_10_i} := Cdep_{soil\_10_i} + Csludge_{soil\_10_i}$$

average concentration in soil over T days:

$$Clocal_{soil_i} := \frac{D_{air_i}}{k_i} + \frac{1}{k_i \cdot T_i} \cdot \left(C_{soil\_10_i} - \frac{D_{air_i}}{k_i} \right) \cdot \left(1 - \exp\left(-k_i \cdot T_i \right) \right)$$

$$PEClocal_{soil_i} := Clocal_{soil_i} + PECregional_{natural\_soil}$$

	$\frac{Clocal_{soil_i}}{ppm}$
$Clocal_{soil}$ =	$1.032 \cdot 10^{-3}$
$Clocal_{agr.soil}$ =	$1.032 \cdot 10^{-3}$
$Clocal_{grassland}$ =	$1.629 \cdot 10^{-3}$

	$\frac{PEClocal_{soil_i}}{ppm}$
$PEClocal_{soil}$ =	$1.557 \cdot 10^{-3}$
$PEClocal_{agr.soil}$ =	$1.557 \cdot 10^{-3}$
$PEClocal_{grassland}$ =	$2.154 \cdot 10^{-3}$

Indicating persistency of the substance in soil

initial concentration after 10 years:

$\frac{C_{soil\_10_i}}{ppm}$
$1.032 \cdot 10^{-3}$
$1.032 \cdot 10^{-3}$
$1.629 \cdot 10^{-3}$

initial concentration in steady-state situation:

$$Facc_i := e^{-365 \cdot d \cdot k_i}$$

$$C_{soil\_ss_i} := \frac{D_{air_i}}{k_i} + Csludge_{soil\_1_i} \cdot \frac{1}{1 - Facc_i}$$

$\frac{C_{soil\_ss_i}}{ppm}$
$1.032 \cdot 10^{-3}$
$1.032 \cdot 10^{-3}$
$1.629 \cdot 10^{-3}$

fraction of steady-state in soil achieved:

$$Fst\_st_i := \frac{C_{soil\_10_i}}{C_{soil\_ss_i}}$$

Fst_st_i
1
1
1

concentration in pore water

$$Clocal_{soil\_porew_i} := \frac{Clocal_{soil_i} \cdot RHO_{soil}}{K_{soil\_water}}$$

	$Clocal_{soil\_porew_i}$ / mg·l^{-1}
$Clocal_{soil\_porew}$ =	$5.330561 \cdot 10^{-3}$
$Clocal_{agr.soil\_porew}$ =	$5.330561 \cdot 10^{-3}$
$Clocal_{grassland\_porew}$ =	$8.417031 \cdot 10^{-3}$

$$PEClocal_{soil\_porew_i} := \frac{PEClocal_{soil_i} \cdot RHO_{soil}}{K_{soil\_water}}$$

	$PEClocal_{soil\_porew_i}$ / mg·l^{-1}
$PEClocal_{soil\_porew}$ =	$8.043321 \cdot 10^{-3}$
$PEClocal_{agr.soil\_porew}$ =	$8.043321 \cdot 10^{-3}$
$PEClocal_{grassland\_porew}$ =	0.01113

concentration in ground water

$PEClocal_{grw} = PEClocal_{agr\_soil\_porew}$

Exposure of Soil

$d := 86400 \cdot s$

$ppm := mg \cdot kg^{-1}$ $a := 365 \cdot d$

$i := 1 .. 3$

chemical : Propanol CAS-Nr.:71-23-8
Formulation of solvents (not specified)

annual average total deposition flux:

$DEPtotal_{ann} := 0.00274 mg \cdot m^{-2} \cdot d^{-1}$

soil-water partitioning coefficient:

$K_{soil\_water} := 0.329$

concentration in dry sewage sludge:

$C_{sludge} := 0 \cdot mg \cdot kg^{-1}$

air-water partitioning coefficient:

$K_{air\_water} := 0.000048$

rate constant for for removal from top soil:

$kbio_{soil} := 0.023 \cdot d^{-1}$

PECregional:

$PECregional_{natural\_soil} := 5.25 \cdot 10^{-4} \cdot mg \cdot kg^{-1}$

Defaults:

mixing depth of soil:

$DEPTHsoil_i :=$

$0.2 \cdot m$
$0.2 \cdot m$
$0.1 \cdot m$

bulk density of soil:

$RHO_{soil} := 1700 \cdot kg \cdot m^{-3}$

average time for exposure:

$T_i :=$

$30 \cdot d$
$180 \cdot d$
$180 \cdot d$

partial mass transfer coefficient at air-side of the air-soil interface:

$kasl_{air} := 120 \cdot m \cdot d^{-1}$

partial mass transfer coefficient at soilair-side of the air-soil interface:

$kasl_{soilair} := 0.48 \cdot m \cdot d^{-1}$

partial mass transfer coefficient at soilwater-side of the air-soil interface:

$kasl_{soilwater} := 4.8 \cdot 10^{-5} \cdot m \cdot d^{-1}$

fraction of rain water that infiltrates into soil:

$Finf_{soil} := 0.25$

rate of wet precipitation:

$RAINrate := 1.92 \cdot 10^{-3} \cdot m \cdot d^{-1}$

dry sludge application rate:

$$APPLsludge_i :=$$

$0.5 \cdot kg \cdot m^{-2} \cdot a^{-1}$
$0.5 \cdot kg \cdot m^{-2} \cdot a^{-1}$
$0.1 \cdot kg \cdot m^{-2} \cdot a^{-1}$

Calculation:

aerial deposition flux per kg of soil:

$$D_{air_i} := \frac{DEPtotal_{ann}}{DEPTHsoil_i \cdot RHO_{soil}}$$

rate constant for valatilisation from soil:

$$k_{volat_i} := \left[\left(\frac{1}{kasl_{air} \cdot K_{air\_water}} + \frac{1}{kasl_{soilair} \cdot K_{air\_water} + kasl_{soilwater}} \right) \cdot K_{soil\_water} \cdot DEPTHsoil_i \right]^{-1}$$

rate constant for leaching from soil layer:

$$k_{leach_i} := \frac{Finf_{soil} \cdot RAINrate}{K_{soil\_water} \cdot DEPTHsoil_i}$$

removal from top soil:

$$k_i := k_{volat_i} + k_{leach_i} + kbio_{soil}$$

concentration in soil

concentration in soil due to 10 years of continuous deposition:

$$Cdep_{soil\_10_i} := \frac{D_{air_i}}{k_i} \cdot \left(1 - \exp\left(-365 \cdot d \cdot 10 \cdot k_i\right)\right)$$

concentration just after the first year of sludge application:

$$Csludge_{soil\_1_i} := \frac{C_{sludge} \cdot APPLsludge_i \cdot a}{DEPTHsoil_i \cdot RHO_{soil}}$$

initial concentration in soil after 10 applications of sludge:

$$Csludge_{soil\_10_i} := Csludge_{soil\_1_i} \cdot \left[1 + \left[\sum_{n=1}^{9} \left(\exp\left(-365 \cdot d \cdot k_i\right)^n \right) \right] \right]$$

sum of the concentrations due to both processes:

$$C_{soil\_10_i} := Cdep_{soil\_10_i} + Csludge_{soil\_10_i}$$

average concentration in soil over T days:

$$Clocal_{soil_i} := \frac{D_{air_i}}{k_i} + \frac{1}{k_i \cdot T_i} \cdot \left(C_{soil\_10_i} - \frac{D_{air_i}}{k_i} \right) \cdot \left(1 - \exp\left(-k_i \cdot T_i \right) \right)$$

$$PEClocal_{soil_i} := Clocal_{soil_i} + PECregional_{natural\_soil}$$

	$\frac{Clocal_{soil_i}}{ppm}$		$\frac{PEClocal_{soil_i}}{ppm}$
$Clocal_{soil} =$	$2.57 \cdot 10^{-4}$	$PEClocal_{soil} =$	$7.82 \cdot 10^{-4}$
$Clocal_{agr.soil} =$	$2.57 \cdot 10^{-4}$	$PEClocal_{agr.soil} =$	$7.82 \cdot 10^{-4}$
$Clocal_{grassland} =$	$4.058 \cdot 10^{-4}$	$PEClocal_{grassland} =$	$9.308 \cdot 10^{-4}$

Indicating persistency of the substance in soil

initial concentration after 10 years:

$\frac{C_{soil\_10_i}}{ppm}$
$2.57 \cdot 10^{-4}$
$2.57 \cdot 10^{-4}$
$4.058 \cdot 10^{-4}$

initial concentration in steady-state situation:

$$Facc_i := e^{-365 \cdot d \cdot k_i}$$

$$C_{soil\_ss_i} := \frac{D_{air_i}}{k_i} + Csludge_{soil\_1_i} \cdot \frac{1}{1 - Facc_i}$$

$\frac{C_{soil\_ss_i}}{ppm}$
$2.57 \cdot 10^{-4}$
$2.57 \cdot 10^{-4}$
$4.058 \cdot 10^{-4}$

fraction of steady-state in soil achieved:

$$Fst\_st_i := \frac{C_{soil\_10_i}}{C_{soil\_ss_i}}$$

Fst_st_i
1
1
1

concentration in pore water

$$Clocal_{soil\_porew_i} := \frac{Clocal_{soil_i} \cdot RHO_{soil}}{K_{soil\_water}}$$

	$\frac{Clocal_{soil\_porew_i}}{mg \cdot l^{-1}}$
$Clocal_{soil\_porew}$ =	$1.327791 \cdot 10^{-3}$
$Clocal_{agr.soil\_porew}$ =	$1.327791 \cdot 10^{-3}$
$Clocal_{grassland\_porew}$ =	$2.09661 \cdot 10^{-3}$

$$PEClocal_{soil\_porew_i} := \frac{PEClocal_{soil_i} \cdot RHO_{soil}}{K_{soil\_water}}$$

	$\frac{PEClocal_{soil\_porew_i}}{mg \cdot l^{-1}}$
$PEClocal_{soil\_porew}$ =	$4.040561 \cdot 10^{-3}$
$PEClocal_{agr.soil\_porew}$ =	$4.040561 \cdot 10^{-3}$
$PEClocal_{grassland\_porew}$ =	$4.80937 \cdot 10^{-3}$

concentration in ground water

$PEClocal_{grw} = PEClocal_{agr\_soil\_porew}$

Exposure of Soil

chemical : Propanol CAS-Nr.:71-23-8
processing generic

$d := 86400 \cdot s$

$ppm := mg \cdot kg^{-1}$

$a := 365 \cdot d$

$i := 1..3$

annual average total deposition flux:

$DEPtotal_{ann} := 0.137 \cdot mg \cdot m^{-2} \cdot d^{-1}$

soil-water partitioning coefficient:

$K_{soil\_water} := 0.329$

concentration in dry sewage sludge:

$C_{sludge} := 0 \cdot mg \cdot kg^{-1}$

air-water partitioning coefficient:

$K_{air\_water} := 0.000048$

rate constant for for removal from top soil:

$kbio_{soil} := 0.023 \cdot d^{-1}$

PECregional:

$PECregional_{natural\_soil} := 5.25 \cdot 10^{-4} \cdot mg \cdot kg^{-1}$

Defaults:

mixing depth of soil:

$DEPTHsoil_i :=$

$0.2 \cdot m$
$0.2 \cdot m$
$0.1 \cdot m$

bulk density of soil:

$RHO_{soil} := 1700 \cdot kg \cdot m^{-3}$

average time for exposure:

$T_i :=$

$30 \cdot d$
$180 \cdot d$
$180 \cdot d$

partial mass transfer coefficient at air-side of the air-soil interface:

$kasl_{air} := 120 \cdot m \cdot d^{-1}$

partial mass transfer coefficient at soilair-side of the air-soil interface:

$kasl_{soilair} := 0.48 \cdot m \cdot d^{-1}$

partial mass transfer coefficient at soilwater-side of the air-soil interface:

$kasl_{soilwater} := 4.8 \cdot 10^{-5} \cdot m \cdot d^{-1}$

fraction of rain water that infiltrates into soil:

$Finf_{soil} := 0.25$

rate of wet precipitation:

$RAINrate := 1.92 \cdot 10^{-3} \cdot m \cdot d^{-1}$

dry sludge application rate:

$$APPLsludge_i :=$$

$0.5 \cdot kg \cdot m^{-2} \cdot a^{-1}$
$0.5 \cdot kg \cdot m^{-2} \cdot a^{-1}$
$0.1 \cdot kg \cdot m^{-2} \cdot a^{-1}$

Calculation:

aerial deposition flux per kg of soil:

$$D_{air_i} := \frac{DEPtotal_{ann}}{DEPTHsoil_i \cdot RHO_{soil}}$$

rate constant for valatilisation from soil:

$$k_{volat_i} := \left[\left(\frac{1}{kasl_{air} \cdot K_{air\_water}} + \frac{1}{kasl_{soilair} \cdot K_{air\_water} + kasl_{soilwater}} \right) \cdot K_{soil\_water} \cdot DEPTHsoil_i \right]^{-1}$$

rate constant for leaching from soil layer:

$$k_{leach_i} := \frac{Finf_{soil} \cdot RAINrate}{K_{soil\_water} \cdot DEPTHsoil_i}$$

removal from top soil:

$$k_i := k_{volat_i} + k_{leach_i} + kbio_{soil}$$

concentration in soil

concentration in soil due to 10 years of continuous deposition:

$$Cdep_{soil\_10_i} := \frac{D_{air_i}}{k_i} \cdot \left(1 - \exp\left(-365 \cdot d \cdot 10 \cdot k_i\right)\right)$$

concentration just after the first year of sludge application:

$$Csludge_{soil\_1_i} := \frac{C_{sludge} \cdot APPLsludge_i \cdot a}{DEPTHsoil_i \cdot RHO_{soil}}$$

initial concentration in soil after 10 applications of sludge:

$$Csludge_{soil\_10_i} := Csludge_{soil\_1_i} \cdot \left[1 + \left[\sum_{n=1}^{9} \left(\exp\left(-365 \cdot d \cdot k_i\right)^n \right) \right] \right]$$

sum of the concentrations due to both processes:

$$C_{soil\_10_i} := Cdep_{soil\_10_i} + Csludge_{soil\_10_i}$$

average concentration in soil over T days:

$$Clocal_{soil_i} := \frac{D_{air_i}}{k_i} + \frac{1}{k_i \cdot T_i} \cdot \left(C_{soil\_10_i} - \frac{D_{air_i}}{k_i} \right) \cdot \left(1 - \exp\left(-k_i \cdot T_i \right) \right)$$

$$PEClocal_{soil_i} := Clocal_{soil_i} + PECregional_{natural\_soil}$$

	$\frac{Clocal_{soil_i}}{ppm}$		$\frac{PEClocal_{soil_i}}{ppm}$
$Clocal_{soil}$ =	$1.285 \cdot 10^{-2}$	$PEClocal_{soil}$ =	$1.337 \cdot 10^{-2}$
$Clocal_{agr.soil}$ =	$1.285 \cdot 10^{-2}$	$PEClocal_{agr.soil}$ =	$1.337 \cdot 10^{-2}$
$Clocal_{grassland}$ =	$2.029 \cdot 10^{-2}$	$PEClocal_{grassland}$ =	$2.081 \cdot 10^{-2}$

Indicating persistency of the substance in soil

initial concentration after 10 years:

$\frac{C_{soil\_10_i}}{ppm}$
$1.285 \cdot 10^{-2}$
$1.285 \cdot 10^{-2}$
$2.029 \cdot 10^{-2}$

initial concentration in steady-state situation:

$$Facc_i := e^{-365 \cdot d \cdot k_i}$$

$$C_{soil\_ss_i} := \frac{D_{air_i}}{k_i} + Csludge_{soil\_1_i} \cdot \frac{1}{1 - Facc_i}$$

$\frac{C_{soil\_ss_i}}{ppm}$
$1.285 \cdot 10^{-2}$
$1.285 \cdot 10^{-2}$
$2.029 \cdot 10^{-2}$

fraction of steady-state in soil achieved:

$$Fst\_st_i := \frac{C_{soil\_10_i}}{C_{soil\_ss_i}}$$

Fst_st_i
1
1
1

concentration in pore water

$$Clocal_{soil\_porew_i} := \frac{Clocal_{soil_i} \cdot RHO_{soil}}{K_{soil\_water}}$$

	$Clocal_{soil\_porew_i}$ / mg·l^{-1}
$Clocal_{soil\_porew}$ =	$6.6389 \cdot 10^{-2}$
$Clocal_{agr.soil\_porew}$ =	$6.6389 \cdot 10^{-2}$
$Clocal_{grassland\_porew}$ =	0.10483

$$PEClocal_{soil\_porew_i} := \frac{PEClocal_{soil_i} \cdot RHO_{soil}}{K_{soil\_water}}$$

	$PEClocal_{soil\_porew_i}$ / mg·l^{-1}
$PEClocal_{soil\_porew}$ =	0.0691
$PEClocal_{agr.soil\_porew}$ =	0.0691
$PEClocal_{grassland\_porew}$ =	0.10754

concentration in ground water

$PEClocal_{grw} = PEClocal_{agr\_soil\_porew}$

Exposure of Soil

$d := 86400 \cdot s$

$ppm := mg \cdot kg^{-1}$ $a := 365 \cdot d$

$i := 1..3$

chemical : Propanol CAS-Nr.:71-23-8
Processing of paints in paint shops

annual average total deposition flux:

$DEPtotal_{ann} := 0.089 \cdot mg \cdot m^{-2} \cdot d^{-1}$

soil-water partitioning coefficient:

$K_{soil\_water} := 0.329$

concentration in dry sewage sludge:

$C_{sludge} := 0 \cdot mg \cdot kg^{-1}$

air-water partitioning coefficient:

$K_{air\_water} := 0.000048$

rate constant for for removal from top soil:

$kbio_{soil} := 0.023 \cdot d^{-1}$

PECregional:

$PECregional_{natural\_soil} := 5.25 \cdot 10^{-4} \cdot mg \cdot kg^{-1}$

Defaults:

mixing depth of soil:

$DEPTHsoil_i :=$

$0.2 \cdot m$
$0.2 \cdot m$
$0.1 \cdot m$

bulk density of soil:

$RHO_{soil} := 1700 \cdot kg \cdot m^{-3}$

average time for exposure:

$T_i :=$

$30 \cdot d$
$180 \cdot d$
$180 \cdot d$

partial mass transfer coefficient at air-side of the air-soil interface:

$kasl_{air} := 120 \cdot m \cdot d^{-1}$

partial mass transfer coefficient at soilair-side of the air-soil interface:

$kasl_{soilair} := 0.48 \cdot m \cdot d^{-1}$

partial mass transfer coefficient at soilwater-side of the air-soil interface:

$kasl_{soilwater} := 4.8 \cdot 10^{-5} \cdot m \cdot d^{-1}$

fraction of rain water that infiltrates into soil:

$Finf_{soil} := 0.25$

rate of wet precipitation:

$RAINrate := 1.92 \cdot 10^{-3} \cdot m \cdot d^{-1}$

dry sludge application rate:

$$APPLsludge_i :=$$

$0.5 \cdot kg \cdot m^{-2} \cdot a^{-1}$
$0.5 \cdot kg \cdot m^{-2} \cdot a^{-1}$
$0.1 \cdot kg \cdot m^{-2} \cdot a^{-1}$

Calculation:

aerial deposition flux per kg of soil:

$$D_{air_i} := \frac{DEPtotal_{ann}}{DEPTHsoil_i \cdot RHO_{soil}}$$

rate constant for valatilisation from soil:

$$k_{volat_i} := \left[\left(\frac{1}{kasl_{air} \cdot K_{air\_water}} + \frac{1}{kasl_{soilair} \cdot K_{air\_water} + kasl_{soilwater}} \right) \cdot K_{soil\_water} \cdot DEPTHsoil_i \right]^{-1}$$

rate constant for leaching from soil layer:

$$k_{leach_i} := \frac{Finf_{soil} \cdot RAINrate}{K_{soil\_water} \cdot DEPTHsoil_i}$$

removal from top soil:

$$k_i := k_{volat_i} + k_{leach_i} + kbio_{soil}$$

concentration in soil

concentration in soil due to 10 years of continuous deposition:

$$Cdep_{soil\_10_i} := \frac{D_{air_i}}{k_i} \cdot \left(1 - \exp\left(-365 \cdot d \cdot 10 \cdot k_i\right)\right)$$

concentration just after the first year of sludge application:

$$Csludge_{soil\_1_i} := \frac{C_{sludge} \cdot APPLsludge_i \cdot a}{DEPTHsoil_i \cdot RHO_{soil}}$$

initial concentration in soil after 10 applications of sludge:

$$Csludge_{soil\_10_i} := Csludge_{soil\_1_i} \cdot \left[1 + \left[\sum_{n=1}^{9} \left(\exp\left(-365 \cdot d \cdot k_i\right)^n \right) \right] \right]$$

sum of the concentrations due to both processes:

$$C_{soil\_10_i} := Cdep_{soil\_10_i} + Csludge_{soil\_10_i}$$

average concentration in soil over T days:

$$Clocal_{soil_i} := \frac{D_{air_i}}{k_i} + \frac{1}{k_i \cdot T_i} \cdot \left(C_{soil\_10_i} - \frac{D_{air_i}}{k_i} \right) \cdot \left(1 - \exp\left(-k_i \cdot T_i \right) \right)$$

$$PEClocal_{soil_i} := Clocal_{soil_i} + PECregional_{natural\_soil}$$

	$\frac{Clocal_{soil_i}}{ppm}$		$\frac{PEClocal_{soil_i}}{ppm}$
$Clocal_{soil} =$	$8.347 \cdot 10^{-3}$	$PEClocal_{soil} =$	$8.872 \cdot 10^{-3}$
$Clocal_{agr.soil} =$	$8.347 \cdot 10^{-3}$	$PEClocal_{agr.soil} =$	$8.872 \cdot 10^{-3}$
$Clocal_{grassland} =$	$1.318 \cdot 10^{-2}$	$PEClocal_{grassland} =$	$1.37 \cdot 10^{-2}$

Indicating persistency of the substance in soil

initial concentration after 10 years:

$\frac{C_{soil\_10_i}}{ppm}$
$8.347 \cdot 10^{-3}$
$8.347 \cdot 10^{-3}$
$1.318 \cdot 10^{-2}$

initial concentration in steady-state situation:

$$Facc_i := e^{-365 \cdot d \cdot k_i}$$

$$C_{soil\_ss_i} := \frac{D_{air_i}}{k_i} + Csludge_{soil\_1_i} \cdot \frac{1}{1 - Facc_i}$$

$\frac{C_{soil\_ss_i}}{ppm}$
$8.347 \cdot 10^{-3}$
$8.347 \cdot 10^{-3}$
$1.318 \cdot 10^{-2}$

fraction of steady-state in soil achieved:

$$Fst\_st_i := \frac{C_{soil\_10_i}}{C_{soil\_ss_i}}$$

Fst_st_i
1
1
1

concentration in pore water

$$Clocal_{soil\_porew_i} := \frac{Clocal_{soil_i} \cdot RHO_{soil}}{K_{soil\_water}}$$

	$Clocal_{soil\_porew_i}$ / mg·l^{-1}
$Clocal_{soil\_porew}$ =	$4.3129 \cdot 10^{-2}$
$Clocal_{agr.soil\_porew}$ =	$4.3129 \cdot 10^{-2}$
$Clocal_{grassland\_porew}$ =	$6.81014 \cdot 10^{-2}$

$$PEClocal_{soil\_porew_i} := \frac{PEClocal_{soil_i} \cdot RHO_{soil}}{K_{soil\_water}}$$

	$PEClocal_{soil\_porew_i}$ / mg·l^{-1}
$PEClocal_{soil\_porew}$ =	0.04584
$PEClocal_{agr.soil\_porew}$ =	0.04584
$PEClocal_{grassland\_porew}$ =	0.07081

concentration in ground water

$PEClocal_{grw} = PEClocal_{agr\_soil\_porew}$

Appendix E Input and Output of propan-1-ol

SimpleBox2.0a - Berechnung regionaler + kontinentaler PEC's

- Anpassung an TGD (1996) / EUSES 1.00: Michael Feibicke (06/98)

INPUT - Propanol

Parameter names acc. SimpleBox20	Unit	Input	Parameter names according Euses
Physicochemical properties			
COMPOUND NAME	[-]	Propanol	Substance
MOL WEIGHT	[g.mol^{-1}]	60,1	Molecular weight
MELTING POINT	[° C]	-126,5	Melting Point
VAPOR PRESSURE(25)	[Pa]	1940	Vapour pressure at 25°C
log Kow	[log10]	0,34	Octanol-water partition coefficient
SOLUBILITY(25)	[mg.l^{-1}]	1000000	Water solubility
Distribution - Partition coefficients			
- Solids water partitioning (derived from K_{oc})			
Kp(soil)	[l.kg$_d^{-1}$]	0,086	Solids-water partitioning in soil
Kp(sed)	[l.kg$_d^{-1}$]	0,215	Solids-water partitioning in sediment
Kp(susp)	[l.kg$_d^{-1}$]	0,429	Solids-water partitioning in sudpended matter
- Biota-water			
BCF(fish)	[l.kg$_w^{-1}$]	1	Biocentration factor for aquatic biota
Degradation and Transfromation rates			
- Characterisation and STP			
PASSreadytest	[y / n]	y	Characterization of biodegradability
- Environmental Total Degradation			
kdeg(air)	[d^{-1}]	2,15E-01	Rate constant for degradation in air
kdeg(water)	[d^{-1}]	4,70E-02	Rate constant for degradation in bulk surface water
kdeg(soil)	[d^{-1}]	2,30E-02	Rate constant for degradation in bulk soil
kdeg(sed)	[d^{-1}]	2,30E-03	Rate constant for degradation in bulk sediment
Sewage treatment (e.g. calculated by SimpleTreat)			
- Continental			
FR(volatstp) [C]	[-]	1,00E-03	Fraction of emission directed to air (STPcont)
FR(effstp) [C]	[-]	1,26E-01	Fraction of emission directed to water (STPcont)
FR(sludgestp) [C]	[-]	0,00E+00	Fraction of emission directed to sludge (STPcont)
- Regional			
FR(volatstp) [R]	[-]	1,00E-03	Fraction of emission directed to air (STPreg)
FR(effstp) [R]	[-]	1,26E-01	Fraction of emission directed to water (STPreg)
FR(sludgestp) [R]	[-]	0,00E+00	Fraction of emission directed to sludge (STPreg)
Release estimation			
- Continental			
Edirect(air) [C]	[t.y^{-1}]	4174	Total continental emission to air
STPload [C]	[t.y^{-1}]	3016	Total continental emission to wastewater
Edirect(water1) [C]	[t.y^{-1}]	1249	Total continental emission to surface water
Edirect(soil3) [C]	[t.y^{-1}]	0	Total continental emission to industrial soil
Edirect(soil2) [C]	[t.y^{-1}]	0	Total continental emission to agricultural soil
- Regional			
Edirect(air) [R]	[t.y^{-1}]	2107	Total regional emission to air
STPload [R]	[t.y^{-1}]	1509	Total regional emission to wastewater
Edirect(water1) [R]	[t.y^{-1}]	615	Total regional emission to surface water
Edirect(soil3) [R]	[t.y^{-1}]	0	Total regional emission to industrial soil
Edirect(soil2) [R]	[t.y^{-1}]	0	Total regional emission to agricultural soil

OUTPUT - Propanol

Zur **Neuberechnung der Daten**: ->Extras ->Optionen ->Berechnen -> Datei_berechnen -> F9 drücken, sonst keine komplette Neuberechnung aller Bezüge!!

Parameter names acc. SimpleBox20	Unit	Output	Parameter names according Euses
Physicochemical properties			
COMPOUND NAME	[-]	Propanol	Substance
Output			
- Continental			
PECsurfacewater (total)	[mg.l^{-1}]	2,94E-04	Continental PEC in surface water (total)
PECsurfacewater (dissolved)	[mg.l^{-1}]	2,94E-04	Continental PEC in surface water (dissolved)
PECair	[mg.m^{-3}]	1,13E-05	Continental PEC in air (total)
PECagr.soil	[mg.kg$_{wwt}^{-1}$]	3,49E-05	Continental PEC in agricultural soil (total)
PECporewater agr.soil	[mg.l^{-1}]	1,80E-04	Continental PEC in pore water of agricultural soils
PECnat.soil	[mg.kg$_{wwt}^{-1}$]	6,30E-05	Continental PEC in natural soil (total)
PECind.soil	[mg.kg$_{wwt}^{-1}$]	6,30E-05	Continental PEC in industrial soil (total)
PECsediment	[mg.kg$_{wwt}^{-1}$]	1,93E-04	Continental PEC in sediment (total)
- Regional			
PECsurfacewater (total)	[mg.l^{-1}]	8,59E-03	Regional PEC in surface water (total)
PECsurfacewater (dissolved)	[mg.l^{-1}]	8,59E-03	Regional PEC in surface water (dissolved)
PECair	[mg.m^{-3}]	9,45E-05	Regional PEC in air (total)
PECagr.soil	[mg.kg$_{wwt}^{-1}$]	2,91E-04	Regional PEC in agricultural soil (total)
PECporewater agr.soil	[mg.l^{-1}]	1,50E-03	Regional PEC in pore water of agricultural soils
PECnat.soil	[mg.kg$_{wwt}^{-1}$]	5,25E-04	Regional PEC in natural soil (total)
PECind.soil	[mg.kg$_{wwt}^{-1}$]	5,25E-04	Regional PEC in industrial soil (total)
PECsediment	[mg.kg$_{wwt}^{-1}$]	5,73E-03	Regional PEC in sediment (total)

European Commission

EUR 22159 EN **European Union Risk Assessment Report**
Propan-1-ol, part I – environment, Volume 82

Editors: S. Pakalin, K. Aschberger, O. Cosgrove, A. Paya-Perez, S. Vegro.

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The report provides the comprehensive risk assessment of Environment part of the substance propan-1-ol. It has been prepared by Germany in the frame of Council Regulation (EEC) No. 793/93 on the evaluation and control of the risks of existing substances, following the principles for assessment of the risks to humans and the environment, laid down in Commission Regulation (EC) No. 1488/94.

The evaluation considers the emissions and the resulting exposure to the environment and the human populations in all life cycle steps. Following the exposure assessment, the environmental risk characterisation for each protection goal in the aquatic, terrestrial and atmospheric compartment has been determined. For human health the scenarios for occupational exposure, consumer exposure and humans exposed via the environment have been examined and the possible risks have been identified.

The environmental risk assessment for propan-1-ol concludes that there is at present no concern for atmosphere, aquatic ecosystem, terrestrial ecosystem and for micro-organisms in the sewage treatment plant. The human health risk assessment for propan-1-ol concludes that there is at present concern for workers and consumers. There is at present no concern for humans exposed via the environment.

The conclusions of this report will lead to risk reduction measures to be proposed by the Commissions committee on risk reduction strategies set up in support of Council Regulation (EEC) No. 793/93.

The mission of the JRC is to provide customer-driven scientific and technical support for the conception, development, implementation and monitoring of EU policies. As a service of the European Commission, the JRC functions as a reference centre of science and technology for the Union. Close to the policy-making process, it serves the common interest of the Member States, while being independent of special interests, private or national.

European Commission – Joint Research Centre
Institute for Health and Consumer Protection (IHCP)
Toxicology and Chemical Substances (TCS)
European Chemicals Bureau (ECB)

European Union Risk Assessment Report

propan-1-ol
Part I - environment

CAS No: 71-23-8 EINECS No: 200-746-9

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RISK ASSESSMENT

Propan-1-ol

CAS-No.: 71-23-8

EINECS-No.: 200-746-9

16.04.2008

FINAL APPROVED VERSION

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The first draft of the Comprehensive Risk Assessment Report of **Propan-1-ol**, a substance chosen from the EU 2nd priority list in 1995 was distributed for the preliminary written procedure in June 2002.

The “in depth discussion” was at the Technical Meeting in March 2003 (TM I’03).

This document is a revised draft of the environmental part of the Risk Assessment Report which is intented to be discussed as “final written approval” at the Technical Meeting in September 2003 (TM III´03).

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Foreword

We are pleased to present this Risk Assessment Report which is the result of in-depth work carried out by experts in one Member State, working in co-operation with their counterparts in the other Member States, the Commission Services, Industry and public interest groups.
The Risk Assessment was 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 wihin 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 Health and Environmental Risks (SCHER) which gives its opinion to the European Commission on the quality of the risk assessment.
If a Risk Assessment Report concludes that measures to reduce the risks of exposure to the substances are needed, beyond any measures which may already be in place, the next step in the process is for the "Rapporteur" to develop a proposal for a strategy to limit those risks.
The Risk Assessment Report is also presented to the Organisation for Economic Co-operation and Development as a contribution to the Chapter 19, Agenda 21 goals for evaluating chemicals, agreed at the United Nations Conference on Environment and Development, held in Rio de Janeiro in 1992 and confirmed in the Johannesburg Declaration on Sustainable Development at the World Summit on Sustainable Development, held in Johannesburg, South Africa in 2002.
This Risk Assessment improves our knowledge about the risks to human health and the environment from exposure to chemicals. We hope you will agree that the results of this in-depth study and intensive co-operation will make a worthwhile contribution to the Community objective of reducing the overall risks from exposure to chemicals.

[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]

CONTENTS

0 OVERALL CONCLUSIONS/RESULTS OF THE RISK ASSESSMENT

CAS No. 71-23-8

EINECS No. 200-746-9

IUPAC Name Propan-1-ol

Overall results of the risk assessment:

(X) i) There is need for further information and/or testing

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

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

Summary of conclusions:

Environment

From the intrinsic properties it is expected that propan-1-ol is of low concern for the environment. The environmental risk assessment was performed, using conservative estimates based on worst-case assumptions at the exposure and effects side. The risk assessment results in the following conclusion:

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

Based on the currently available data, propan-1-ol represents no risk to the environment for the area of production, processing, formulation and use.

Human Health

Workers

i) There is need for further information and/or testing

Further toxicological data (90-day rat inhalation study) is needed in order to perform a robust occupational risk assessment for repeated dose toxicity (systemic effects).

Propan-1-ol is also notified as an active substance within the scope of the Biocide Directive 98/8/EC. The necessary data on repeated dose toxicity is existing, but is owned by a company who wishes to use it in the framework of other EU regulation (the Biocides Directive). The company is so far not willing to make the study available to support risk assessment in the context of the Existing Substances Regulation, and there are no provisions in the Biocides Directive that would force them to share the data with other companies. The information on repeated dose toxicity is requested for reasons of human health. For the sake of animal protection it is hoped that the companies involved will be able to negotiate and share the data.

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

Occupational risk assessment reveals that for certain exposure scenarios there is a need for limiting the risks. The toxicological endpoints of concern especially are reprotoxicity (fertility impairment and developmental toxicity) and local effects by inhalation (acute sensory irritation and respiratory tract irritation by repeated exposure). Sensory irritation may occur following high short-term exposure levels. Results from the required 90-day rat inhalation study may have an impact on the risk assessment for fertility impairment and respiratory tract irritation by repeated exposure.

Slight effects to the skin following repeated dermal exposure cannot be excluded. No concern is expressed for eye irritation; it is assumed that corresponding classification of propan-1-ol will result in necessary risk reduction measures.

Concern especially concentrates on the use of paints (scenario 3) and on the use of cleaning formulations (scenario 4a).

Consumers

i) There is need for further information and/or testing

Further toxicological data is needed in order to perform a sound risk assessment for repeated dose toxicity (cf. conclusion i for workers).

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

The risk characterisation for the oral exposure scenario via mouth hygiene products and the aggregated exposure scenario of consumers via mouth hygiene products, cosmetics, disinfectants and general cleaning products leads to conclusion iii) because of concern for men with respect to fertility impairment.

The risk characterisation for the oral exposure scenario via mouth hygiene products and the aggregated exposure scenario of consumers via mouth hygiene products, cosmetics, disinfectants and general cleaning products leads to conclusion iii) because of concern with respect to developmental toxicity.

Sensory irritation may occur following short-term inhalation exposure during the application of propan-1-ol containing disinfectants, hardener solutions, and wall paper removers.

The provisional risk characterisation for repeated dose toxicity reveals that there may be a need for limiting the risks due to the oral exposure scenario via mouth hygiene products and the aggregated exposure scenario of consumers via mouth hygiene products, cosmetics, disinfectants and general cleaning products. The results of the required inhalation study may influence the outcome of the risk characterisation for consumers.

Man exposed indirectly via the environment

i) There is need for further information and/or testing

Further toxicological data is needed in order to perform a sound risk assessment for repeated dose toxicity (cf. conclusion i for workers).

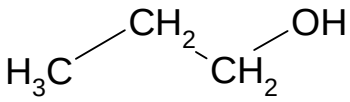
ii) There is at present no need for further information and/or testing and for risk reduction measures beyond those which are being applied allready.

Conclusion (ii) is reached for the provisional risk characterisation for repeated dose toxicity and for all other toxicological endpoints for the local and the regional scenario.

1 GENERAL SUBSTANCE INFORMATION

Identification of the substance

CAS No.:	71-23-8
EINECS No.:	200-746-9
IUPAC Name:	Propan-1-ol
Synomyma:	1-Hydroxypropane
	1-Propanol
	Ethylcarbinol
	n-Propanol
	n-Propyl alcohol
	Propanol-1
	Alcohol C_3
Empirical formula:	C3H8O
Structural formula:	



Molecular weight:	60.1 g/mol

Purity/impurities, additives

Purity:	> 99%
Impurities:	methanol
	ethanol
	C6 aldehydes
	propyl propionate
	2-methylvaleraldehyde
	≤ 0.2% w/w aldehyde

< 0.1 w/w dipropyl ether

≤ 0.1% w/w water

≤ 0.003% w/w acetic acid

Table 1.1: Physico-chemical properties

Physical state	clear colourless liquid with characteristic odour	
Melting point	-126.5 °C	CRC Handbook (1991/92)
Boiling point	97.1 °C at 1013 hPa	Hiaki et al. (1994)
Density	0.803 g/cm^3 at 20 °C	Wilhoit & Zwolinski (1973)
Vapour pressure	19.4 hPa at 20 °C	Hiaki et al. (1994)
Surface tension	67.1 mN/m at 25 °C c=1 g/l	CRC Handbook (1991/92)
Partition coefficient	0.34 (shake flask method)	Hansch & Anderson (1967)
Water solubility	completely soluble	Yaws et al. (1990)
Flash point	22 °C (corrected to the presence of iso-propanol) 23.5 °C (99.9% pure)	CHEMSAFE DIN 51755, ISO 3679
Auto flammability	385 °C	CHEMSAFE DIN 51794
Flammability	flammable	CHEMSAFE
Explosive properties	not explosive	due to structural reasons
Oxidizing properties	no oxidizing properties	due to structural reasons

Vapour pressure

The values given for the vapour pressure at 20 °C vary between 19 and 20.3 hPa. In the safety data sheet of the BASF AG a value of 19.4 °C is quoted, in the data sheet of the Hoechst AG the vapour pressure is quoted with 20 hPa. In both cases no other information is given. Also without any further information Sasol has quoted a value of 20 hPa at 20 °C. For the risk assessment the value of 19.4 hPa at 20°C is recommended. This value is derived from the Antoine equation determined by Boublik, T., Fried, V. & Hala, E. (1984) "The vapour pressures of pure substances" 2nd ed., Elsevier, Amsterdam.

Partition coefficient n-octanol/water

The values for the partition coefficient n-octanol/water are varying between 0.25 and 0.38. The safety data sheets of the BASF AG, Hoechst AG and Union Carbide are quoting values between 0.25 and 0.34 without further information. Furthermore the partition coefficients are calculated. The following values are found: 0.271 (according to Rekker with program PRO-LOGP, ver.2 from CompuDrug Ltd.), 0.38 (Abraham M.H., Chadha H.S., Whiting G.S., Mitchell R.C. (1994), J. Pharm. Sci. Vol. 83, No. 8, 1085-1100). Further undocumented values are quoted by Petrasol B.V. Gorinchem and BASF AG (1989): Labor fuer Umweltanalytik by 0.25 and 0.271, respectively. Other values from literature are in the above mentioned range.

For risk assessment the value of 0.34 of Hansch C. & Anderson S.M. is recommended. The authors have great experience in the field of measuring and calculating octanol/water partition coefficients. They used some kind of shake flask method (Hansch C. & Anderson S.M. (1967), J. Org. Chem. 32, 2583).

Flash point

The value of 23.5 °C was determined for n-propanol with a purity of 99.9%. The tests were conducted according to DIN 51755 (Testing of mineral oils and other combustible liquids; determination of flash point by the closed tester according to Abel-Pensky) and ISO 3679 (Paints, varnishes, petroleum and related products - determination of flashpoint – rapid equilibrium method). The value of 23.5 °C was corrected for commercial n-propanol to 22 °C because of iso-propanol which is usually present as an impurity.

Classification

- (Classification according to Annex I, 30th ATP.[4])

Highly flammable	R 11	Highly flammable
Irritant	R 41	Risk of serious damage to eyes
	R 67	Vapours may cause drowsiness and dizziness

- (Proposal of the rapporteur)

Flammable	R 10	Flammable
Irritant	R 41	Risk of serious damage to eyes
	R 66	Repeated exposure may cause skin dryness or cracking

A value of 23.5 °C was determined for the flash point of n-propanol with a purity of 99.9%. This value of 23.5 °C was corrected for commercial n-propanol which usually contains iso-propanol as an impurity to 22 °C.

The classification for liquid substances with a flash point between 21 and 55 °C is "flammable".

Therefore the legal classification according to Annex I for propan-1-ol which is at the moment "highly flammable" must be corrected. The original classification resulted from measurements of n-propanol contaminated with impurities (flash point < 21 °C).

Taking also into account the defatting solvent character of propan-1-ol it is assumed that frequent contact can lead to skin dryness or cracking of skin. Consequently, classification with R 66 (Repeated exposure may cause skin dryness or cracking) is proposed.

According to the data presented below and the criteria of Directive 93/21/EEC propan-1-ol has not to be classified as dangerous for the environment.

Propan-1-ol is classified according to water-hazard class 1 (slightly hazardous to water).

[4] The classification of the substance has been established by Commission Directive 2008/58/EC of 21 August 2008 adapting to technical progress for the 30th time Council Directive 67/548/EEC on the approximation of the laws, regulations and administrative provisions relating to the classification, packaging and labelling of dangerous substances (OJ L 246, 15.09.2008, p.13).

2 GENERAL INFORMATION ON EXPOSURE

2.1 PRODUCTION

According to the information from the currently available IUCLID data sets there is one production site of propan-1-ol in the EU. The chemical is imported by 5 other companies from outside of the EU. There is no information on possible exports of propan-1-ol.

Based on the production and import quantities approximately 30 100 t/a of propan-1-ol are used in the EU.

Propan-1-ol is produced almost exclusively by the reaction of ethene with synthesis gas. Reaction is performed at 25-30 MPa and 140-180 °C in the liquid phase in the presence of cobalt carbonyl hydrogen as catalyst. After the separation of the catalyst the raw mixture obtained can be hydrogenated in the gaseous phase on the nickel catalyst (0.2-0.3 MPa, 115 °C) or on the copper catalyst (3-5 MPa, 130-160 °C) and in the liquid phase (8 MPa, 115 °C) on the nickel catalyst. By means of subsequent distillation the production of pure propan-1-ol is achieved (Weissermel and Arpe, 1988; Falbe et al., 1980).

2.2 PROCESSING / APPLICATION (CATEGORIES OF USE, AMOUNTS)

In Western Europe propan-1-ol is mainly used as solvent for the formulation of disinfectants, pharmaceutical products, cleaning agents, paints, coating materials, enamel and lacquer paints, printing inks and cosmetics (GDCh, 1997).

Propan-1-ol is processed chemically to intermediates such as propylamines, carboxylic acid esters and halogenated hydrocarbons, which in turn are needed for the synthesis of herbicides, aroma and perfume substances, cosmetics and pharmaceuticals (GDCh, 1997).

Of the propan-1-ol produced by BASF AG, 10 % were processed to intermediates and 90 % were used as solvent; application areas as solvent were paints, surface coatings and inks, cosmetics and pharmaceuticals, detergents and other (BASF, 1994).

Hoechst AG manufactured 5 000 t of propan-1-ol in 1993; about 3 500 t were processed to n-propylamines, 1 500 t of propan-1-ol were sold. In 1995, the production of propan-1-ol was ceased; the requisite amounts of propan-1-ol for the production of the n-propylamine derivatives are supplied from Bay City, USA (Hoechst AG, 1994 and 1995b).

Based on the available information the following consumption’s of propan-1-ol are estimated for Western EU (CEH, 1995):

- 55 % were used as solvent, hereof:

  approx. 35 % to lacquer, paints, prints,

  approx. 20 % to cosmetics,

  approx. 35 % pharmaceutics (disinfectants),

  approx. 5 % cleaning/washing agents,

  approx. 5 % to other

- 45 % were processed as an intermediate for the production of:

  approx. 75 % to n-propylacetate,

  approx. 20 % to propylchlorformiate and

  approx. 5 % to reactive resins.

Because of the various direct applications of propan-1-ol in end products it has to be expected that the handled amount of propan-1-ol in Europe may increase through import. No further information is available on import or export, as well as on residual content of propan-1-ol in end products.

The use of propan-1-ol in cleaning agents, pesticides, thinners, paints, printing inks and solvents is described in SPIN – Substances in Preparations In the Nordic countries data base. The information contained in SPIN is listed in Table 2.2.1.

Table 2.2.1 Information on propan-1-ol in consumer products in the nordic countries obtained from SPIN data base (July 2003)

Country	**Year**	**Number of preparations**	**Quantity of propan-1-ol contained in preparations [t]**
FIN	2001	181	**3341**
N	2001	110	**2675**
DK	2001	208	**1925**
S	2000	202	**743**

The main use categories of the preparations containing propan-1-ol are cleaning/washing agents (N), reprographic agents and solvents (DK), activators and dyestuffs (FIN), solvents and de-icing agents (S).There are also non-industrial sources of propan-1-ol: It is contained in landfill gas; it is formed from plants and animals through putrefaction and decomposition; alcohol-forming bacteria are involved here. The substance is contained in aromas of fruits and other foodstuffs. It is a natural component of alcoholic beverages that have been obtained through fermentation of plant raw materials (GDCh, 1997). The quantity of formed propan-1-ol and the resultant environmental concentration in the different compartments cannot be quantified. However, it is assumed that these are very low and can be neglected.

The following table shows the main, industrial and use categories and the mass balance of propan-1-ol for the EU.

Table 2.2.2 Use categories and mass balance of propan-1-ol

Main category (MC)	**Industrial category (IC)**	**Use category (UC)**	**Mass balance [in % of use]**
Non-dispersive use (**3**)	Chemical industry (**3**)	Intermediate (**33**)	**45**
Non-dispersive use (**3**)	Other (**0**)	Solvent (**48**)	**3**
Wide dispersive use (**4**)	Personal/domestic (**5**)*	Solvent (**48**)	**33**
Wide dispersive use (**4**)	Paint, lacquers and varnishes industry (**14**)	Solvent (**48**)	**19**

* Sum of quantity used as solvent for cosmetics, pharmaceutics (disinfectants) and cleaning/washing agents.

3 ENVIRONMENT

4 HUMAN HEALTH

4.1 HUMAN HEALTH (TOXICITY)

4.1.1 Exposure assessment

4.1.1.1 General discussion

The main application of propan-1-ol (approx. 55 %) is its use as a solvent for the formulation of different products. The following use pattern is valid for the EU (CEH, 1995):

approx. 20 %	lacquer, paints, printing inks,
approx. 20 %	pharmaceuticals (disinfectants),
approx. 10 %	cosmetics (e.g. skin care, mouth hygiene),
approx. 2 %	cleaning / washing agents,
approx. 3 %	other.

45 % of the handled propan-1-ol is processed to intermediates such as n-propylacetate, propylchlorformiate and reactive resins, which in turn are needed for the synthesis of herbicides, aroma and perfume substances, cosmetics and pharmaceuticals (GDCh, 1997).

For workers the inhalation and dermal routes of exposure are likely to occur.

According to the Swedish product register, propan-1-ol is used in a variety of products which are listed as consumer products, especially in cosmetics, disinfectants, painting materials, hardeners and in cleaning products. The products reported by industry to the Federal Institute for Health Protection of Consumers and Veterinary Medicine (BgVV) also contain propan-1-ol. A summary is given in the table under 4.1.1.3.

4.1.1.2 Occupational exposure

Industrial activities involving propan-1-ol present opportunities for exposure. In the fields of production and further processing, inhalation and dermal exposure of workers to propan-1-ol may occur during sampling, filling and mixing processes as well as during cleaning, maintenance and repair work. Exposure ranges depend on the particular operation and the risk reduction measures in use.

Occupational exposure limits (OEL) and short term limits (STEL) are established in the EU and the USA (c.f. Table 4.1.1.2.1).

Table 4.1.1.2.1: Occupational exposure levels (ARIEL, 2000)

Country	OEL	STEL
Austria, Denmark, France, Netherlands, Swiss	500 mg/m^3 (200 ml/m^3)	-
Finland, Ireland	500 mg/m^3 (200 ml/m^3)	620 mg/m^3 (250 ml/m^3)

Country	OEL	STEL
Spain, United Kingdom, USA:OSHA	500 mg/m^3 (200 ml/m^3)	625 mg/m^3 (250 ml/m^3)
Belgium	499 mg/m^3 (200 ml/m^3)	623 mg/m^3 (250 ml/m^3)
USA:ACGIH	492 mg/m^3 (200 ml/m^3)	614 mg/m^3 (250 ml/m^3)
Norway	245 mg/m^3 (100 ml/m^3)	-
Germany	-	-

The following scenarios are regarded to be relevant for occupational exposure:

Scenario 1: Production of propan-1-ol and further processing as an intermediate (4.1.1.2.1)
Scenario 2: Preparation of formulations, e. g. paints, printing inks, disinfectants, cleaners (4.1.1.2.2)
Scenario 3: Use of paints (4.1.1.2.3)
Scenario 4: Use of cleaning formulations (4.1.1.2.4)
Scenario 5: Use of printing inks (4.1.1.2.5)
Scenario 6: Use of disinfectants (4.1.1.2.6)

Further applications of propan-1-ol are possible, e. g. as a solvent during core making, in hardeners and glues. The situation is often not clear, since in the literature the term “propanols” is used instead of referring to propan-1-ol or propan-2-ol. Since propan-2-ol is of major economic importance, the corresponding exposure situations are judged to be of minor relevance for propan-1-ol.

The assessment of inhalation exposure is mainly based on measured exposure levels from which – if possible – 90th percentile are derived as representing reasonable worst case situations. For the purpose of exposure assessment only data measured later than 1990, if available, are taken. Scenarios are clustered as far as possible to make the description transparent. If quantitative exposure data is not available, model estimates are taken.

Beside inhalation exposure, dermal exposure is assessed for each scenario. Two terms can be used to describe dermal exposure:
Potential dermal exposure is an estimate of the amount of a substance landing on the outside of work wear and on the exposed skin.
Actual dermal exposure is an estimate of the amount of a substance actually reaching the skin. There is an agreement between the EU-memberstates, within the framework of existing substance, to assess – as a rule – dermal exposure as exposure to hands and parts of the forearms. In this, the main difference between both terms – potential and actual – is the protection of hands and forearms by work wear and – more important – the protection by gloves. Within this exposure assessment, the exposure reducing effect achievable by gloves is only considered if information is provided, that for a certain scenario gloves are a widely accepted protection measure and that the gloves are fundamentally suitable for protection against the substance under consideration. As a measure for the latter, tests according to DIN En 374 are taken as a criteria. For most down stream uses it is commonly known, that gloves are not generally worn. In these cases, dermal exposure is assessed as actual dermal exposure for the unprotected worker. Since often quantitative information on dermal exposure is not available, the EASE model is used for assessing dermal exposure at the most.

4.1.1.2.1 Production of propan-1-ol and further processing as an intermediate in the large-scale chemical industry (scenario 1)

Scenario 1 is related to the production of propan-1-ol and its further processing as a chemical intermediate in the large-scale chemical industry.

Propan-1-ol is continuously produced in closed systems, almost exclusively by the reaction of ethene with synthesis gas (H_2, N_2, CO). The reaction is carried out at high pressure and 140-180 °C in the liquid phase in the presence of cobalt carbonyl hydrogen as a catalyst. After the separation of the catalyst the raw mixture is hydrogenated using nickel or copper as a catalyst. By means of subsequent distillation the production of pure propan-1-ol is achieved (Weissermel and Arpe, 1988; Falbe et al., 1980).

The processing to intermediates such as n-propylacetate, propylchlorformiate, reactive resins, and pharmaceuticals also occurs in closed systems. According to information of the producers the residual content of propan-1-ol in the intermediates is 0 - 0.2 %.

The transfer to user companies occurs by means of tank cars, rail tankers or tank containers. A minor amount is filled into drums. According to information provided by a manufacturer, in-company transfer occurs via closed pipelines using gas displacement devices (Hoechst AG, 1997). Another company uses a central filling station for the distribution of propan-1-ol to the reactors (BASF AG, 1999).

Exposure associated with transporting the chemical could result from loading, unloading and drumming operations. For the large-scale chemical industry high standards of control at the workplace are assumed to be practiced even if the containment is breached, e.g. during filling, cleaning, maintenance, repair works and taking of samples. Inhalation exposure in other fields is normally minimized by technical equipment (e.g. special designed filling stations, Local Exhaust Ventilation, LEV).

Daily exposure over the full shift is assumed for production and further processing of propan-1-ol in the large-scale chemical industry.

Inhalation Exposure

Workplace measurements

Table 4.1.1.2.2: Propan-1-ol exposure at workplaces during production and further processing (scenario 1) (provided by one producer, a second user / producer provided short-term values)

Job category / activities	Years of measurement	Number of samples	Technical measures	Measurement data [mg/m³]	50th Percentile [mg/m³]	90th Percentile [mg/m³]
8 h TWA						
In total	1994–1999	348 (p)	closed system	-	1.1	2.6
Production	1994–1999	16 (p)	closed system	-	0.1	0.7
Further processing	1994–1999	218 (p)	closed system	-	1.1	2.4

Job category / activities	Years of measurement	Number of samples	Technical measures	Measurement data [mg/m^3]	50th Percentile [mg/m^3]	90th Percentile [mg/m^3]
Pilot plant	1994–1999	32 (p)	LEV	-	1.1	4.1
Laboratory	1994–1999	58 (p)	LEV	-	1.1	2.75
Storage filling	1994–1999	17 (p)	LEV	-	0.75	3.5
Short-term values						
Further processing filling cleaning	 1994–1999 1994–1999	 1 (p) 1 (p)	 LEV LEV	 510 550	 - -	 - -

p: personal sampling
LEV: local exhaust ventilation
8 h TWA: 8 h time weighted average

For the purpose of measuring propan-1-ol concentration in workplace air the substance is adsorbed to silica gel and then desorbed using ethylenglycole / water and detected gas-chromatographically. The detection limit of the method amounts to 0.55 mg/m^3 (sampling volume: 10 l) (BASF AG, 1999). Due to the measurement method and the sampling strategy applied, the measurement results (Table 4.1.1.2.2) are regarded as valid. Since 2 of 3 producers and users submitted measurement results which cover different activities, the measurement results are assumed to be representative.

Based on the available measurement results, 4.1 mg/m^3 is regarded to represent a reasonable worst case situation for all activities during production and further processing in the chemical industry. In addition, the short-term value 550 mg/m^3 (sampling time < 1 h) obtained during cleaning is used for exposure assessment.

It is to be assumed that the substance is processed daily. Consequently, the duration and the frequency of exposure to propan-1-ol are assumed to be daily and for the entire length of shift.

Conclusions

Inhalation exposure has to be assessed for the production and the further processing of propan-1-ol as a chemical intermediate in the large-scale chemical industry (scenario 1).

For the assessment of health risks from daily inhalation exposure to propan-1-ol during the production and further processing an 8 h time weighed average concentration (8 h TWA) of 4.1 mg/m^3 should be taken. Short-term exposure up to 550 mg/m^3 (sampling time < 1 h) is possible during filling and cleaning.

Dermal exposure

When producing and further processing dermal exposure could occur during activities like drumming, sampling, cleaning, maintenance and repair work. For the unprotected worker, according to the EASE model, potential dermal exposure is assessed as follows:

Input parameters: Non dispersive use, direct handling, intermittent
Level of exposure: 0.1 – 1 mg/cm^2/day.

Considering an exposed area of 210 cm^2 (area corresponds to the surface area of the palms of hands) the model yields an exposure level of 21 - 210 mg/person/day. The extent of protection of the personal protective equipment (here gloves) depends inter alia on the suitability of the recommended glove material with regard to the permeation properties of the substance.

According to information provided by the manufactures (safety data sheets), in the case of propan-1-ol, suitable gloves tested according to EN 374 are worn. For assessing actual dermal exposure levels, it has to be considered that the substance is manufactured and further processed primarily in closed systems and that the use of PPE (here gloves and eye protection) during exposure relevant activities is highly accepted in the large-scale chemical industry. The exposure assessment is made based on the assumption, that a vast majority of workers belonging to scenario 1 are protected by suitable gloves. However, in spite of this, dermal exposure may occur due to e. g.

- unintended contamination during the handling of used gloves,
- limited protection of suitable gloves at real working conditions (e. g. mechanical stress).

As a rule, for the use of suitable gloves, low levels of daily dermal exposure are to be expected. Since no measurement results are available, a protection efficiency of 90 % is taken as a default value leading to an exposure level of 21 mg/person/day.

Conclusions

For assessing the health risks from daily dermal exposure in the area of production and further processing (scenario 1), an exposure level of 21 mg/person/day should be taken.

Exposure to the eyes is largely avoided by using eye protection.

4.1.1.2.2 Preparation of formulations, e. g. paints, printing inks, disinfectants, cleaners (scenario 2)

Propan-1-ol is used as an additive (solvent) in different formulations, the main products are paints, printing inks, disinfectants and cleaners. The cleaners are, in part, applied for degreasing purposes.

According to the available information concentrations of propan-1-ol in the mainly applied products are as follows:

Paints, lacquers:	approx. < 25 %
Printing inks:	approx. < 40 %
Disinfectants:	approx. < 60 %
Cleaners (incl. de-icing formulations):	approx. < 90 %.

The listed applications of propan-1-ol cannot be regarded as complete. Further uses of the

substance in formulations are possible, e. g. as a solvent during core making, in hardeners and glues. However detailed information is not available. Therefore, these uses cannot be implemented in this exposure assessment.

It is expected that the formulation processes of paints, printing inks and cleaners have often several steps in common, e.g. filling of starting materials, mixing, adjusting, and filling the products into drums or other containers. Since detailed information on the production of formulations containing propan-1-ol is not available, the formulation of paints is described exemplary (Stoye et al., 1998).

An all-embracing description of paint production processes cannot be given, mainly because paint manufacturers range from small companies producing only a few hundred tones of paint up to large companies producing several hundred thousand tonnes annually. A further difficulty is the multiplicity of formulations which results in widely varying batch sizes. Only a few product lines in the paint industry are highly automated and continuos, e.g. white emulsion paints.

Generally paint-making processes can be subdivided into five main steps:

1) The millbase (premixing) is prepared by transferring the liquid or powdery starting material into premixers. The charging and metering steps can be performed manually by dumping sacks or cans or automated via filling stations and fixed pipelines.

2) The mixture is dispersed in closed or open devices (agitator mills) and is then fed as a single-pigment paste in paste mixers (Step 2 is not necessary if ready-prepared pigment mixtures are used).

3) The formulation of the paste mixture or dispersion is completed in differently sized end product mixers.

4) The final product is adjusted by adding film-former solutions or other additives manually or via pipelines.

5) The end product is sieved, filtered and dispensed from the mixers via filling lines into containers, drums or small packages.

The above given description shows that a wide variety of paint making processes are practised. Accordingly different levels of protection are realised: closed and open systems, manual or automated charging. In addition, the general use of PPE (here: suitable gloves and eye protection) cannot be presupposed for the paint industry at all, despite the fact that there are single companies with a reasonable high level of protection.

In the view of occupational exposure it is to be assumed, that the production of other formulations (disinfectants, cleaners) is similar to the production of paints. Exposure relevant activities are filling, charging, cleaning, sampling, repair, maintenance activities as well as possibly mixing.

Duration and frequency of exposure are assumed to be full shift and daily, although transfer at the beginning of the process and drumming may be done only during part of the day, especially if production is discontinuously. In this case, duration of inhalation and dermal exposure may be shorter than the shift length.

Inhalation exposure

Workplace measurements

Measurements were performed by the German Worker's Compensation Funds (BGAA 1997, see table 4.1.1.2.3), France (INRS, 2000) and by the Monitoring Authorities (MA) of the Federal States of Germany. Due to the measurement method applied and the measurement strategy, the measurement results from the German Worker's Compensation Funds are regarded as valid. The data from Monitoring Authorities of the Federal States of Germany are taken as an additional description of occupational exposure.

Table 4.1.1.2.3: Exposure to propan-1-ol at workplaces during the production of formulations (scenario 2)

Job category / activities	Years of measurement	Number of samples	Technical measures	Measurement data [mg/m^3]	50th Percentile [mg/m^3]	90th Percentile [mg/m^3]
8 h TWA						
Production of paints mixing, filling, weighing (BGAA)	1991-1995	41 (12[(1)])	no LEV	-	2.8	15.5
	1991-1995	25 (11[(1)])	LEV	-	2.0	14.8
Production of paints, works at mixing tanks (INRS)	1987 - 2000	43	-	0.5 - 132	1.5[(2)]	7
Printing plate (MA)	1992-1995	6 (s, p)	-	0.6 - 11	-	-

[(1)] number of premises [(2)] median; p: personal sampling; s: stationary sampling ; LEV: local exhaust ventilation; 8 h TWA: 8 h time weighted average

Short-term values at the production of paints are located at 10 mg/m^3 (BGAA, 1997). Based on the data provided by INRS (0.5 – 132 mg/m³) it can be concluded, that higher short-term exposure levels are possible. As a measure for short term exposure, the highest value of 132 mg/m^3 is taken.

For the production of paints, similar 8 h TWA were measured at workplaces with and without LEV (see table 4.1.1.2.3). For a better understanding, it should be kept in mind, that occupational exposure levels at similar workplaces (here exposure to propan-1-ol during paint production) depend on the level of technical protection (here: LEV) and on the amount of the substance in use. Often, if the handling of large amounts of a substance is required, workplaces are equipped with LEV, whereas workplaces at which small amounts are handled are possibly not equipped with LEV. This circumstance might lead not only to similar exposure levels at workplaces with and without LEV but also to the situation, that exposure is higher at workplaces with LEV than at those without LEV.

At workplaces without local exhaust ventilation the 90th percentile amounts to 15.5 mg/m^3. This should be considered to represent the reasonable worst case situation for daily inhalation exposure during the preparation of formulations containing propan-1-ol. In addition a short-term value of 132 mg/m³ should be considered.

EASE estimation (EASE for Windows Version 2.0, Aug. 1997)

Exposure by inhalation to vapour during the preparation of formulations (e.g. paints, printing inks, disinfectants and cleaners) with local exhaust ventilation (vapour pressure: 1940 Pa):

Input parameters: T = 20 °C, non dispersive use, LEV present
Level of exposure: 25 – 50 mg/m^3 (10 - 20 ml/m^3)

Exposure by inhalation to vapour during the preparation of formulations (e.g. paints, printing inks, disinfectants and cleaners) without local exhaust ventilation (vapour pressure: 1940 Pa):

Input parameters: T = 20 °C, non dispersive use, dilution ventilation present
Level of exposure: 250 - 350 mg/m^3 (100 - 140 ml/m^3)

Representative information on the duration and frequency of exposure is not available. Information provided by the Federal Authorities in Germany indicate, that the daily duration is shorter than shift length (< 2.5 h/day) on a daily basis.

Considering a daily duration of 2.5 hours leads to an 8 h TWA of 8 - 16 mg/m^3 (3.1 – 6.2 ml/m^3) for workplaces with LEV and 78 - 109 mg/m^3 (31 - 44 ml/m^3) for workplaces without LEV.

Conclusions

The preparation of formulations, e.g. paints, printing inks, disinfectants and cleaners are clustered because of the similarity of the exposure scenarios.

A comparison of EASE estimates and measurement results reveals that the EASE model overestimates exposure for workplaces without LEV (with dilution ventilation). For workplaces with LEV, model estimates and workplace measurements are in the same order of magnitude.

For the assessment of the health risks from daily inhalation exposure to propan-1-ol during the production of formulations a 8 h TWA of 15.5 mg/m^3 (90th percentile of a measurement collective) should be taken.

Dermal exposure

For the field of preparation of formulations, e.g. paints, lacquers, disinfectants and cleaners, it is to be assumed, that PPE (here gloves and eye protection) is not regularly worn. The corresponding dermal exposure is assessed for the unprotected worker in application of the EASE model.

Input parameters: T = 20 °C, non dispersive use, direct handling, intermittent
Level of exposure: 0.1 - 1 mg/cm^2/day

Considering an exposed area of 420 cm^2 (area corresponds to the surface area of the palms of two hands), this dermal exposure amounts to 42 - 420 mg/person/day for daily dermal exposure during handling (e.g. filling) of the pure substance and of mixtures.

Conclusions

For assessing the health risks of daily dermal exposure in the area of production of formulations (scenario 2), an exposure level of 42 – 420 mg/person/day should be taken. The higher level is regarded to represent the reasonable worst case situation. This exposure assessment is based on the assumption that suitable gloves are not worn.

It cannot be presupposed that eye protection is regularly used. For assessing the risks, hand eye contacts as well as possible splashes to the eye should be considered.

4.1.1.2.3 Use of paints (scenario 3)

Lacquers and paints are applied by brushing, rolling, spraying, dipping or covering by pouring in different industrial and skilled trade sectors, e.g. treatment and processing of metal and wood, mechanical engineering, electronic industry, vehicle production and repair as well as building trade.

According to the available information, propan-1-ol is used in a percentage of < 25 % in different lacquer systems, e.g. synthetic enamels, in alcohol diluted resins and building paints. In addition propan-1-ol is used as a thinner with probably higher concentrations. In a comprehensive review on paints and lacquers the use of propan-1-ol in lacquers is judged to be of minor economic relevance compared to other solvents like acetone, propan-2-ol, xylole, toluole etc. (Baumann, Muth, 1997).

Spraying can be performed manually or automatically (spray cabins). Regarding the measurement results presented below (table 4.1.1.2.4), no detailed information on the processes is available. In addition to inhalation exposure caused by the evaporation of the substance, droplets aerosols may be a source of exposure.

In the view of occupational exposure, the application of paints by rolling and brushing etc. is relevant as well as the preparation of paints and cleaning after finishing painting. During painting the use of PPE (here respiratory protection, gloves and eye protection) is not regarded to be a general measure to reduce exposure.

Inhalation exposure

Workplace measurements

Table 4.1.1.2.4: Exposure to propan-1-ol at workplaces during use of paints (scenario 3)

Job category / activities	Years of measurement	Number of samples	Technical measures	Measurement data [mg/m^3]	50th Percentile [mg/m^3]	90th Percentile [mg/m^3]
8 h TWA						
Rolling, brushing in the building trade (BGAA)	1991-1995	33 (14 [(1)])	no LEV	-	3.0	494
Spray painting: metal industry, building trade (BGAA)	1991-1995	80 (11 [(1)])	no LEV	-	6.5	33.0
		92 (57 [(1)])	LEV	-	2.0	15.6
Spray painting, paint mixing (auto-mobile ind.) (MA)	1991	3	LEV	1 – 4	-	-
	1991	1	-	6	-	-

[(1)] number of premises; LEV: local exhaust ventilation; 8 h TWA: 8 h time weighted average

Due to the measurement method and the measurement strategy applied, the measurement results from the German Worker's Compensation Funds (BGAA, 1997) are regarded as valid. The data from the Monitoring Authorities (MA) of the Federal States of Germany are taken as an additional description of occupational exposure.

Additional statements to the measured results (BGAA, 1997):

Rolling, brushing in the building trade: the data come predominatly from the construction industry. Levels in the region of 90th percentile were measured for large scale brush and roller application.

Short-term values were measured in the construction sector for the roller application of surface coatings and are located at 2204 mg/m^3 (BGAA, 1997).

The German Worker's Compensation Funds for construction industries (Bau BG) has simulated workplaces of painters in order to get information on the exposure during coating work. Measurements performed during brushing and rolling of paints in a non-ventilated room reveal exposure levels of propan-1-ol of 82 – 2541 mg/m^3 (n = 4, sampling time 78 - 120 min). For the purpose of spray painting diluted paints were used. During spray painting in a ventilated room, exposure levels of propan-1-ol were located between 3 and 134 mg/m^3 (n = 66, sampling time 1 h). The 90th percentile amounts to 87 mg/m^3 (Gerner et al., 1997).

Additional data from 1990 – 1994 provided by Finnish Institute of Occupational Health are located between 5 and 320 mg/m^3.

On the basis of the presented exposure data, the 494 mg/m^3 obtained at real workplaces is regarded to represent the reasonable worst case situation of daily exposure during painting. The exposure situation "spray painting" is not treated separately from other painting techniques, because the measurement results obtained during spray painting (90th percentile, n

= 172, 68 premises) are considerably lower than other measurement results (s. table 4.1.1.2.4).

However, the data reveal, that the 50^{th} percentile (3.0 mg/m^3) for rolling and brushing is two orders of magnitude below the 90^{th} percentile. The 50^{th} percentile is regarded to represent the typical exposure situation for the given scenario.

It is assumed, that activities relevant for exposure performed daily during the entire length of the shift.

EASE estimation (EASE for Windows Version 2.0, 1997)

Rolling, brushing

Exposure by inhalation to vapour during the use of paints without local exhaust ventilation (vapour pressure 1940 Pa):

Input parameters: T = 20 °C, wide dispersive use, direct handling with dilution ventilation
Exposure level: 500 – 750 mg/m^3 (200 - 300 ml/m^3)

The EASE estimation is performed for the pure substance. The EASE estimates cannot be corrected for the partial vapour pressure, because the compositions of the formulations are not known in detail.

Conclusions

For the assessment of health risks from daily inhalation exposure to propan-1-ol during the use of paints (large scale roller, brusher application) an 8 h TWA of 494 mg/m^3 should be taken. The EASE estimate of 500 – 750 mg/m^3 (200 - 300 ml/m^3) is in good agreement with that exposure level. It should be considered that the typical value is expected to be considerably lower. The typical value is expected to be in the range of 3 mg/m^3. Higher short-term exposure levels occur (2204 mg/m^3, sampling time < 1 h).

Dermal exposure

Taking into consideration that personal protective equipment is not generally worn during painting works, the estimation of dermal exposure levels is performed for the unprotected worker.

Input parameters: T = 20 °C, wide dispersive use, direct handling, intermittent
Exposure level: 1 – 5 mg/cm^2/day

The estimation is performed for a formulation containing up to 25 % propan-1-ol and an affected skin area of 1300 cm^2 (area corresponds to the surface area of the hands and part of the forearms). The estimated exposure levels amount to 325 - 1625 mg/person/day.

Conclusions

For assessing the risk of daily dermal exposure during painting works (scenario 3), an exposure level of 325 - 1625 mg/person/day should be taken. The higher level is regarded to represent the reasonable worst case situation.

It cannot be presupposed that eye protection is regularly used. For assessing the risks, hand eye contacts as well as possible splashes to the eye should be considered.

4.1.1.2.4 Use of cleaning formulations (scenario 4)

Propan-1-ol is used in cleaning products (e.g. degreasing products) usually in concentrations of 30 %. According to available information propan-1-ol is also applied as an de-icing agent (< 90 % propan-1-ol). The only known application is de-icing of locks. Further information is not available.

Usually cleaners are applied manually using cloths e.g. for the purpose of degreasing metal parts or machines (e.g. printing machines). On the other side, the use of cleaners in degreasing baths is also possible.

Manual application of products containing propan-1-ol is a type of wide dispersive use, sometimes without the presence of any personal or technical control measure.

Inhalation exposure

Workplace measurements

Table 4.1.1.2.5: Exposure to propan-1-ol at workplaces during use of cleaners (scenario 4)

Job category / activities	Years of measurement	Number of samples	Technical measures	Measurement data [mg/m³]	50th Percentile [mg/m³]	90th Percentile [mg/m³]
8 h TWA						
Cleaning (degreasing devices, manual cleaning) (BGAA)	1991 - 1995	12 (8 (1))	no LEV	-	89	446
	1991 - 1995	22 (10 (1))	LEV	-	4.8	72
Short-term values						
Cleaning of printing machines (MA)	1991	1	-	4	-	-

(1) number of premises; LEV: local exhaust ventilation; 8 h TWA: 8 h time weighted average

Due to the measurement method and the measurement strategy applied, the measurement results from the German Worker's Compensation Funds (BGAA, 1997) are regarded as valid. The data from Monitoring Authorities (MA) of the Federal States of Germany are taken as an additional description of occupational exposure. The concentration of propan-1-ol in the applied formulations is not known.

The Finnish Institute of Occupational Health provided measurement values obtained during the cleaning of printing machines. The values (n = 6) range from 7.25 to 70 mg/m³.

Information on the frequency and duration of cleaning activities is not available. Full shift cleaning is regarded to be rather exceptional. On the other side, it is probable that exposure relevant activities are performed daily.

Short-term values are not available. Since for propan-1-ol, short-term exposures higher than 900 mg/m³ may cause respiratory depression (c.f. section 4.1.3.2.2, section Irritation/Corrosivity: inhalation) it has to be estimated whether short-term exposures higher than 900 mg/m³ are likely to occur. Depending on the activity under consideration, short-term

exposure may be considerably higher than shift averages, which amount to 446 mg/m³ for cleaning activities without LEV. For cleaning activities it is regarded to be reasonable, that short-term value might be 2 times higher that the shift averages. Taking this level into account, for the purpose of estimating a short-term value, it is concluded that short-term exposure levels higher than 900 mg/m³ are possible.

EASE estimation (EASE for Windows Version 2.0, 1997)

Exposure by inhalation to vapour during the use of cleaners with local exhaust ventilation (vapour pressure: 1940 Pa):

Input parameters: T = 20 °C, wide dispersive use, direct handling with LEV
Exposure level: 125 – 175 mg/m^3 (50 - 70 ml/m^3)

Exposure by inhalation to vapour during the use of cleaners without local exhaust ventilation (vapour pressure: 1940 Pa):

Input parameters: T = 20 °C, wide dispersive use, direct handling with dilution ventilation
Exposure level: 500 – 750 mg/m^3 (200 - 300 ml/m^3)

The EASE estimation is performed for the pure substance. The EASE estimates cannot be corrected for the partial vapour pressure, because the compositions of the formulations are not known in detail.

The consideration of a daily duration exposure (duration not known) would lead to shift averages being lower than the EASE estimates.

Conclusion

The EASE estimates and the workplace measurement results are in agreement for cleaning at workplaces. For the purpose of assessing inhalation exposure levels, the measurement results are taken.

For the assessment of health risks from daily inhalation exposure to propan-1-ol an 8 h TWA of 446 mg/m^3 should be taken for workplaces without LEV and 72 mg/m^3 for workplaces with LEV. In addition, for workplaces without LEV, short-term values higher than 900 mg/m³ should be considered.

Dermal exposure

Taking into consideration that personal protective equipment is not generally worn, the estimation of dermal exposure levels is performed for the unprotected worker.

Input parameters: T = 20 °C, wide dispersive use, direct handling, intermittent
Exposure level: 1 – 5 mg/cm^2/day

The estimation is performed for formulations containing up to 30 % propan-1-ol and an affected skin area of 840 cm^2 (area corresponds to the surface area of the hands). The estimated exposure levels amount to 252 – 1260 mg/person/day.

Conclusion

For assessing the health risks of daily dermal exposure during cleaning activities (scenario 4), an exposure level of 252 – 1260 mg/person/day should be taken. The higher level is regarded to represent the reasonable worst case situation.

It cannot be presupposed that eye protection is regularly used. For assessing the risks, hand eye contacts as well as possible splashes to the eye should be considered.

4.1.1.2.5 Use of printing inks (scenario 5)

Propan-1-ol is a constituent of printing inks which are use in silk screen printing, flexo printing and rotogravure printing (Baumann, Herberg-Liedtke, 1991). It can be contained in quantities of 0.2 - 42.5 % by weight .

In the printing industry, the printing process is performed mainly automatically, whereas the mixing and refilling of printing inks can be done manually. In addition, exposure is possible during cleaning, maintenance and repair.

Inhalation and dermal exposures are expected in particular in the case of activities in which printing inks are handled (e.g. mixing, weighing of inks, application of inks)

Inhalation exposure

Workplace measurements

Table 4.1.1.2.6: Exposure to propan-1-ol at workplaces during printing (scenario 5) (BGAA, 1997; INRS, 2000)

Job category / activities	Years of measurement	Number of samples	Technical measures	Measurement data [mg/m³]	50th Percentile [mg/m³]	90th Percentile [mg/m³]
8 h TWA						
Printing industry:						
silk screen printing	1991 – 1995	57 (27[(1)])	no LEV	-	5	18
(BGAA)	1991 – 1995	29 (15[(1)])	LEV	-	3	82
Printing operations (INRS)	1987 - 2000	251	-	0.1 - 1439	29[(2)]	109

[(1)] number of premises, [(2)] median; LEV: local exhaust ventilation; 8 h TWA: 8 h time weighted average

For the printing industry at workplaces with LEV higher measurement results were measured compared to workplaces without LEV (see table 4.1.1.2.6). For a better understanding, it should be kept in mind, that occupational exposure levels at similar workplaces (here exposure to propan-1-ol during printing) depend on the level of technical protection (here: LEV) and on the amount of the substance in use. Often, if the handling of large amounts of a substance is required, workplaces are equipped with LEV, whereas workplaces at which small amounts are handled are possibly not equipped with LEV. This circumstance might lead to the situation, that exposure is higher at workplaces with LEV than at those without LEV.

Additional measurement results are given in a report on exposure in the paper industry (Ahrens, Jöckel, 1996). In the field of finishing and packaging 14 measurements of propan-1-ol were performed in 2 companies (1974-1993). The 90th percentile is located at 22 mg/m^3 and the maximum amounts to 76 mg/m^3. It is not clear, during which activities (cutting to sizing, printing, folding or packaging) the measurement results were obtained and no information is available whether the workplaces were equipped with LEV or not. However, measurement values of propan-1-ol from 1990 – 1994 provided by the Finnish Institute of Occupational Health range from 1 - 93 mg/m^3 (n = 32) confirm the exposure levels of BGAA (1997), INRS (2000) and Ahrens, Jöckel (1996).

Taking all exposure information into account, 109 mg/m^3 is regarded to represent the reasonable worst case situation. There are no information available about the number of workers involved in the printing processes. It is assumed, that activities relevant for exposure are performed daily during the whole shift.

Short-term exposure levels are not available. Since for propan-1-ol, short-term exposures higher than 900 mg/m³ may cause respiratory depression (c.f. section 4.1.3.2.2, section Irritation/ Corrosivity, inhalation) it has to be estimated whether short-term exposures higher than 900 mg/m³ are likely to occur. Based on the data provided by INRS which range from 0.5 – 1439 mg/m³ it is concluded, that short-term exposure levels higher than 900 mg/m³ may occur.

EASE estimation (EASE for Windows Version 2.0, Aug. 1997)

Exposure by inhalation to vapour during printing with local exhaust ventilation (vapour pressure: 1940 Pa):

Input parameters:	T = 20 °C, non dispersive use, LEV present
Level of exposure:	25 – 50 mg/m^3 (10 - 20 ml/m^3)

Exposure by inhalation to vapour during printing without local exhaust ventilation (vapour pressure: 1940 Pa):

Input parameters:	T = 20 °C, non dispersive use, dilution ventilation present
Level of exposure:	125 - 175 mg/m^3 (50 - 70 ml/m^3)

The EASE estimation is performed for the pure substance. The EASE estimates cannot be corrected for the partial vapour pressure, because the compositions of the formulations are not known in detail.

Conclusions

The EASE estimates do not correspond to the measurement results and measurement values described below. Since measurement data from different sources reveal similar exposure levels, the exposure estimation is based on measurement results.

For the assessment of health risks from daily inhalation exposure to propan-1-ol during printing an 8 h TWA of 109 mg/m^3 (90th percentile of a measurement collective) should be taken. Short-term exposure levels higher than 900 mg/m³ are likely to occur.

Dermal exposure

Taking into consideration that personal protective equipment is not generally worn, the estimation of dermal exposure is performed for the unprotected worker.

Input parameters:	T = 20 °C, non dispersive use, direct handling, intermittent
Exposure level:	0.1 - 1 mg/cm^2/day

The estimation is performed for a formulation containing up to 42.5 % propan-1-ol and an affected skin area of 840 cm^2 (area corresponds to the surface area of the hands). The estimated exposure levels amount to 36 - 357 mg/person/day.

Conclusions

For assessing the risks of daily dermal exposure in the printing area (scenario 5), an exposure level of 36 - 357 mg/person/day should be taken. The higher level is regarded to represent the reasonable worst case situation.

It cannot be presupposed that eye protection is regularly used. For assessing the risks, hand eye contacts as well as possible splashes to the eye should be considered.

4.1.1.2.6 Use of disinfectants containing propan-1-ol (scenario 6)

Propan-1-ol is used for disinfecting procedures in hospitals and other medical establishments. The most important applications are disinfecting patient's skin and hygienic and surgical hand disinfecting of medical personnel. This is performed prior to all types of operations. Another application is for surfaces and instruments which must be disinfected rapidly, e.g. examination tables, working surfaces and rooms. Propan-1-ol and propan-2-ol are reaching their maximum potency in a approx. 60 % solution. (Harke, 1998).

The known propan-1-ol concentration in such preparations range from 1 - 70 %. If sprays are used, the concentration of propan-1-ol is approx. 10 %. For surface disinfection e.g. floors or operation rooms rather high amounts of solutions containing less propan-1-ol are used. This is concluded from a German workplaces survey on disinfections works where it is tated, that for room disinfection solutions with less than 5 % alcohols (BG/BIA-Empfehlung) are used.

Activities like disinfecting patient`s skin and hospital personnel hands are performed on short terms. Based on a general knowledge on the disinfecting activities in hospitals, it is to be assumed that the daily duration of exposure is considerably lower than shift length.

Inhalation exposure

Workplace measurements

Within the framework of a research project of the Monitoring Authorities of the Federal States of Germany two short-term values were obtained during skin disinfection (4 and 61 mg/m^3, stationary sampling, sampling time = 0.5 h).

EASE estimation (EASE for Windows Version 2.0, 1997)

Exposure by inhalation to vapour during the use of disinfectant without local exhaust ventilation (vapour pressure: 1940 Pa):

Input parameters: T = 20 °C, non dispersive use, direct handling with dilution ventilation
Exposure level: 250 – 350 mg/m^3 (100 - 140 ml/m^3)

Taking into account a daily duration of 1 h/shift, exposure levels reduce to 8 h TWA of 31 – 44 mg/m^3 (12.5 – 17.5 ml/m^3).

The EASE estimation is performed for the pure substance. The EASE estimates cannot be corrected for the partial vapour pressure, because the compositions of the formulations are not known in detail.

Conclusions

For the assessment of health risks from daily inhalation exposure to propan-1-ol during use of disinfectants an 8 h TWA of 44 mg/m^3 (EASE estimation) should be taken. Short-term exposure is typical for nursing activities. In addition, a short term value of 61 mg/m^3 should be considered.

Dermal exposure

Hand and surface disinfection is regarded to represent the exposure situation with highest dermal exposures, because no PPE is used.

Hand disinfection:

Input parameters: T = 20 °C, non dispersive use, direct handling, intermittent
Exposure level: 0.1 - 1 mg/cm^2/day

The estimation is performed for formulation containing up to 70 % propan-1-ol and an affected skin area of 840 cm^2 (area corresponds to the surface area of the hands). The estimated exposure levels amount to 59 - 588 mg/person/day.

Surface disinfection:

Input parameters:	T = 20 °C, wide dispersive use, direct handling, extensive
Exposure level:	5 - 15 mg/cm^2/day

The estimation is performed for a formulation containing up to 5 % propan-1-ol and an affected skin area of 840 cm^2 (area corresponds to the surface area of the hands). The estimated exposure levels amount to 210 - 630 mg/person/day.For room disinfection, 2 measurement results on exposure of bare hands are published in the Technical Notes for Guidance for Human Exposure to Biocidal Products. They were obtained during dilution and mixing of disinfectant and cleaning surfaces with a wrung cloth or mop and wringer bucket (Schnipper et al. 1996): 1.7 and 70.2 mg/min. The duration was 22 minutes for an operation room and 79 minutes for an isolation room. Taking the higher level and considering a duration of 79 minutes lead to an exposure level of 5545.8 mg substance on the hands. Taking into account the concentration of propan-1-ol (5 %) an exposure level of 277 mg/person/day is obtained.

For assessing the risks, the highest level of 630 mg/person/day is taken.

Conclusions

For assessing the health risks of daily dermal exposure during use of disinfectants (scenario 6), an exposure level of 210 – 630 mg/person/day should be taken. The higher level is regarded to represent the reasonable worst case situation. The use of protective equipment (PPE) is excluded for hand disinfection but might be taken into account for surface disinfection. Since no information on the suitability of the gloves is feasible, this effect cannot be taken into account quantitatively.

It cannot be presupposed that eye protection is regularly used. For assessing the risks, hand eye contacts as well as possible splashes to the eye should be considered.

4.1.1.2.7 Summary

55 % of propan-1-ol are applied as a solvent in formulations like paints, lacquers, pharmaceuticals (disinfectants), cosmetics and cleaning agents. The remaining 45 % is used as a chemical intermediate.

For occupational exposure there are 6 main scenarios:

- Scenario 1: production and further processing of propan-1-ol
- Scenario 2: preparation of formulations
- Scenario 3: use of paints
- Scenario 4: use of cleaning agents
- Scenario 5: use of printing inks

- Scenario 6: use of disinfectants

Relevant inhalation and dermal exposure levels are given in table 4.1.1.2.7 A and 4.1.1.2.7 B, respectively. The main sources of exposure are cleaning and painting works (scenario 3 and 4).

Additional there is incomplete information on other uses of the substance, e. g. as a solvent during core making, in hardeners and glues. The situation is often not clear, since in the literature the term “propanols” is used instead of referring to propan-1-ol or propan-2-ol. Since propan-2-ol is of major economic importance compared to propan-1-ol, the corresponding exposure situations are judged to be of minor relevance for propan-1-ol.

For the large scale chemical industry, it is assumed that the production and further processing of propan-1-ol is mainly performed in closed systems. Exposure occurs if the systems are breached for certain activities, e.g. filling (scenario 1, table 4.1.1.2.7 A). As concerning dermal exposure producers provided information that suitable gloves (tested according to EN 374) are used regularly during production and further processing. For all other scenarios (2 - 6), dermal exposure is assessed for the unprotected worker.

Table 4.1.1.2.7 A: Summary of inhalation exposure data (reasonable worst case) of propan-1-ol which are relevant for occupational risk assessment

Inhalation exposure								
Scenario number, Area of production and use	**Form of exposure**	**Activity**	**Duration [h/day]**	**Frequency [days/year]**	**Shift average concentration [mg/m^3]**	**Method**	**Short-term concentration [mg/m^3]**	**Method**
Production an use as a chemical intermediate								
1. Production and further processing as an intermediate	vapour (liquid)	charging, drumming, cleaning, repair, maintenance	shift length (assumed)	daily	4.1	90th percentile	550	workplace measurements (duration: < 1 h)
Formulation								
2. Formulation of products, e.g. paints, printing inks, disinfectants, cleaners	vapour (liquid)	charging, drumming, cleaning, repair, sampling	shift length (assumed)	daily	15.5	90th percentile	132	expert judgement
Use of formulations								
3. Use of paints (< 25 % propan-1-ol) (1)	vapour (liquid)	brushing, rolling	shift length (assumed)	daily	494	90th percentile	2204	measurements (duration: < 1 h)
4. Use of cleaning formulations (< 30 % propan-1-ol)	vapour (liquid)	application	not shift length (assumed)	daily	446 (4a: without LEV) 72 (4b: with LEV)	90th percentile 90th percentile	> 900 (2) -	expert judgement -
5. Use of printing inks (< 42.5 % propan-1-ol)	vapour (liquid)	mixing, weighing, application	shift length (assumed)	daily	109	90th percentile	> 900 (3)	expert judgement
6. Use of disinfectants (< 60 % propan-1-ol)	vapour (liquid)	disinfecting	1 h / day (assumed)	daily	44	EASE estimation	61	measurements (duration: 0.5 h)

(1) The exposure level for the typical case is considerably lower (3 mg/m^3).
(2) assumption: short term values higher than 2 x shift average are possible.
(3) expert judgement based on the range of the available measurement results

Table 4.1.1.2.7 B: Summary of dermal exposure data (reasonable worst case) of propan-1-ol which are relevant for occupational risk assessment

Dermal exposure								
Scenario number, Area of production and use	**Form of exposure**	**Activity**	**Frequency [days/year]**	**Contact level [1)]**	**Level of exposure [mg/cm^2/day]**	**Exposed area [cm^2]**	**Shift average [mg/person/day]**	**Method (use of gloves)**
Production and further processing								
1. Production and further processing as an intermediate	liquid	drumming, charging, sampling cleaning, repair, maintenance [2)]	daily	intermittent	0.1 – 1	210	21	EASE (suitable gloves)
Further processing to formulations								
2. Formulation of products, e.g. paints, printing inks, disinfectants	liquid	charging, drumming, cleaning, repair, sampling	daily	intermittent	0.1 - 1	420	420	EASE (without gloves)
Use of formulations								
3. Use of paints (< 25 % propan-1-ol)	liquid	brushing, rolling	daily	intermittent	1 - 5	1300	1625	EASE (without gloves)
4. Use of cleaning formulations (< 30 % propan-1-ol)	liquid	application	daily	intermittent	1 - 5	840	1260	EASE (without gloves)
5. Use of printing inks (< 42.5 % propan-1-ol)	liquid	mixing, weighing, application	daily	intermittent	0.1 - 1	840	357	EASE (without gloves)
6. Use of disinfectants (< 5 % propan-1-ol)	liquid	disinfecting	daily	extensive	5 – 15	840	630	EASE (without gloves)

[1)] Contact level according to the EASE model

4.1.1.3 Consumer exposure

The table below gives an overview on use of propan-1-ol in consumer products.

Table 4.1.1.3.1 Overview on use of propan-1-ol in consumer products

Product-category	**Maximum content**	**Exposure**
Cosmetic products		
Skin care	2 %	dermal
Mouth hygiene	45 %	oral
Disinfectants	65 %	by inhalation, dermal
Other chemical products		
Lacquers	15 %	by inhalation
Paints, varnishes	unknown	by inhalation
Thinners/diluents	unknown	by inhalation
Cleaners (all kinds)	90 %	by inhalation
Hardener solutions	45 %	by inhalation
Removers of wall paper	6 %	by inhalation

Because there is no information about the amounts of propan-1-ol in thinners/diluents and paints/varnishes, it is assumed that it is similar to lacquers. For estimation of exposure, these three product categories were assumed to contain a maximum amount of 15 %.

Exposure by inhalation

For estimation of exposure to propan-1-ol the CONSEXPO program (RIVM) was used. First of all, for all calculations the physical data were set to the molecular weight of 60, the octanol/water coefficient of 0.34, the vapour pressure was set to 1.94 kPa, and the water solubility to 1 g/cm³. For all scenarios, a common assumption was made that the products are applicated to an area from which the substance is evaporated. Therefore, for the exposure scenario in all cases the CONSEXPO-"Painting" model has been chosen. The assumptions for modelling are given in table 4.1.1.3.2 below.

It should be considered that CONSEXPO 3.0 calculates two concentrations 1) mean per event and 2) average per year. The mean per event characterises the concentration which occurs for

the user when he/she is in contact to the substance. This value is overestimating the concentration because it refers to the personal volume which is only of relevance during working. The rest of the contact time is referred to normal room volume. From this reason for the user two phases of exposure are relevant 1. during use and 2. after use. CONSEXPO does not consider that point. Therefore, the user concentration is an overestimation.

The scenarios chosen for exposure estimations are shortly characterised as follows:

1. Use of disinfectants. This scenario characterizes the application of a disinfectant aqueous solution to a surface, e.g. table, furniture, beds or even parts of walls and floors. This work will mostly occur in bedrooms.

2. Use of household cleaners. This scenario comprises a number of uses with short duration where small areas are cleaned by application of relative small volumes of a cleaning liquid to the surface of e.g. furniture, doors, or glass windows. Because the frequency per day can vary greatly, it is assumed that one use will occur every day. The use of these products may occur in any rooms.

3. Use of paints. This scenario comprises the use of paints for hobby applications, e.g. building models of aeroplanes, ships etc..

4. Hardener solutions. These chemicals are used for repair, in particular of machines and vehicles. From this reason the use of this chemical takes place outside the flat or house in a garage, the size of which is greater than normal rooms.

5. Removers of wall paper. To remove all wall paper in a room of ca 40 m^2, an area between 40 and 50 m^2 has to be painted.

6. Cleaning the kitchen floor. For cleaning the floor in the kitchen, the cleaning solution of about 60 g has to be dissolved in water 1:100.

Table 4.1.1.3.2 Assumptions made for the estimation of inhalation exposure to propan-1-ol (For all scenarios, it is assumed that the product is applied to a surface to build a film/layer)

Use category	Disinfectants (areas)	All purpose cleaners	Paints for hobby purpose	Hardener solutions	Removers of wall paper	Kitchen cleaner	Dimension
Contact scenario	medical care	house keeping	painting	reparing boats or caravans	working at the house	house keeping	
Exposure scenario	painting	painting	painting	painting	painting	painting	
Location	inhouse	inhouse	inhouse	garage	inhouse	inhouse	
Annual frequency of use	52	300	12	1	3	52	1/year
Total duration of contact	12	12	3.5	4	12	2	hours
Use duration	0.5	0.5	0.5	1	3	0.5	h
Exposure scenario							
Volume of room	40	40	25	100	40	20	m^3
Personal volume	5	5	5	5	5	5	m^3
Release area	2	2	0.005	5	50	7.5	m^2
Mass of product	50	2	35	100	1000	60	g
Air exchange rate	8	8	5	20	8	8	m^3/h
Product density	1	1	1	1	1	1	g/ml
Weight faction	65	90	17	45	6	10	%
Fraction of upper layer	0.95	0.95	0.1	0.30	0.50	0.95	
Layer exchange rate	1	1	0.4	0.1	0.1	1	1/h
Mol weigth matrix	50	50	450	100	100	50	g/mol
Substance data							
Part coefficient	0.34						log_{pow}
Vapour pressure	19.4						hPa
Water solubility	1						g/cm^3
Molecular weight	60						g/mol

According to evaluations of the "German Federal Statistical Office", room volumes in Germany vary greatly from ca. 25 m³ up to > 100 m³. The value of 40 m³ was taken because this is a volume which lies in the lower range of living and sleeping rooms, where people spend most of their time. Kitchen room volume was taken from RIVM publication (1999). The value for room ventilation of 0.2 lies in the lower range of ventilation rates as measured by Guo et al. (1992).

The exposure by inhalation was estimated for males and females, either as users or non-users. The anthropometric data needed for this calculation are depicted in the table 4.1.1.3.3.

Table 4.1.1.3.3 Anthropometric data for estimation of exposure to propan-1-ol (defaults from CONSEXPO)

	Body weight*) (kg)	Inhalation rate**) (m³/h)	Exercise	Type of exposure
Male	70	1.62	light	user
Female	60	1.446	light	bystander

*)TGD-defaults **) Consexpo defaults

Taken these assumptions, the following room concentrations were calculated taking the CONSEXPO software, either for users and non-users (bystanders). For users, a fictitous "personal volume" of 5 m³ was assumed, for non-users, the room volume was taken. All estimations of exposure are given for users and non-users. The inhalation absorption rate was assumed to account for 75%.

Table 4.1.1.3.4 Estimated exposure to propan-1-ol by inhalation

Use category	Disin-fectants (areas)	All purpose cleaners	Paints for hobby purpose	Hardener solutions	Removers of wall paper	Kitchen cleaner	Dimen-sion
Cumulated worst case estimated concentrations, CONSEXPO 3.0							
Mean event concentration, non user	303	17	78		261	178	mg/m³
Year average concentration, non user	21	7	0.37		1.1	2.1	mg/m³
Mean event concentration, user	2290	134	388	1611	1520	617	mg/m³
Year average concentration, user	163	5	1.8	1.5	6.2	7.3	mg/m³

Interpretation of concentrations:

CONSEXPO calculates two concentrations 1) mean per event and 2) average per year. Taking the mean concentration per event for the user the total exposure will be overestimated because it refers to the personal volume which is only of relevance during working but not for the rest of the contact, which is not considered by CONSEXPO. However, this overestimation can be accepted under precautional aspects. The value is only used for characterisation of acute risks.

Table 4.1.1.3.5 shows the estimations for exposure, related to body weight, and per day. The values are given for all three populations, as non-users and users, where adequate. Because the absorption is considered in the riskcharacterisation chapter, it is not considered here. The estimates were calculated by the formula: E = C * IR *D, where E is the exposure (mg/kg/bw), C the concentration calculated by CONSEXPO, referred to mean concentration per event or the average concentration per year (table 4.1.1.3.4), IR is the inhalation rate (table 4.1.1.3.3), and D the total duration of contact (table 4.1.1.3.2).

It should be noted that the yearly average concentration for the non-user of 21 mg/m³ from disinfectants is in the same range as the room air concentration estimate for workers exposure (Chapter 4.1.1.2.6 (Use of disinfectants containing propan-1-ol (scenario 6))

Table 4.1.1.3.5 Worst case estimated exposures (CONSEXPO)

Cumulated worst case estimated Exposure	Medical care		Household cleaner		Painting		Repairing boats		Wall paper remover		Kitchen cleaner		Dimension
		activity		activity		activity		activity		activity		activity	
Male, acute exposure	63	nonuser	3.54	nonuser	81	user	112	user	317	user	6.18	non-user	mg/kg/day
Male, (semi)chronic exposure	4	nonuser	1	nonuser	0.37	user	0.1	user	0.23	user	0.07	non-user	mg/kg/day
Female, acute exposure	497	user	29	user	10	nonuser	no exp	nonuser	57	nonuser	22.3	user	mg/kg/day
Female, (semi)chronic exposure	35	user	12	user	0.39	nonuser	no exp	nonuser	0.2	nonuser	0.26	user	mg/kg/day

Data show variability of exposure estimates in relation to the type of preparation and its scenario of usage. For acute exposure, the highest estimated value should be taken. The maximum exposure by inhalation per event (= estimated potential dose rate [I_{inh}]) amounts to 497 (disinfection) mg/kg bw/day for males and females.

The estimation of exposure scenarios of inhalation performed by CONSEXPO revealed that dermal exposure via surrounding air and oral exposures can be neglected.

Dermal exposure

Dermal exposure by use of cosmetics:

For dermal exposure of propan-1-ol in a cosmetic product the estimation is based on the use of 8 g of a skin-care preparation containing 2 % of propan-1-ol. Under the assumption that under worst case conditions 100 % of propan-1-ol will be absorbed dermally, the estimated exposure $U_{der,pot}$ according to TGD will be 160 mg per day (= 2.6 mg/kg bw/day).

Dermal exposure by use of disinfectants:

For dermal exposure by use of propan-1-ol as an ingredient in disinfectants the TGD rules were applied: The concentration (C_{der}) is calculated by the formula (3, page 183 TGD), where average concentration of the undiluted product (Cp) is 0.65 g/ml, and the dilution factor is 1. The concentration on skin is 6.5 mg/cm³. According to eq. (4) the amount on skin is 5.46 mg (including a fraction of 01. remaining on skin) and (5) the potential uptake per kg of bw is 0.09 mg/kg bw/day. The following defaults were assumed: Surface area 840 cm² (hands), thickness of layer 0.01 cm, body weight 60 kg.

The same scenario can be taken for the use of kitchen cleaners, where according to table 4.1.1.3.2, a volume of 60 ml is taken, which is diluted 1:100. According to the equation mentioned for disinfectants, the dermal concentration reveals 7.8 mg/cm³, and the potential uptake 0.11 mg/kg bw.

Oral exposure

Oral exposure by use of mouth hygiene:

The SCCNFP guidance document estimates an amount of 3 g of mouth wash products. This would lead to an exposure of 19.3 mg/kg per day of 1-propanol for a male adult (amount of product swallowed * weight fraction/bw).

As a constituent of fusel oils propan-1-ol may be present in alcoholic beverages in different but low concentrations. Exposure is possible by drinking alcoholic beverages, but can be neglected and is not considered here.

Table 4.1.1.3.6 Total exposure for consumers (users) is summarized as follows:

	Estimated exposure per day (mg/kg bw)		Estimated exposure per year (mg/kg bw)	
Chronic exposure[5]				
Dermal exposure				
disinfectants		0.09		30
Cosmetics		2.6		950
kitchen cleaners		0.11		40
Oral exposure				
Mouth hygiene products		19.3		7000
Inhalation exposure				
	user	non-user	user	non-user
Disinfectants	35	4	12775	1460
General cleaning products	12	1	4380	365
Paints	0.39	0.02	1058	7.3
Hardeners	0.1	0	37	0
Wall paper remover	0.2	0.1	73	36
Kitchen cleaner	0.26	0.07	94.9	25
Sum (chronic exposure)	102[6]	27[7]	26446	1893
Acute exposure				
Dermal exposure				
Disinfectants		0.09		
Cosmetics		2.6		
Oral exposure				
mouth hygiene		19.8		
Inhalatory exposure				
Disinfectants		497		
All purpose cleaners		29		
Paints/thinners/lacquers		126		
Hardener solutions		112		
Removers of wall paper		317		
Kitchen cleaner		22.3		
Highest amount of exposure		≈ 500		

Chronic exposure to propan-1-ol is characterized predominantly by use of cosmetics, mouth hygiene products, disinfectants and general cleaning products and amounts up to ~102 mg/kg bw/day, under consideration of users in the inhalation scenario. Consideration of non-users for inhalation exposure would reveal an overall exposure of 27 mg/kg per day. Dermal exposure via air after evaporation does not play a role for overall exposure.

Disinfectants represent the major source of acute exposures, particularly for the users. A weekly use of these products is assumed. The concentration of propanol of ~ 2300 mg/m³ is

[5] The daily exposure values for semi(chronic) exposures for users were multiplied by 365.
[6] Sum includes inhalation exposure (users), dermal and oral exposure
[7] Sum includes inhalation exposure (non-users) dermal and oral exposure

estimated for this kind of use, the corresponding acute exposure by intake due to this use is 500 mg/kg/day.

It should be kept in mind, that measurements may vary per se considerably: the value of 44 mg/m³ found for occupational use of disinfectants must not convincingly represent a reasonable worst case situation. This value is comparable to the average concentration of consumers as non-users representing the normal consentration in a room.

Secondly, conservative assumptions with different degrees of uncertainty have been set as inputs in the CONSEXPO calculation; in particular the room volume and ventilation represent values at the lower end of a distribution. A sensitivity analysis for the CONSEXPO paint-scenario showed that the variable "fraction of upper layer" (f_{ul}) is proportional to the room air concentration. This means, that doubling f_{ul} doubles the concentration. This value has a quality score of 4[8] in the RIVM documents. From this reason, the uncertainty of the number of f_{ul} is high. In the model, a high value was taken shifting the estimate to the extreme. Furthermore, the model considers the "personal volume", e.g. a fictive space around the user having a small volume of 5 m³. Changing these values, e.g. select a higher room volume and air exchange or lower fraction of upper layer would result in a marked decrease of room concentrations.

Values to be taken for risk characterisation

For acute toxicity the highest amount of exposure should be taken which is 2290 mg/m³ resulting from the use of disinfectants.

For chronic inhalation toxicity, the sum of concentration of all scenarios is ~ 32 mg/m³ (non-users). This should be considered as a precautional value. It should be considered, that for aggregation of exposure the concentrations must not be added, because one person would not use all the products together. Furthermore, medical care in the household requiring extensive disinfecting approach is a relative rare situation. Therefore, for chronic exposure a value of 30 mg/m³ is proposed to be taken forward to the risk characterisation which results from calculations of yearly exposure concentrations to disinfectants, all-purpose cleaners, and kitchen cleaners (cf. table 4.1.1.3.4).

Cosmetics are mainly contributing to dermal exposure of consumers (2.6 mg/kg bw/day) whereas disinfectants and kitchen cleaners can be considered negligible for risk characterisation due to 20-fold lower exposure values and in view of the lower use frequency (weekly).

Oral exposure by mouth hygiene products may lead to a daily dose of 19.3 mg/kg bw, which should be added to inhalation doses for aggregation of exposure.

[8] Single data source supplemented with expert judgment, parameter value doubtful as default value (RIVM)

4.1.1.4 Indirect exposure via the environment

In accordance with the TGD, the indirect exposure of man to propan-1-ol via the environment, e.g. via food, drinking water and air, must be determined. In the form of a worst-case scenario, the most significant point source (in this case: propan-1-ol as solvent; processing of paints) is considered for calculation purposes. This result is then compared with a second calculation which is based on the regional background concentrations (see chapter 3.1.7).

The results of these calculations with the corresponding input values are summarised in Appendix A7. It is necessary to note, however, that the calculation model applied is as yet only provisional. It requires revision as soon as further information is available.

Table 4.1.1.4.1 Input parameter for calculation of indirect exposure

Parameters	Local scenario	Regional scenario
Annual average PEC in surface water[1] [mg/l]	0.040	$8.59*10^{-3}$
Annual average PEC in air[1] [mg/m^3]	0.062	$9.45*10^{-5}$
PEC in grassland [mg/kg]	0.014	-
PEC in agricultural soil [mg/kg]	-	$2.91*10^{-4}$
PEC in porewater of agricultural soil [mg/l]	0.046	$1.50*10^{-3}$
PEC in groundwater under agricultural soil [mg/l]	0.046	-

[1] For the estimation of indirect exposure via the environment, the local concentrations calculated for the emission period have to be averaged over the whole year.

The resultant daily doses for the substance are as follows:

- DOSEtot = 0.036 mg/kg$_{\text{body weight}}$ day (local scenario)
- DOSEtot = $3.119*10^{-4}$ mg/kg$_{\text{body weight}}$ day (regional background concentrations)

The calculated uptake quantities result via the following routes.

Table 4.1.1.4.2 Route percentages of indirect exposure

Uptake route	**% of total uptake**	
	local	**regional**
drinking water	3.61	78.7
fish	0.07	1.76
plant shoot	58.8	10.5
root	0.67	2.53
meat	< 0.01	< 0.01
milk	0.02	0.01
air	36.8	6.49

Drinking water is the most significant route of uptake when using the regional approach. However, the local model indicates consumption of leaf crops and inhalation as main routes for indirect exposure.

4.1.1.5 (Combined exposure)

4.1.2 Effects assessment: Hazard identification and Dose (concentration) - response (effect) assessment

4.1.2.1 Toxico-kinetics, metabolism and distribution

Studies in animals

Absorption, distribution and excretion

Inhalation

There are no data from in vivo experiments available.

Using ex-vivo study on a rabbit trachea Fiserova-Bergerova (1985) showed that concentrations of vapours of propan-1-ol are reduced by 54 % during passage through the trachea (further details are not given).

Dermal

No data available.

Oral

Following single oral administration of 174 mg ^{14}C-propan-1-ol to Wistar-rats gavaged as an aqueous solution concentration in blood was peaked one hour after administration, total recovery rate being about 80% 72 hours after dosage, about 74% of the radioactivity was eliminated in expired air, 5% via the urine and 0.4% in faeces (Fahelbum and James, 1979). Six hours after oral dosing following distribution of radioactivity in tissues of rats (µmol of the dose/g tissue) was found: blood (0.4), brain (0.2), heart (0.3), kidney (0.7), liver (1.3).

The propan-1-ol concentration in blood serum peaked one hour after administration of 50 mmol (3000 mg) propan-1-ol/kg bw by gastric tube to female Wistar-rats (Beauge et al., 1979). From their data a peak blood level of approximately 1800 mg/l after about 1.5 hours could be derived. Thereafter blood levels decreased rapidly to below detection limits at 5 hours after administration.

In mice, peak blood levels of 320, 480 and 510 mg/l were seen at ten minutes (e.g. the first collection point) after orally dosing (intubation) with 1000, 2000 and 4000 mg/kg, respectively. At 20 minutes after administration, levels were 290, 420, and 480 mg/l, respectively. Propan-1-ol was below detection limits 40 minutes after applying the lowest dose and after 80 minutes after applying the two higher doses. An elimination half-life of 57 min was approximated (Maickel and Nash, 1985). In rabbits, 0.9% of an oral dose of 800 mg/kg was excreted conjugated with glucuronic acid in the urine within 24 hours (Kamil et al., 1953).

Other

In-vitro experiments on permeability of 60 mg/l aqueous ^{14}C-propan-1-ol through mucosa preparations of dogs and rabbits indicate that already in oral cavity adsorption occurs (Siegel et al., 1981).

In in vitro experiments using a two-compartment model consisting in water and rabbit or human tissue (kidney, brain, lung, muscle, fatty) propan-1-ol was found to be present in the aqueous compartment only (Kühnholz et al., 1984).

Rietbrock and Abshagen (1971) referred to several studies in which a linear elimination of propan-1-ol from the blood was reported in several animal species. However, when re-examining one of these reports in which 0.8, 1.2, and 1.6 g/kg had been administered to rabbits, they concluded that propan-1-ol was exponentially eliminated from the blood at the two lower doses, while no conclusion could be drawn concerning the highest dose because of lack of essential data. Performing their own experiments by dosing rats intraperitoneally with 1 g/kg, they found an exponential elimination with a half-life of 45 minutes.

Biotransformation:

Propan-1-ol was rapidly oxidized to its corresponding aldehyde by the human and rat liver alcohol dehydrogenase (ADH), more specifically by the Class I isozymes (Ehrig et al., 1988; Sinclair et al., 1990), and to a lesser extent - predominantly in chronic exposure - by the NADPH-dependent microsomal ethanol oxidizing system (MEOS) involving cytochrome P450 (Cytochrome P450 isozyme 3a, isolated from hepatic microsomes of rabbits) (Morgan et al., 1982). Propionaldehyde was then converted to propionic acid which was conjugated with coenzyme A (CoA). AldDH2 (aldehyddehydrogenase) is known to be involved in the metabolism of ethanol via acetoaldehyde. It is further known that functional important polymorphism of AldDH2 exists (Ginsberg et al., 2002). There are no data on the involvement of AldDH2 in the step in which propionaldehyde is converted to propionic acid. In a structure-activity assessment, however, it seems justified to make the assumption that this enzyme is involved and that polymorphism plays a role in determining the internal exposure (AUC) to propionaldehyde. This assumption will be forwarded to the section on MOS. In man and animals propionyl-CoA is carboxylated to methylmalonyl-CoA, this is followed by trans-carboxylation to succinyl-CoA, which subsequently enters the tricarboxylic cycle to be metabolized to carbon dioxide and water (Halarnkar and Blomquist, 1989; Rietbrock and Abshagen, 1971).

Enzymatic esterification of propan-1-ol does not require former oxidation to propionic acid but direct reaction with fatty acids to corresponding esters was demonstrated in rats by Carlson (1993).

In man and rabbits conjugation of propan-1-ol with glucuronic acid or sulfate appeared to be of minor importance (Bonte et al., 1981a, 1981b; Kamil et al., 1953).

Beaugé et al. (1979) reported a rate of metabolism of 510 mg/kg/h which was calculated from an elimination curve consisting of five measurements of blood propanol levels in rats exposed to a single oral (intubation) dose of 3000 mg/kg. Intrahepatic fatty acid metabolism was clearly altered by administration of propan-1-ol. Orally dosed animals were injected i.p. with

albumin-bound palmitic acid (2.5 µCi/kg) 1 h before sacrifice. The administration of propan-1-ol results in a large increase of 1-^{14}C-palmitate incorporation into serum triacylglycerols.

Besides in the liver, propan-1-ol can be metabolized in nasal mucosa as was concluded from combined in vitro/in vivo experiments using surgically isolated upper respiratory tracts of Syrian hamsters and Syrian hamster nasal homogenates. This metabolism may be NAD-dependent oxidation catalyzed by ADH. Its rate was dependent on the inspiratory flow rate (Morris and Cavanagh, 1987).

Endogenous formation of propan-1-ol was observed in drinking experiments using propanol-free alcoholic beverages. Its origin is not clear (Iffland et al., 1989).

Human data

Inhalation

Based on retention data from Pedersen (1987) of ethanol (33%, 4-h human-exposure, 100-140 ppm) and butan-1-ol (46-54%, 0.5-h human-exposure, 120-200 ppm) it may be assumed that the absorption of propan-1-ol will be approximately 40-50% accordingly with measured tissue-gas-partition coefficients of aliphatic C1-C4 alcohols (Fiserova-Bergerova and Diaz, 1986). This is an extrapolated value with a comparatively high uncertainty.

Studies on seven adult male human subjects showed that about 55% of ethanol was absorbed during prolonged inspiration of ethanol (vapour) containing air for periods up to 80 min (ethanol concentration of 10-12 mg/l) (Kruhoffer, 1983). This concentration was chosen to obtain satisfactory accuracy in the analyses of the ethanol concentrations in inspired and expired air, and yet not causing inconvenience to the subjects participating. The ethanol concentrations in air were periodically monitored by mass spectrometry or determined in samples of inspired or expired air by enzymatic analysis. The experiments revealed furthermore that the fractional absorption was not detectably affected by variations in tidal volume (0.7 - 2.1 liters).

In a study on modelling of respiratory exchange of polar solvents Johanson (1991; cited also by Gargas et al., 1993) summarized experimentally observed relative respiratory uptake of some water-soluble solvents in relation to their blood:air partition coefficient. The uptake data for ethanol ranging from about 40 to 75% were collected from a variety of sources cited by Johanson (1991).

Dermal

The penetration through human skin was qualitatively demonstrated in volunteers: Rubbing hands and underarms for five minutes with propan-1-ol containing antiseptics (estimated amount propan-1-ol applied: 9-15 g) resulted in peak levels in blood taken from a foot vein from 0.2 to 0.4 mg/l (Peschel et al., 1992).

Oral

In studies with human volunteers ingesting orange juice containing alcohols (5 mg/kg bw propan-1-ol and 0.8 g /kg bw ethanol) blood levels of propan-1-ol peaked 15 minutes after the ending of the drinking period (30 or 60 minutes), indicating a rapid absorption from the GI tract (Bilzer et al., 1990).

Schmutte et al. (1988) could not detect propan-1-ol in the blood of 9 from 10 volunteers within 15 min after finishing drinking (16 whole blood samples/person, gas-liquid chromatography) of propan-1-ol doses in water of up to 12.5 mg/kg, probably due to a significant 'first pass' effect.

For evaluating the concentration of propan-1-ol in saliva, drinking test was performed with 10 test persons. The alcoholic drink was wine. During the test , the test persons each received 1 g ethanol (given equivalent as wine) /kg bw for 1 hour and the quantities of other alcohols, naturally contained in the beverage. Propan-1-ol concentrations in saliva were found to be up to 4 to 5 times higher than those in blood after the consumption of wine (Hein et al., 1989).

Other

Wehner and Schieffer (1989) administered doses of 25, 50, 100, 200, and 300 mg propan-1-ol intravenously to one male (bw 69 kg) and one female (bw 72 kg) volunteer. Based on a three compartment open system model and a non-linear elimination process controlled by Michaelis-Menten kinetics the authors calculated a Michaelis-Menten constant (K_m) of approximately 10 mg/l and a maximal initial velocity of metabolism (Vmax) of 2.5 mg/l/min.

Tissue-gas partition coefficients were determined for propan-1-ol using head-space methods. Human tissues were obtained by autopsy. Blood and several representative tissues were examined: blood (866 $\pm$ 55), muscle (651 $\pm$ 28), kidney (713 $\pm$ 33), lung (698 $\pm$ 37), brain gray (749 $\pm$ 23) , fat (287 $\pm$ 8). For liver a tissue-gas partition-coefficient of 564 was calculated. The solvent tend to be more soluble in plasma (969 $\pm$ 60) than in erythrocytes (799 $\pm$ 99). It has been shown that solubility of propan-1-ol not increases with lipid content in blood and tissues (Fiserova-Bergerova and Diaz, 1986).

Permeation rates (flux) of pure liquid propanol and aqueous solutions of propan-1-ol, respectively, were determined in a diffusion cell using abdominal skin from human adults: through epidermis - 96 µg/cm²/h vs. 6 µg/cm²/h (Scheuplein and Blank, 1973).

Interactions

Propan-1-ol has a high affinity for ADH. Its absence in blood in some drinking experiments may therefore be explained by a complete oxidation during the first passage through the liver (Iffland et al., 1989).

From enzyme kinetic data it appeared that propan-1-ol is a better substrate for ADH than ethanol and 2-propanol are and hence can retard the metabolism of these alcohols (Rietbrock and Abshagen, 1971). On the other hand, when the concentration of ethanol in blood was high

enough to saturate ADH, the metabolism of propan-1-ol was found to be retarded in drinking experiments with volunteers (human data from Bilzer et al., 1990; Bilzer and Penners, 1985; Bonte et al., 1981b; Ehrig et al., 1988 and Wehner and Schieffer, 1989).

From experiments with two volunteers (a) absence of ethanol; b) blood ethanol concentration 0.3 vs. 1.3 $^{o}/_{oo}$), injected intravenously with 25 - 300 mg propan-1-ol, and using a three compartment model, Wehner and Schieffer (1989) calculated a hepatic clearance of 10.2 l/min (7 to 8 times higher than for ethanol) and pointed out that this value is derived from the model whereas the liver blood flow in humans is four times lower. Hence, the calculated constants should be taken with caution. An increase of the ethanol exposition causes a prolongation of the mean residence time of propan-1-ol in the body. This phenomenon was interpreted as the result of an inhibition of the propan-1-ol metabolism by ethanol.

Conclusions

There are no data on the toxicokinetics of propan-1-ol concerning exposure by inhalation. Based on comparison with data on other short-chain alcohols retention after inhalation may be approximately 40-50 %. From the available data it can be concluded that dermal absorption occurs. Considering the physicochemical properties (molecular weight 60 g/mol, complete water solubility and a log Pow of 0.34) absorption through the skin can be assumed. The compound is readily absorbed from the GI tract (about 80%). Propan-1-ol is readily metabolized via its aldehyde to propionic acid which can then be converted by a number of pathways (e.g., the citrate cycle). Its metabolism may be retarded by already-present ethanol.

Taking into account the indicated high uncertainties in the inhalatory absorption figure and the described differences for ethanol in inhalation absorption in humans ranging from 30-75% (Gargas et al. 1993; Kruhoffer 1983) an inhalation absorption rate of 75% is proposed as a reasonable worse case assumption for risk characterisation purposes. Under worst case assumptions 100% absorption through dermal and oral exposure route, respectively, was taken performing calculations in risk characterisation.

4.1.2.2 Acute toxicity

Animal studies

Oral

For young rats weighing 90 to 120 g a LD50 value of 1870 (1340 - 2000) mg/kg is reported (Smyth et al. 1954; Kennedy and Graepel 1991). For young adult rats in the weight range of 180 - 350 g the LD50 value is 6500 (5800 - 7280) mg/kg with death occurring within 2 - 18 hours (Taylor et al. 1964). Another LD50 value of approx. 8000 mg/kg was obtained with rats of unspecified age (BASF AG 1974). Clinical signs consisted of dyspnoea, apathy, ventro-lateral position atonia and exsiccosis (BASF AG 1974) or of coma and scrawny appearance (Taylor et al. 1964). Autopsy findings showed dilatation of the heart, passive hyperemia, reddening of the glandular part of the stomach and liquid contents of the intestine (BASF AG 1974). For mice a LD50 value of 5467 mg/kg (Savini 1968) and for rabbits a LD50 value of 2823 mg/kg and a median narcotic dose of 1441 mg/kg (Munch 1972) are documented.

Inhalation

Sprague Dawley rats (5 males and 5 females/group) were exposed for 4 hours to vapour concentrations of propanol by whole body exposure either by dynamic or by static exposure (according to OECD TG 403; Union Carbide Corp. 1992). Dynamic generation and exposure system included airflow, while with the static generation and exposure system no additional air was supplied. For the dynamic exposure the rats were individually housed while for the static exposure rats were housed per sex (5 per cage) and exposed in a 120 liter chamber.

Three dynamic exposure concentrations were tested: 13548 ppm (33870 mg/m^3), 9741 ppm (24350 mg/m^3), 5185 ppm (12960 mg/m^3). No rats died during exposure or during the 14-day post exposure period. On the day of exposure there was evidence of ocular and nasal irritation and hyperactivity. Additional signs observed for animals of the medium and higher concentration groups included abdominal breathing, increased shallow respiration, narcosis and/or prostration and other signs. During the post-exposure period no clinical signs were observed. Mean body weight gains for all groups were normal at days 7 and 14. No macroscopic lesions were observed at the end of the 2-week observation period. CNS depression appeared at dose levels above the cut-off values of 20000 mg/m^3 regarding EU classification R 67.

One static exposure concentration (16760 ppm = 41 880 mg/m^3) was tested for either 4 hours or 6 hours. After the 4 hour exposure 2/5 males and 0/5 females died. After a 6 hour exposure 5/5 males and 3/5 females died. Death occurred during exposure and within 1 day thereafter. Clinical signs were similar to the dynamic exposure but an additional sign prior to narcosis was ataxia. Body weight gain was normal during the second week. In animals that died the main autopsy findings were discoloration of the lungs and dark purple discoloration of the liver. No pathological findings were observed in animals sacrificed at the end of the 2-week observation period (Union Carbide Corp. 1992).

There are data on two inhalation hazard tests. In both tests the rats were exposed to a saturated vapour atmosphere at 20 degrees Celsius, which is equivalent to a substance concentration of 47 050 mg/m^3. After an 8-hour exposure 1/12 rats died and after a 7-hour exposure there were no deaths (0/12). Irritant reactions of the mucous membranes, increased respiration followed by narcosis were observed. Autopsy of the animal that died showed dilatation of the heart, passive hyperemia, edema and hyperemia of the lung (BASF AG 1974, 1980).

Dermal

Four rabbits were exposed for 24 hours under occlusive conditions. A dermal LD50 value of 5.04 ml/kg (4052 mg/kg) is reported. The observation time was 14 days. There are no data on clinical signs and autopsy findings (Smyth et al. 1954).

Human data

A woman died 4-5 hours after ingestion of approximately 400 - 500 ml (322 - 401 g) of the substance as a solvent in a hair lotion. It is mentioned that she could have ingested this preparation and perfumes more than once in the past. Autopsy revealed a "swollen brain" and lung oedema, the substance was detected in the bowel (Dürwald and Degen 1956). Taking into account a median body weight of 70 kg the lethal dose for humans may be in the range of 4.60 - 5.77 g/kg bw. Though the authors concluded that death was caused by the ingestion of propanol it may be possible that other unknown components in the hair lotion may have contributed to the death.

In 4 subjects with normal smell (normosmics) and two subjects lacking a functional sense of smell (anosmics) the threshold for odor was ca. 15 ppm (28 mg/m^3) for normosmics and ca. 2600 ppm (6500 mg/ m^3) for anosmics (Cometto-Muniz and Cain 1990).

Conclusion

The oral LD50 ranges from 1870, 6500 to 8000 mg/kg in the rat. For rabbits this value is 2823 mg/kg and for the mouse 5467 mg/kg. It is concluded that these data do not warrant a classification labelling for acute oral toxicity. None of the tests were conducted according to OECD Guidelines.

The value of 42000 mg/m³ (resp. 16800 ppm) after a 4-hour exposure is considered as an approximate estimate of a LC50 value (study according to OECD TG 403). In two inhalation hazard tests there were 1/12 or 0/12 deaths after an exposure of up to 8 hours to a substance concentration of 47 000 mg/m^3. These findings do not warrant a classification and labelling for acute inhalation toxicity.

The dermal LD50 value is 4052 mg/kg for the rabbit. There is no need of classification and labelling for acute dermal toxicity. This test was not conducted according to OECD.

A case of acute oral poisoning in one women suggests that the lethal dose may be in the range of 4.6 - 5.8 g/kg bw. Though the authors concluded that death was caused by the ingestion of propanol it may be possible that other unknown components in the hair lotion that was ingested may have contributed to the death. Based on these data no classification and labelling for acute oral toxicity is warranted.

The odor threshold for normosmics is 28 mg/m^3 (15 ppm) and the pungency threshold is 6500 mg/m^3 (2600 ppm) for anosmics.

4.1.2.3 Irritation

Animal studies

A weak skin irritation was observed in 5 rabbits after a 24-hour contact with 0.01 ml of undiluted substance. A grade 1-reaction was observed indicating the least visible capillary injection (Smyth et al. 1954). An unspecified number of rats were exposed from 1.5 minutes

up to 20 hours of the undiluted substance (amount not specified); 24 hours later a weak reddening was observed. At 8 days there were weak scales and weak scabs (BASF AG 1974).

Instillation of a very small volume (0.005 ml) of the substance resulted in grade 5-reactions of the rabbit cornea. This corresponds a necrosis of 63 % to 87 % of the cornea (Smyth et al. 1954). Instillation of 50 mg of undiluted substance to the eyes of rabbits resulted in partial gray discoloration, mild reddening and strong oedema of the conjunctiva and the nictitating membrane, mild opacity and suppuration at day 1. At 8 days strong reddening, strong oedema and strong corneal opacity together with vascularization, suppuration and scar formation was observed (BASF AG 1974).

There are several reports on the RD50 (50% decrease in respiratory rate). Mice (4 animals per group) were exposed for 10 minutes to 7 concentrations of n-propanol ranging from ca. 4000 ppm (10 000 mg/m^3) to ca. 28 000 ppm (70 000 mg/m^3). A RD50 value of 12 704 ppm (31 760 mg/m^3) was determined. The substance is considered a weak/very weak sensory irritant (Kane et al. 1980). In normal mice or in cannulated mice (trachea) (4 animals per group) the sensory irritation or the pulmonary irritation, respectively were determined after a 30-minute exposure. Sensory irritation and pulmonary irritation are effects due to activation of the trigenimus nerves and the vagus nerves, respectively. For both effects RD50 values were obtained within the first minutes and remained almost constant during the exposure period. The RD50 values are 17 967 ppm (44 230 mg/m^3) for sensory irritation and 15 593 ppm (38 980 mg/m^3) for pulmonary irritation. Respiratory rates after a 30-minute exposure were in the range of untreated mice at the end of the observation period of 50 minutes (Kristiansen et al. 1986). Nielsen and Bakbo (1985) quote two references with RD50 values of 12 704 ppm (31 760 mg/m^3) and 4770 ppm (11 930 mg/m^3) for mice.

Human data

Ten subjects received a closed patch test for 10 minutes with 0.3 ml substance to the volar aspect of both forearms. One forearm was hydrated by the immersion in water at 33 degrees Celsius for 10 minutes before the application of the patches. No reaction was seen at the nonhydrated site. Hydration resulted in 7/10 subjects in trace to notable erythema formation (Haddock and Wilkin 1982).

Twenty volunteers rubbed 3 to 5 ml of the substance (50 %) into the hands until dry 15 times a day, 5 days a week and for 2 weeks per preparation. Appearance, intactness and turgor was slightly but significantly affected. These reactions were significantly less obvious when cosmetic ingredients were added to the preparation (Rotter et al. 1991).

Propanol and other substances cause formation of vacuoles on the cornea but do not result in scar formation. These findings have been observed with furniture foremen and lacquer workers after accidental exposure (Heydenreich 1966).

Conclusion

A weak skin irritation was observed in rabbits after an exposure of up to 24 hours in two tests that were not conducted according to OECD Guidelines. However, it can be concluded that these findings do not warrant a classification and labelling for skin irritation. The data obtained with humans (patch test with 0.3 ml substance for 10 minutes) did not indicate a skin irritating effect in untreated forearms. In humans no skin irritation was observed on the untreated skin but slight erythema was observed when the treated skin site was immersed in water for 10 minutes at 33 degrees Celsius before exposure to the substance. After frequent exposure of the hands to a 50 % preparation for two weeks appearance, intactness and turgor was slightly but significantly affected. Taking also into account the defatting solvent character of propan-1-ol it is assumed that frequent contact can lead to skin dryness or cracking of skin. Consequently, classification with R 66 (Repeated exposure may cause skin dryness or cracking) is proposed.

Instillation of the substance to the eyes of rabbits resulted in serious damage (e.g. strong oedema, strong opacity, vascularization). The data were obtained with two tests that were not conducted according to OECD Guidelines. Nevertheless, a classification as "Xi, Irritant" and labelling as "R 41, Risk of serious damage to eyes" is warranted. This labelling is supported by data on humans. After accidental exposure at the workplace to propanol and other substances vacuolization of the cornea without subsequent scar formation has been reported.

There are several reports on RD50 testing (50% decrease in respiratory rate). A RD50 value of 12 704 ppm (31 760 mg/m^3) was determined for mice. The substance is considered a weak/very weak sensory irritant (Kane et al. 1980). In normal mice or in cannulated mice (trachea) the sensory irritation or the pulmonary irritation were determined after a 30-minute exposure and resulted in a RD50 value of 17 967 ppm (44 230 mg/m^3) for sensory irritation and a RD50 value of 15 593 ppm (38 980 mg/m^3) for pulmonary irritation obtained within the first minutes, which remained almost constant during the exposure period (Kristiansen et al. 1986). Nielsen and Bakbo (1985) quote two references with RD50 values of 12 704 ppm (31 760 mg/m^3) and 4770 ppm (11 930 mg/m^3) for mice. On the basis of these data the substances is considered a weak sensory irritant and labelling for respiratory irritation is not considered appropriate.

4.1.2.4 Corrosivity

Animal studies

The substance is not corrosive to skin but causes serious damage to eyes (Smyth et al. 1954; BASF AG 1974).

Human data

Data obtained on humans do not demonstrate a corrosive effect (Haddock and Wilkin 1982; Rotter et al. 1991). Accidental ocular exposure results in vacuolization of the cornea but not in scar formation, indicating a serious damage to eyes (Heydenreich 1966).

Conclusion

The animal data obtained for skin and eye irritation demonstrate a weak effect on the skin and an irritating effect to the eye. There is no corrosive effect on humans (see 4.1.2.3). There is no need to classify the substance as corrosive.

4.1.2.5 Sensitisation

Animal studies

With three test methods (Mouse Ear Swelling Test, MEST; Magnusson Kligman test and Buehler test) a negative result was obtained. With all three tests the substance concentration tested was 100% and all tests were in compliance with international testing standards (Gad et al. 1986).

Human data

A positive patch test reaction to propan-1-ol (test concentration: undiluted or 50 %) was elicited in a woman sensitive to 2-propanol and 1-butanol but not to primary alcohols with less than 3 C atoms and other substances. The woman worked in the laboratories of a cosmetic company and she was primarily exposed to commercial isopropyl alcohol (Ludwig and Hausen 1977).

Fifty subjects were patch tested with nine 24-hour applications of 0.2 ml substance over a three week period and challenge patching 10 to 14 days later. In all subjects the response was negative (Gad et al. 1986).

Conclusion

Animal and human data demonstrate that the substance has no skin sensitizing potential. There is only one report of a woman showing allergic skin reactions after patch testing to propan-1-ol and 2-propanol and other substances. There is no need to classify and label the substance for skin sensitizing properties.

There are no data on sensitization by inhalation.

4.1.2.6 Repeated dose toxicity

Animal studies

Oral

Only a few data are available concerning the oral exposure of animals.

In a study designed for research purposes a non specified number of rats (Wistar) received a dose of 25 720 mg/kg propan-1-ol an aqueous solution of 32 % (v/v) in drinking water. Doses were calculated as 25 ml water uptake per day at an average body weight of 250 g. The application period lasted from one week up to 3 months (Wakabayashi et al. 1991). The calculation of the dose in the study has been estimated from the only information given in the paper (i.e. 32% v/v in drinking water). The amount of water intake per day and body weight cannot be exactly verified.

Electron microscopic studies of the liver showed a mixed population of small and enlarged mitochondria, i.e. irregularly shaped megamitochondria with few cristae, and normally sized but irregularly shaped mitochondria with a decreased number of cristae. The number of enlarged mitochondria per hepatocyte increased with prolongation of the duration of the experiment and more than 1 month was required to induce those changes.

In a 7-day gavage study 6 rats (no strain and sex identification) received 800 or 1620 mg/kg propanol-1-ol per day. Propan-1-ol caused a significant, dose-related decrease of 46-73% in the hepatic contents of the vitamins of the B-group, i.e. thiamin, riboflavin, pyridoxine, niacin and pantothenic acid after an oral application for 7 days (Shehata and Saad 1978). The finding of diminished Vitamin B concentrations in the liver can only be taken as an isolated information. Without further results on clinical biochemistry and haematology the adversity of these data cannot be assessed. Comprehensive description of histopathological findings in the liver is lacking.

In a study with 25 male mice three dose groups received 1000, 2000 and 4000 mg/kg/day on five consecutive days per gavage. Body temperature, rotarod performance and levels of alcohol in blood were examined at five time points (10, 20, 40, 80 and 120 min) each day. The maximum blood levels for propan-1-ol were seen at 10 min after administration. The maximal hypothermic response was observed at either 10 or 20 min after the administration of propan-1-ol, thereafter body temperature returned toward normal levels. The reduction of body temperature (1° to 2°C) was dose-related and significant. The acute effects of propan-1-ol on motor activity, as measured by impairment of rotarod performance, were dose- and time-dependent and could be correlated with concentration curves for propan-1-ol in the blood. Within 2 hours after administration the rotarod performance improved (Maickel and Nash 1985).

In a limited repeated dose study on male rats (drinking water/4 months) a nominal dose of 3000 mg/kg did not induce relevant toxic effects. Investigated parameters were restricted to body weight, food- and water consumption and alcohol uptake as well as liver weight and histology (Hillbom et al. 1974). Neither inflammation nor cirrhosis was seen in any of the livers. Thus, the dose of 3000 mg/kg bw/day is considered as NOAEL.

There is a vague indication on toxic effects of propan-1-ol in rats (at 240 mgkg bw twice weakly) treated for life-time from a non-guideline cancer study (Gibel et al., 1975, see 4.1.2.8). Hepatotoxic effects were observed in ‘nearly all animals irrespective of the applied alcohol’ (results from 2-methylpropanol and 3-methylpropanol were not considered in this report). Observed effects were congestion, steatosis, necrosis, fibrosis, zirrhosis and extramedullary and medullary hyperplasia of the haematopoietic bone marrow parenchyma. Neither the lesions were explicitely assigned to one of the alcohols tested nor the incidences

of these lesions were reported for propan-1-ol. The authors concluded that liver toxicity and haematotoxicity was related to all three test substances. **This study of limited validity (cf. 4.1.2.8, too) cannot to be used for NOAEL derivation**.

Inhalation

Only insufficient data are available concerning the inhalative exposure of animals.

Rats (Sprague-Dawley) were exposed to 100, 500 and 1000 ppm (246, 1230, 2460 mg/m^3) for 6 hours/day 5 resp. 4 days/week up to 2 weeks (9 exposure days). No mortalities occur during this study. Exposure-related clinical observations were limited to slightly swollen periocular tissue, and minimal perinasal and periocular encrustations in the 1000 ppm exposure concentration group (Bushy Run 1992). Because there was no correlation in histopathology and other parameters at all doses, 500 ppm (1230 mg/m^3) can be regarded as NOAEC for irritation after repeated exposure.

Dermal

There are no data available.

Human data

There are no human data on repeated toxicity.

Conclusion

All available information (oral and inhalation studies) are insufficient with regard to current requirements. Nevertheless, it seemed to be clear from these insufficient data base that major toxic effects resulting from repeated dose treatment comprise narcotic/neurotoxic (alcohol related) reactions in relation to relative high doses/concentrations.

The limited repeated oral investigations by administration of propan-1-ol to rats indicate hepatotoxic effects at high doses.

Based on the available oral repeated toxicity data an overall NOAEL of 3000 mg/kg bw/day will be established. Regarding the inhalative route a NOAEC of 500 ppm (1230 mg/m^3) for irritative effects can be derived from limited studies described.

Because of the lack of valid data for propan-1-ol, a scientifically sound risk characterisation on repeated dose toxicity cannot be performed. Formally, there is a conclusion i, i.e. the need for further information and/or testing (90-day rat inhalation study).

Propan-1-ol is also notified as an active substance within the scope of the Biocide Directive 98/8/EC. The necessary data on repeated dose toxicity is existing, but is owned by a company

who wishes to use it in the framework of other EU regulation (the Biocides Directive). The company is so far not willing to make the study available to support risk assessment in the context of the Existing Substances Regulation, and there are no provisions in the Biocides Directive that would force them to share the data with other companies. The information on repeated dose toxicity is requested for reasons of human health. For the sake of animal protection it is hoped that the companies involved will be able to negotiate and share the data.

4.1.2.7 Mutagenicity

Bacterial assays

In two gene mutation tests with Salmonella typhimurium propan-1-ol was negative; however, detailed data on methodologies and results are not available. Negative results were reported with and without S-9 mix for tester strains TA98 and TA100 by Khudoley et al. (1987; dose not given) and for tester strain TA100 by Stolzenberg and Hine (1979; doses up to 6000 µg/plate).

Hilscher et al. (1969) investigated the potential of 4 % propan-1-ol (32 000 µg/ml) for induction of revertants in E. coli strain CA 274 without S-9 mix and found an inconclusive result. In a specific sub-strain of E. coli SA500 propan-1-ol induced mutations suppressing the lethal loss of replication control from a prophage fragment of bacteriophage lambda (Hayes et al., 1990). Investigations in this non-routine system were done without S-9 mix. Induction of mutations was limited to extremely high doses which led to strong toxic effects. With a dose of 5 % (v/v; equivalent to 40000 µg/ml) the mutation frequency was increased by a factor of 1.6 as compared to the negative control; the relative survival was 5 %. With doses of 6 % and 7 % propan-1-ol stronger increases in mutation frequencies were obtained; however, relative survival ranged from 0.08 to 0.008 %. Altogether, the finding is inconclusive.

In the SOS chromotest with E. coli (induction of SOS repair) a negative result was obtained with and without S-9 mix for doses up to 100 mmol/l (6000 µg/ml; von der Hude et al. 1988).

Yeast assays

An induction of aneuploidy in Aspergillus nidulans was reported by Crebelli et al. (1989) in the absence of exogenous metabolic activation. Exposure to extremely high doses of 1.8% and 2.0 % (v/v; 14 400 and 16 000 µg/ml) led to aneuploidy frequencies of 1.64 % and 5.41 %, whereas in the negative control the aneuploidy frequency was 0.26 %. These doses led to clear cytotoxicity (39 % and 14 % relative survival), after exposure to 2.5 % propan-1-ol (20 000 µg/ml) relative survival was decreased to 9 %. For the same dose range there was no induction of mitotic recombination.

Mammalian cell culture assays

A well conducted chromosomal aberration assay with V79 cells (BASF, 2003) was neagative with and without S-9 mix for doses up to 600 µg/ml in two independent experiments. The highest tested dose corresponds to 10 mmol/l limitation. No toxic effects were observed.

In V79 cells without S-9 mix, propan-1-ol did not induce micronuclei in a dose of 50 µl/ml (40 000 µg/ml; Lasne et al. 1984). Treatment was for 1 h, followed by 18 h-recovery; 7000 cells were analyzed.

Tests for induction of sister chromatid exchanges (SCE) were negative in V79 cells with and without S-9 mix for doses up to 100 mmol/l (6000 µg/ml; von der Hude et al. 1987) and in CHO cells without S-9 mix for a dose of 0.1 % (800 µg/ml; Obe and Ristow 1977).

In vivo assays

An in vivo chromosomal aberration test with rats (bone marrow assay) led to an inconclusive finding due to limitations in data presentation (Bariljak and Kozachuk 1988). In the exposed group (8 animals, 500 cells) 2.2 % of bone marrow cells carried chromosomal aberrations (exclusive gaps), in the negative control group (10 animals) no chromosomal aberrations were found in 600 cells. Furthermore, it is also reported on polyploidy frequencies 2.4 % in the exposed group and 0.5 % in the negative control. The dose of propan-1-ol is given as 1/5 of the LD-50, the route of administration is not specified.

Conclusion

Bacterial genotoxicity tests were negative (with the exception of one inconclusive finding); however, the data are not fully reliable due to inadequate reporting. In yeast, aneuploidies were induced only in extremely high doses and only in a small dose range (14 400 to 16 000 µg/ml). In mammalian cell cultures propan-1-ol was negative for chromosomal aberrations (tested with and without S-9 mix), micronuclei (tested only without S-9 mix) and for sister chromatid exchanges (tested with and without S-9 mix). The only in vivo test was inconclusive.

There is no relevant concern with respect to mutagenicity. Propan-1-ol should not be classified as a mutagen.

4.1.2.8 Carcinogenicity

There are only invalid carcinogenicity studies on rats (Gibel et al. 1975) and mice (Bushy Run 1980) available.

A group of 18 Wistar rats of both sexes received doses of 240 mg propan-1-ol/kg bw by gavage, twice a week, for their lifetime (Gibel et al. 1975). The control group, comprising 25 rats, received saline. The average survival time was 570 days for the exposed rats, and 643 days for the control group. It was reported that "nearly all rats" showed liver damage including congestion, steatosis, necrosis, fibrosis, and extramedullary and medullary

hyperplasia of the haematopoietic bone marrow parenchyma. However, the incidence of these lesions were not reported.

Table 4.1.2.8

Tumour incidence in Wistar rats exposed orally to propan-1-ol for lifetime

Organ / tissue affected	Tumour types	Incidence	
		exposed	controls
Blood	myeloid leukemia	2/18	0/25
Liver	carcinoma	1/18	0/25
Liver	sarcoma	2/18	0/25
Other	carcinoma	0/18	0/25
	sarcoma	0/18	0/25
	benign tumours[a]	10/18	3/25

[a] Mostly papillomas and mammary fibroadenomas

This study is considered to be inadequate for the assessment of carcinogenicity. The dosing regime did not conform to standard protocols. Too few animals were used in each dose group, the sex ratio of each group was unclear, no data were provided on the histological type of liver sarcoma, and no statistical analysis was conducted.

The increased incidences of malignant tumors in liver and bone marrow and of the overall tumor rate compared to the control animals (Gibel et al. 1975) might raise some concern that propan-1-ol has a carcinogenic potential. Negative genotoxicity data indicate that propanol is not a genotoxic compound. If at all, other modes of action should be considered. Since it was reported in this study that nearly all dosed rats showed liver fibrosis and necrosis, hepatotoxicity might be taken into account.

In the other lifetime study male C3H mice were dermally treated with 40 mg undiluted propan-1-ol/application 3 times per week (Bushy Run 1980). No skin tumours were observed. This study, although the conclusion reaches no evidence for skin carcinogenesis, is invalid, because of a number fundamental limitations and the complete lack of any to date requirements (i.e. insufficient number of animals, only one sex only one dose group, age of animals at the start of dosing, different age control and test group at the beginning of the study, arbitrary choice of locations for histopathology, inadequate reporting of the study).

Conclusion

There is no valid carcinogenicity study available. Thus, a risk assessment for carcinogenicity can not be performed. However, taking into account the negative mutagenicity data it is concluded that carcinogenicity should not be an endpoint of concern.

4.1.2.9 Toxicity for reproduction

Fertility Impairment

Human data

No data available.

Animal studies

Inhalation

Information on fertility data is available from investigations on behavioral teratology with Sprague Dawley rats (Nelson et al., 1985; 1989). Propan-1-ol vapors at target concentrations of 3500 and 7000 ppm (8730, and 17460 mg/m³) were administered by whole chamber exposure to either males or to sperm-positive females mated to unexposed males. As exposures were conducted approximately three months apart in this study, separate sham-treated control groups (filtered air) were run for the two concentrations of propan-1-ol. Exposures for females (n=15/group) were conducted during 7 hours/day on gestation days 1-20 (sperm positive=day 0), and for males (n = 18/group, consisting of three sets of six males with exposures beginning on succeeding weeks) for 7 hours/day for a period of six weeks. Exposed males were subsequently mated (1:1) to unexposed virgin females for a maximum of 5 days.

It was reported for the exposed females that the higher concentration of propan-1-ol reduced maternal weight gain and feed intake (data not given), whereas no effects were found on maternal weight gain or feed intake at 3500 ppm. No significant differences were found among any of the groups for the length of gestation, the number of live pups per litter, or neonatal survival.

With the trial on 3500 ppm propan-1-ol, the number of pregnant/number bred was 17/18 for maternally-exposed rats, 17/18 for paternally-exposed rats, and 18/18 for the controls. With the trial on 7000 ppm propan-1-ol, the number of pregnant/number bred was 17/17 for maternally-exposed rats, 2/16 for paternally-exposed rats, and 18/18 for the controls. In spite of sperm plugs apparent for the 16 paternally exposed males, only two litters resulted. One had a litter of 12, and the other had a litter of only two pups. Mated females from the exposed males without litters were sacrificed, and after examination of the uterus indicated that pregnancy had not occurred, i.e., no resorption sites were detected. Because of the infertility in males in the 7000 ppm exposed group, the last six exposed males were retained (the others had been sacrificed before the infertility was noted) and were subsequently mated at biweekly intervals. The fertility effects observed in males revealed to be reversible within 13 weeks. The numbers of males which produced litters were as follows: 1/6 in week 1, 2/6 in week 3, 4/6 in week 5, 4/6 in week 7, 4/6 in week 9, 3/6 in week 11, 6/6 in week 13, and 6/6 in week 15. It was reported, that once a male sired a litter, he was generally fertile thereafter.

Conclusion

In summary, propan-1-ol did not induce detectable effects on fertility in males and in females of the 3500 ppm (8730 mg/m^3) exposed group and in females of the 7000 ppm (17460 mg/m^3) exposed group. According to the exposure conditions of the study, the statements related to female fertility are limited to the functional aspects of maintaining adequate and sufficient conditions for early extrauterine development of the conceptus and for its nidation. Exposure of male rats to 7000 ppm propan-1-ol for 6 weeks produced reversible infertility. Based on the latter finding the NOAEC/fertility is established at 3500 ppm (8730 mg/m^3). With the assumption for rats of a respiratory rate of 0.8 L/min/kg, the effective concentrations (reversible effects on male fertility) of 7000 ppm for 7h/day can be converted to an oral uptake of approximately 5.8 g/kg bw/day. Thus, the effects on male fertility revealed from this investigation at very high inhalatory concentrations of propan-1-ol are not considered appropriate to justify classification and labelling as toxic to reproduction.

Oral, dermal

Oral or dermal studies are not available.

Developmental Toxicity

Human data

No data available.

Animal studies

Inhalation

Propan-1-ol was tested for developmental toxicity in a teratogenicity study with Sprague-Dawley rats (Nelson et al., 1988; 1990; 1996). During this study propan-1-ol vapors at target concentrations of 3500, 7000 and 10000 ppm (8730, 17460, and 24940 mg/m^3) were administered by whole chamber exposure to groups of 15 sperm positive females during gestation days 1-19 (sperm positive=day 0) for 7 h/day. Sham-treated controls received filtered air. Maternal weights were recorded daily for the first week and weekly thereafter. Feed and water intake was recorded at weekly intervals (g.d. 0, 7, 14, 20). Necropsy was performed on g.d. 20, when numbers of corpora lutea, resorptions (classified as early, middle or late) and live fetuses were recorded. Fetuses were examined for external malformations, weighed, and sex was determined by external examination. Half of the fetuses were randomly selected and examined for either skeletal malformations or variations or for visceral malformations or variations.

No clinical effects were observed in the exposed dams. It was reported that at 10000 ppm feed intake was reduced throughout gestation and maternal body weight was significantly affected at the end of gestation (data not given). At 7000 ppm feed intake was reduced during the last two weeks of gestation, but body weight gain was not significantly affected. At an exposure of 3500 ppm feed intake was reduced by about 10 % compared with controls, but this was not

statistically significant. No differences were observed in the exposed groups and in the controls on numbers pregnant/numbers bred or on the mean number of corpora lutea/dam or on the mean number of implants/dam. Three out of fifteen litters exposed to 10000 ppm were totally resorbed, and thus the ratio of implants resorbed/litter was statistically significantly increased ($p<0.05$) in this group to 57 % in comparison to 6 % in controls. Fetal weights were statistically significantly reduced ($p<0.05$) in the exposure groups of 7000 and 10000 ppm, but not at 3500 ppm. After exposure to 10000 ppm the incidence of external malformations was significantly increased compared with the controls (approximately one-third of the fetuses having a short or missing tail or ectrodactyly. A similar increase was seen for skeletal malformations (mostly rudimentary cervical ribs) and for visceral malformations (principally cardiovascular or urinary defects). These were all increased after exposure to 10000 ppm, whereas only skeletal malformations were increased at 7000 ppm. After exposure to 3500 ppm no changes in skeletal or visceral malformations or variations were revealed when compared to the controls.

During the above mentioned study on behavioral teratogenicity of propan-1-ol (Nelson et al., 1985; 1989) the female Sprague Dawley rats that had been exposed to either 3500 or 7000 ppm during gestation days 1-20 were allowed to litter and their offspring subjected to behavioral testing. After parturition the litters were arbitrarily culled to 4 pups of each sex and fostered to untreated dams. Offspring were weaned from their foster mothers on postnatal day 25, and weighed individually on days 7, 14, 21, 28, and 35. Seven behavioral tests spanning postnatal days 10 through approximately 90 were applied at several stages of postnatal development. Tests on neuromuscular ability, activity, and learning were: ascent of a wire mesh screen, rotorod, open field and optically monitored activity, running wheel, avoidance conditioning, and progressive fixed ratio schedule of reinforcement. Female and male pups were selected randomly, ear-marked and assigned to the test groups on postpartum day 10. For each test, one female and one male were used from each litter. It is reported from the study (data not given) that behavioral testing revealed no differences from the controls after maternal exposures to propan-1-ol.

Oral

Neonatal rats were exposed to propan-1-ol via an artificial milk formula for 4 consecutive days and a recovery period of 10 days. On postnatal days 5, 6, 7 and 8 the neonates received propan-1-ol doses of 3800, 7500, 3000 and 7800 mg/kg bw respectively via stomach tube. Twenty-one of the animals completed the experiment. Seven of the animals died: four resulting from surgical complications and three to apparent propanol overdose. It is reported that during the period of propanol administration, the alcohol exposed animals were observed to be intoxicated, frequently showing an impaired righting response. After the 18th day of age there were no effects on body weight or on absolute weight of kidneys, heart, or liver. But the absolute and relative brain weights were decreased in the exposed neonatal rats. The amount of DNA was in all brain areas decreased. Cholesterol levels were decreased in the forebrain and cerebellar samples (Grant and Samson 1984).

The study by Grant and Samson (1984) is of limited validity and not appropriate to be used as a study to derive a NOAEL for developmental toxicity (or repeated dose toxicity) due to the following reasons: a completely artificial rearing procedure had been used including implantation of a gastric catheder by surgical procedure to 5 day old pups; the pups were

taken off their dams during the whole treatment period and kept isolated in individual plastic cups to compensate for maternal deprivation; 25% of neonates died due to surgical complications as well as from apparent propan-1-ol overdose. It is reported that the alcohol exposed pups were intoxicated during the period of propan-1-ol administration (cf. impaired righting response indicating acute neurotoxic effects) and, furthermore, that pups suffered from acute withdrawl symptoms 8 h after the last exposure (including spontaneous seizures, full body shakes, severe head bobbing, etc.).

The test conditions used in the study are characterised as testing effects of alcoholic insults after acute intoxication rather than testing repeated exposure to not acutely toxic alcohol dosages. Effects that were reported from the study were only assigned to either treated or to non-treated neonates. Thus, although different dosages (3000 - 7800 m**g**/kg bw) had been investigated, there is no indication any for dose response/dependency. This further suggests that acute toxicity testing was driving the study.

Dermal studies are not available.

Conclusion

In a study with Sprague-Dawley rats (Nelson et al., 1988; 1990; 1996) propan-1-ol did not induce detectable developmental effects in the conceptuses of dams exposed to 3500 ppm (8730 mg/m^3) during the whole period of gestation. At maternally toxic concentrations of 7000 (17460 mg/m^3) and 10000 ppm (24940 mg/m^3) , embryotoxic, fetotoxic and teratogenic effects were observed. Based on the findings of reduced fetal body weights and higher incidences of rudimentary cervical ribs at gestational exposures of 7000 ppm, a NOAEC/developmental toxicity of 3500 ppm is established from this study. With the assumption for rats of a respiratory rate of 0.8 L/min/kg, the effective concentrations (embryonic death, fetal growth retardation, skeletal and visceral malformations) of 7000 and 10000 ppm for 7 h/day can be converted to an oral uptake of approximately 5800 mg/kg bw/day, respectively, 8300 mg/kg bw/day. Thus, the effects revealed from exposures to very high inhalatory concentrations of propan-1-ol are not considered appropriate to justify classification and labeling as toxic to reproduction.

4.1.3 Risk characterisation

4.1.3.1 General aspects

Propan-1-ol may be incorporated to human body by oral, dermal and inhalative route.

There are no data on the toxicokinetics of propan-1-ol concerning exposure by inhalation. Based on comparison with data on other short-chain alcohols retention after inhalation may be approximately 40-50%. From the available data it can be concluded that dermal absorption occurs. Considering the physicochemical properties (molecular weight 60 g/mol, complete water solubility and a log Pow of 0.34) absorption through the skin can be assumed. The compound is readily absorbed from the GI tract (about 80%). Propan-1-ol is readily metabolized via its aldehyde to propionic acid which can then be converted by a number of pathways (e.g., the citrate cycle). Its metabolism may be retarded by already-present ethanol.

Taking into account the indicated high uncertainties in the inhalatory absorption figure and the described differences for ethanol in inhalation absorption in humans ranging from 30-76% an inhalation absorption rate of 75% is proposed as a reasonable worse case assumption for risk characterisation purposes. Under worst case assumptions 100% absorption through dermal and oral exposure route, respectively, was taken performing calculations in risk characterisation.

The oral LD50 ranges from 1870, 6500 to 8000 mg/kg bw in the rat. For rabbits this value is 2823 mg/kg bw and for the mouse 5467 mg/kg. bw It is concluded that these data do not warrant a classification labelling for acute oral toxicity. None of the tests were conducted according to OECD Guidelines.

The value of 42000 mg/m³ (resp. 16800 ppm) after a 4-hour exposure is considered as an approximate estimate of a LC50 value (study according to OECD TG 403). In two inhalation hazard tests there were 1/12 or 0/12 deaths after an exposure of up to 8 hours to a substance concentration of 47000 mg/m^3. These findings do not warrant a classification and labelling for acute inhalation toxicity.

The dermal LD50 value is 4052 mg/kg bw for the rabbit. There is no need of classification and labelling for acute dermal toxicity. This test was not conducted according to OECD.

A case of acute oral poisoning in one women suggests that the lethal dose may be in the range of 4.6 - 5.8 g/kg bw. Though the authors concluded that death was caused by the ingestion of propanol it may be possible that other unknown components in the hair lotion that was ingested may have contributed to the death. Based on these data no classification and labelling for acute oral toxicity is warranted.

The odor threshold for normosmics is 28 mg/m^3 (15 ppm) and the pungency threshold is 6500 mg/m^3 (2600 ppm) for anosmics.

A weak skin irritation was observed in rabbits after an exposure of up to 24 hours in two tests that were not conducted according to OECD Guidelines. However, it can be concluded that these findings do not warrant a classification and labelling for skin irritation. The data obtained with humans (patch test with 0.3 ml substance for 10 minutes) did not indicate a skin

irritating effect. In humans no skin irritation was observed on the untreated skin but slight erythema was observed when the treated skin site was immersed in water for 10 minutes at 33°C before exposure to the substance. After frequent exposure of the hands to a 50% preparation for two weeks appearance, intactness and turgor was slightly but significantly affected. Taking also into account the defatting solvent character of propan-1-ol it is assumed that frequent contact can lead to skin dryness or cracking of skin. Consequently, classification with R 66 (Repeated exposure may cause skin dryness or cracking) is proposed.

Instillation of the substance to the eyes of rabbits resulted in serious damage (e.g. strong oedema, strong opacity, vascularization). The data were obtained with two tests that were not conducted according to OECD Guidelines. Nevertheless, a classification as "Xi, Irritant" and labelling as "R 41, Risk of serious damage to eyes" is warranted. This labelling is supported by data on humans. After accidental exposure at the workplace to propanol and other substances vacuolization of the cornea without subsequent scar formation has been reported.

There are several reports on RD50 testing (50% decrease in respiratory rate). A RD50 value of 12704 ppm (31760 mg/m^3) was determined for mice. The substance is considered a weak/very weak sensory irritant. In normal mice or in cannulated mice (trachea) the sensory irritation or the pulmonary irritation were determined after a 30-minute exposure and resulted in a RD50 value of 17967 ppm (44230 mg/m^3) for sensory irritation and a RD50 value of 15593 ppm (38980 mg/m^3) for pulmonary irritation obtained within the first minutes, which remained almost constant during the exposure period. Nielsen and Bakbo (1985) quote two references with RD50 values of 12704 ppm (31760 mg/m^3) and 4770 ppm (11930 mg/m^3) for mice. On the basis of these data the substances is considered a weak sensory irritant and labeling for respiratory irritation is not considered appropriate.

The animal data obtained for skin and eye irritation demonstrate a weak effect on the skin and an irritating effect to the eye. There is no corrosive effect on humans. There is no need to classify the substance as corrosive.

Animal and human data demonstrate that the substance has no skin sensitizing potential. There is only one report of a woman showing allergic skin reactions after patch testing to propan-1-ol and 2-propanol and other substances. There is no need to classify and label the substance for skin sensitizing properties.

There are no data on sensitization by inhalation.

All available information on repeated dose toxicity (oral and inhalation studies) are insufficient with regard to current requirements. Nevertheless, it seemed to be clear from these insufficient data base that major toxic effects resulting from repeated dose treatment comprise narcotic/neurotoxic (alcohol related) reactions in relation to relative high doses/concentrations.

In limited repeated oral investigations by administration of propan-1-ol to rats hepatotoxic effects were observed at high doses. For oral repeated toxicity an overall NOAEL of 3000 mg/kg bw/day will be established. Regarding the inhalative route a NOAEC of 500 ppm (1230 mg/m^3) for irritative effects can be derived from limited studies described. Because of the lack of valid data for propan-1-ol, a scientifically sound risk characterisation on repeated dose toxicity cannot be performed. Formally, there is a conclusion i, i.e. the need for further information and/or testing (90-day rat inhalation study).

Bacterial genotoxicity tests were negative (with the exception of one inconclusive finding); however, the data are not fully reliable due to inadequate reporting. In yeast, aneuploidies were induced only in extremely high doses and only in a small dose range (14 400 to 16 000 µg/ml). In mammalian cell cultures propan-1-ol was negative for chromosomal aberrations (tested with and without S-9 mix), micronuclei (tested only without S-9 mix) and for sister chromatid exchanges (tested with and without S-9 mix). The only in vivo test was inconclusive.There is no relevant concern with respect to mutagenicity. Propan-1-ol should not be classified as a mutagen.

There is no valid carcinogenicity study available. Thus, a risk assessment for carcinogenicity can not be performed. However, taking into account the negative mutagenicity data it is concluded that carcinogenicity should not be an endpoint of concern.

In an inhalation study propan-1-ol did not induce detectable effects on fertility in males and in females of the 3500 ppm (8730 mg/m^3) exposed group and in females of the 7000 ppm (17460 mg/m^3) exposed group. According to the exposure conditions of the study, the statements related to female fertility are limited to the functional aspects of maintaining adequate and sufficient conditions for early extrauterine development of the conceptus and for its nidation. Exposure of male rats to 7000 ppm propan-1-ol for 6 weeks produced reversible infertility. Based on the latter finding the NOAEC/fertility is established at 3500 ppm (8730 mg/m^3). With the assumption for rats of a respiratory rate of 0.8 L/min/kg, the effective concentrations (reversible effects on male fertility) of 7000 ppm for 7h/day can be converted to an oral uptake of approximately 5800 mg/kg bw/day. Thus, the effects on male fertility revealed from this investigation at very high concentrations of propan-1-ol are not considered appropriate to justify classification and labelling as toxic to reproduction.

In an inhalation study with Sprague-Dawley rats propan-1-ol did not induce detectable developmental effects in the conceptuses of dams exposed to 3500 ppm (8730 mg/m³) during the whole period of gestation. At maternally toxic concentrations of 7000 (17460 mg/m³) and 10000 ppm (24940 mg/m³), embryotoxic, fetotoxic and teratogenic effects were observed. Based on the findings of reduced fetal body weights and higher incidences of rudimentary cervical ribs at gestational exposures of 7000 ppm (17460 mg/m³) a NOAEC/developmental toxicity of 3500 ppm (8730 mg/m³) is established from this study. With the assumption for rats of a respiratory rate of 0.8 L/min/kg, the effective concentrations (embryonic death, fetal growth retardation, skeletal and visceral malformations) of 7000 (17460 mg/m³) and 10000 ppm (24940 mg/m³) for 7 h/day can be converted to an oral uptake of approximately 5800 mg/kg bw/day, respectively, 8300 mg/kg bw/day. Thus, the effects revealed from exposures to very high concentrations of propan-1-ol are not considered appropriate to justify classification and labeling as toxic to reproduction.

4.1.3.2 Workers

4.1.3.2.1 Introductory remarks

Propan-1-ol is a colourless liquid with a vapour pressure of 19.4 hPa at 20°C. The substance is easily soluble in water and organic solvents. About 45 % of the handled propan-1-ol is processed to intermediates for the synthesis of further chemicals, approximately 55 % is used as solvent for the formulation of different products. The occupational exposure scenarios have been described and discussed in section 4.1.1.2. Exposure routes to be considered at the workplace are inhalation of propan-1-ol vapour and skin contact with the liquid substance and its formulations. For worker risk assessment the average and short-term exposure levels as reported in tables 4.1.1.2.7 A and B are taken forward to risk characterisation.

The toxicological data have been described and discussed in section 4.1.2. Quantitative human toxicity data are not available, thus risk estimations have to be based on animal data. The experimental threshold levels identified during hazard assessment are taken forward to occupational risk assessment. Respiratory tract irritation and reproductive toxicity at high inhalative exposure levels seem to be the most prominent effects of propan-1-ol.

Systemic availability for different routes of exposure

Most of the toxicity data on propan-1-ol originate either from oral or inhalative studies. Since workers are exposed by inhalation or by skin contact, risk assessment frequently requires route to route transformation. Absorption data for the different routes usually serve as indicator for potential systemic availability. However, absorption data do not in any case reflect the correct relationship between effective doses.

Propan-1-ol is readily and almost completely absorbed via the gastrointestinal tract. For risk characterisation, 100% oral absorption is assumed.

Considering the physicochemical properties (molecular weight 60 g/mol, complete water solubility and a log Pow of 0.34) relevant absorption through the skin can be assumed. For dermal risk assessment, an absorption percentage of 100% is used. From an in vitro experiment with human epidermis a permeation rate of 96 µg/cm^2/h is reported. Thus the maximum amount of propan-1-ol penetrating through 1 cm^2 of skin in 8 hours is approximately 0.8 mg (0.1 mg/cm^2/h x 1 cm^2 x 8 h). This information on dermal penetration rates should be reflected if dermal scenarios are considered to be of concern based on the assumption of 100% dermal absorption.

For propan-1-ol data on absorption by inhalation are not available. Based on structure-activity-relationships the assumption of a 75% absorption by inhalation seems reasonable (see chapter 4.1.2.1).

Occupational exposure and internal body burden

Occupational exposure levels and internal body burdens are listed in table 4.1.3.2.A. For inhalation the table contains shift-average and short-term levels. Available data do not allow for a specification of durations of short-term exposures. For risk characterisation purposes, it might be reasonable to assume a duration of short-term exposures of about 30 minutes.

The internal body burden is based on shift-average levels and on the assumption of 100% dermal absorption and 75% absorption by inhalation.

Table 4.1.3.2.A: Occupational exposure levels and internal body burden

Area of production and use	Inhalation short-term (mg/m³)	Inhalation shift average (mg/m³)	Dermal contact shift average (mg/p/d)	Internal body burden of workers after repeated exposure (mg/p/d)		
				Inhalation (1)	Dermal	Combined
1. Production and further processing	550	4.1	21	31	21	52
2. Formulation of products	132	15.5	420	116	420	536
3. Use of paints	2,204	494	1,625	3,705	1,625	5,330
4a. Use of cleaning formulations (- LEV)	>900	446	1,260	3,345	1,260	4,605
4b. Use of cleaning formulations (+ LEV)	-	72	1,260	540	1,260	1,800
5. Use of printing inks	>900	109	357	818	357	1,175
6. Use of disinfectants	61	44	630	330	630	960

(1) shift average x 10 m^3 x 0.75

Default values for physiological parameters

Body weight, rat	250 g
Body weight, worker	70 kg
Respiratory rate, rat (reduced activity)	0.8 l/min/kg
Respiratory rate, worker (reduced activity)	0.2 l/min/kg
Respiratory volume, worker, 8 hours (reduced activity)	6.7 m³
Respiratory volume, worker, 8 hours (moderate activity)	10 m³

Calculation of MOS values

For toxicological endpoints with relevant quantitative data MOS values are calculated as quotient of experimental NOAEL/C from animal studies and workplace exposure assessments. If the route of application in animal studies is different from the actual occupational exposure the dose units of the experimental data have to be adapted previously to MOS calculation. For this procedure the physiological default values from above are used to modify the effects data. As result a socalled “starting point” is identified.

MOS values for inhalative and dermal route are considered separately. The combined MOS-value is calculated as quotient of the internal NAEL (i.e. the external NOAEL multiplied by the percentage of absorption) and total internal body burden. If different NOAELs for different application routes are available for a certain endpoint, as e.g. in case of acute toxicity, the lowest value is used for the assessment of the combined risks if it is not possible to identify a value which is most relevant under toxicological aspects. With respect to the

possible outcome of an assessment for combined risks, interest focuses on scenarios with conclusion ii at both exposure routes. Based on theoretical considerations combined exposure generally will not increase the most critical route-specific risk component more than twice. Against this background it is recognized, that combined risks only rarely will determine concern.

Evaluation of MOS values

Risk assessment based on MOS values implies the identification of a minimal MOS as criterion for deciding between conclusion ii and iii. In order to obtain consistent results for different chemicals, substance-specific assessment factors, which may vary depending on data availability and the specific toxicological endpoint to be evaluated are identified. Scientifically based adjustment factors describe the extrapolation of animal data to the worker population. The uncertainties in the specific calculations are weighed by expert judgement and expressed as an additional "uncertainty factor". The value of the minimal MOS results from the multiplicative combination of the different assessment factors.

If the MOS value for a certain exposure scenario is below the minimal MOS for a specific endpoint, the corresponding risk situation is considered to be of concern. A MOS value higher than the minimal MOS indicates no concern.

In a parallel procedure, which gives identical but more direct results, the toxicological starting point carried forward to risk characterisation may be divided by the endpoint-specific assessment factors. As a result, an exposure level is identified which, when compared directly with the occupational exposure levels, may serve as trigger for decisions. In the context of this risk assessment report it will be called the "critical exposure level". Concern will be expressed for scenarios above this trigger value.

Interspecies extrapolation

For propan-1-ol there is no reason to suggest substance-specific susceptibility differences among species. For the purpose of occupational risk assessment, scaling on the basis of metabolic rate is used as default assumption for interspecies extrapolation. For oral or dermal data the concept of metabolic rate scaling results in lower effective dose levels for humans compared to experimental animals. For mice the scaling factor is 7, for rats 4, for rabbits 2.2 (for calculation see NO_NL, 1999).The scaling factor is calculated by the formula $(BW_{human}/BW_{animal})^{0.25}$. According to the revised TGD, an additional default factor of 2.5 for remaining interspecies differences shall be used.

For inhalation exposure, the principle of metabolic rate scaling implies that a specific inhalation exposure level (in mg/m^3) is toxicologically equivalent in rats and humans. However, care has to be taken to rely the extrapolation between species on directly comparable conditions: under study conditions rats are thought to be at a state of reduced activity; the according human breathing volume in 8 hours is 6.7 m^3 (0.2 l/min/kg x 60min/h x 8h x 70 kg). Workers, however, are assumed to breathe 10 m^3 during a normal working day under conditions of light to moderate activity. Thus for workers the amount of substance inhaled must be spread over a 1.5 times higher breathing volume. Maintaining toxicological equivalence means, that compared to the experimental levels the according occupational air concentrations will be 1.5 times lower.

Intraspecies differences

Humans differ in sensitivity due to biological factors. The actual risks for a single person may either be less or more pronounced than estimated for the average human. It is recognised that in order to cover the most sensitive person a very high default assessment factor would be required.

Based on an evaluation of empirical data by Schneider et al. (2004) it is anticipated that a factor of 5 generally will be sufficient to protect the major part of the worker population (about 95%). Using a lower factor of 3, for instance, would be protective for about 90% of the population whereas a factor of 10 would include 99% of the population. The empirical data do not allow to decide, if a lower factor would be sufficient for certain toxicological effects, like for instance local effects in the airways. In the absence of further specific information a default intraspecies variation factor for local effects is not defined.

For propan-1-ol the chosen default factor of 5 for intraspecies differences might not cover the susceptability of people with a low activity of aldehyde dehydrogenase (polymorphism).

Duration adjustment

From substance-specific data for various chemicals it is known, that the duration of a study may significantly influence the NOAEL. Longer study duration frequently implies a lower NOAEL. Based on empirically determined average values, duration adjustment for systemic effects for subacute to chronic exposures uses the default factor of 6; duration adjustment for subchronic to chronic exposure is accounted for with a factor of 2 (Kalberlah and Schneider, 1998).

For substances causing respiratory tract effects by inhalation a separate evaluation of empirical data was performed. Duration adjustment factors identified for the local effects are comparable to those for systemic effects (Kalberlah, F. et al., 1999).

Since there is no better information available the values cited above will be used for duration adjustment of repeated dose toxicity data on propan-1-ol concerning systemic and local effects.

Uncertainty considerations

The default adjustment factors outlined above are based upon evaluation of literature data for different chemicals. From a statistical point of view the individual parameters have to be understood as point estimates belonging to probability density distributions. Each factor is taken as geometric mean (point near the maximum) from its density function. The multiplicative combination of all factors is therefore supposed to result in a central tendency estimate. It addresses a likely situation for that percentile of the population reflected by the intraspecies factor.

To complete the assessment, the uncertainty included in the procedure outlined above should be addressed and, if necessary, used to modify the minimal MOS in terms of precaution. On that purpose several aspects should be taken into account, which by their nature are not easy to quantify. Examples are the reliability of the data base, the variability in assessment factors,

the different steps necessary to bridge data gaps, the biological relevance of the observed effects.

4.1.3.2.2 Occupational risk assessment

Acute toxicity

Systemic effects, inhalation

In two inhalation hazard tests (rats, 47,000 mg/m³ for up to 8 hours) a death rate of 1/12 or 0/12 was observed. An approximate LC50 (4h) in rats is reported to be 42,000 mg/m^3. At 12,960 mg/m^3 signs of toxicity were ocular and nasal irritation and hyperactivity, no mortality occurred. As starting point for MOS calculation the air concentration of 12,960 mg/m^3 is chosen, a dose without severe acute effects in rats. This starting point for acute toxicity is supported by the information, that there were no clinical effects in exposed dams at a concentration of 24,940 mg/m³ (see teratogenicity study). Evaluation of MOS values has to account for the following aspects: (a) for intraspecies variation in humans a factor of 5 is used, (b) study duration was 4 hours compared to occupational exposure of 8 hours, (c) physiological differences between humans at rest and workers account for a factor of 1.5. Based on the overall information on acute effects at very high concentrations of propan-1-ol a further factor for possible remaining differences is not used. Altogether the minimal MOS calculates to 15 (5 x 8/4 x 1.5). The critical exposure level is identified as 864 mg/m^3 (12,960 mg/m^3 / 15).

The highest shift average value for inhalation is reported as 494 mg/m^3 for use of paints (scenario 3), the according MOS value calculates to 26 (12,960 / 494) which does not give reason for concern (table 4.1.3.2.B). In this approach it has been assumed that the total daily dose is responsible for acute toxicity. Except for scenario 1 the total dose resulting from the average exposure levels is higher than the total dose which is calculated based on available short-term levels. For scenario 1, a short-term level of 550 mg/m³ (30 minutes) is equivalent to an average level of 34 mg/m³ (8 hours), which is higher than the reported average level, but clearly lower than the estimated critical exposure level of 864 mg/m³.

Risk assessment for acute inhalation toxicity is based on the starting point of 12,960 mg/m^3, an air-borne concentration with signs of toxicity. For repeated dose toxicity there is the need for further testing (90-day rat inhalation study). Depending on the study design of the 90-day rat inhalation study, especially the choice of the highest concentration tested, the result of the required study might influence the outcome of this risk assessment for acute effects by inhalation.

Conclusion: ii

Table 4.1.3.2.B: Acute toxicity, systemic effects

	Inhalation			Dermal			Combined		
Starting point for MOS calculation	12,960 mg/m^3			283,640 mg/p/d			131,040 mg/p/d		
Minimal MOS	15			33			20		
Critical exposure level	864 mg/m^3			8,600 mg/p/d			6,552 mg/p/d		
	Exposure (mg/m^3)	MOS	Conclusion	Exposure (mg/p/d)	MOS	Conclusion	Internal body burden (mg/p/d)	MOS	Conclusion
1. Production and further processing	4.1	3,160	ii	21	13,507	ii	52	2,520	ii
2. Formulation of products	15.5	836	ii	420	675	ii	536	244	ii
3. Use of paints	494	26	ii	1,625	175	ii	5,330	25	ii
4a. Use of cleaning formulations (- LEV)	446	29	ii	1,260	225	ii	4,605	28	ii
4b. Use of cleaning formulations (+ LEV)	72	180	ii	1,260	225	ii	1,800	73	ii
5. Use of printing inks	109	119	ii	357	795	ii	1,175	112	ii
6. Use of disinfectants	44	295	ii	630	450	ii	960	137	ii

Systemic effects, dermal contact

The LD50 in rabbits is reported to be 4,052 mg/kg, with occlusive exposure for 24 h. A dermal exposure level without lethality has not been reported. No further information is given. Compared to the oral LD50 for rabbits of 2,823 mg/kg the dermal value seems to be plausible and relevant for risk assessment. It has to be recognized, that according to this data the rabbit skin seems to be no essential permeation barrier for propan-1-ol. As starting point for MOS calculation the human dose corresponding to the dermal LD50 in rabbits is calculated to 283,640 mg/person (4,052 mg/kg x 70 kg).

Evaluation of MOS values has to account for the following aspects: (a) metabolic rate scaling from rabbits to humans reveals a factor of 2.2, (b) in this case assessment starts from the LD50, from a comparison with inhalation data it is assumed, that at 3 times lower doses acute effects might be slight, (c) for intraspecies variation in humans a factor of 5 is used, (d) a further factor for remaining differences and uncertainties is not considered necessary

especially because of the rather strict experimental exposure conditions (occlusive exposure for 24 hours). Altogether the minimal MOS calculates to 33 (2.2 x 3 x 5). The critical exposure level is identified as 8,595 mg/person (283,640 mg/person / 33).

The highest dermal exposure level is reported to be 1,625 mg/person for use of paints (scenario 3). The according MOS value calculates to 175 (283,640 / 1,625) which does not give reason for concern (table 4.1.3.2.B).

Conclusion: ii

Systemic effects, combined exposure

Risk assessment concerning combined exposure will be based on the experimental air concentration of 12,960 mg/m^3 in the rat as outlined under *acute toxicity, inhalation*. This air concentration is converted in an equivalent internal dose scaled per bodyweight, taking into account the amount inhaled by the rat during 4 hours of exposure and an inhalative absorption rate of 75 %. As starting point for combined MOS calculation the equivalent internal dose for rats is identified as 1,872 mg/kg (13.0 mg/l x 0.8 l/min/kg x 60 min/h x 4 h x 0.75), the according dose for humans is 131,040 mg/person (1,872 mg/kg x 70 kg/person).

Evaluation of the MOS values has to account for the following aspects: (a) assessment starts from a rat dose scaled per bodyweight, thus for species extrapolation a factor of 4 is introduced, (b) for intraspecies variation in humans a factor of 5 is used. Altogether the minimal MOS calculates to 20 (4 x 5). The critical internal body burden is identified as 6,552 mg/person (131,040 mg/person / 20).

The highest combined exposure level is reported to 5,330 mg/person for the use of paints (scenario 3, table 4.1.3.2.B). The according MOS value calculates to 25 (131,040 / 5,330), which is above the minimal MOS but might be addressed as a borderline situation. However no concern will be expressed. No further scenario is in the concern region.

Risk assessment for combined exposure is based on the starting point of 12,960 mg/m^3, an air-borne concentration with signs of toxicity. For repeated dose toxicity there is the need for further testing (90-day rat inhalation study). Depending on the study design of the 90-day rat inhalation study, especially the choice of the highest concentration tested, the result of the required study might influence the outcome of this risk assessment for combined exposure.

Conclusion: ii

Irritation/Corrosivity

Dermal contact

In humans dermal exposure resulted in no or weak reactions. In rabbits weak skin irritation was observed which was not sufficient for classification. There is no concern from dermal irritation at the workplace.

Conclusion: ii

Contact to the eyes

From human and rabbit data propan-1-ol has shown the potential to cause serious damage to the eyes. Eye contact to liquid propan-1-ol critically depends on the proper handling of the substance and the use of eye glasses. Even though suitable personal protective equipment (PPE) usually should be available at the working places in question, unintended contact by non-proper use is considered to represent an incident which may occur in different exposure situations. Therefore a risk from eye irritation principally has to be considered.

On the grounds that control measures exist, which should be able to efficiently minimize exposure, conclusion ii is proposed. However, these control measures must be implemented and complied with to reduce the risk of damage to the eyes.

Conclusion: ii

Inhalation/sensory irritation

For propan-1-ol sensory irritation is reported from animal data, the majority of RD50-values lying in the range of 30,000 mg/m^3. No information on an exposure level without respiratory depression is given. Alarie introduced the air concentration of 0.03 x RD50 as prediction of an exposure level with a minimal or low degree of sensory irritation in humans, which was confirmed by other authors (Alarie 1981, Bos et al 1992, Schaper 1993). The according air concentration for propan-1-ol is estimated to 900 mg/m^3.

900 mg/m^3 is chosen as starting point concerning respiratory depression. For evaluation of the resulting MOS values no further aspects have to be taken into account, an uncertainty factor does not seem necessary. The according minimal MOS is 1, the critical exposure level is 900 mg/m^3. In workers the stinging and burning sensation caused by stimulation of the trigeminus nerve which is closely connected to respiratory depression generally is perceived within few minutes after exposure. Thus stimulation of the trigeminus nerve, unlike other effects, seems not to depend significantly on exposure duration, the main trigger for effects seems to be the air concentration of the substance. For this approach a focus of MOS calculation therefore lies at short term exposures, however shift average values are included in the assessment too.

Inhalation/other local effects

The RD50 is only used to evaluate effects in the airways, which are caused by stimulation of the trigeminus nerve. Other local effects are not covered by this parameter. Additional information on portal-of-entry effects of propan-1-ol comes from a study with inhalative exposure of rats for 9 exposure days, 6 hours per day. The NOAEC for irritation is recorded to be 1,230 mg/m^3. At 2,460 mg/m^3 slightly swollen periocular tissue and minimal perinasal and periocular encrustations were observed. No data is available which allow to assess whether the irritation threshold after single exposure might be different to that for 9 days of exposure.

The NOAEC (6 h/d) of 1,230 mg/m^3 is used for MOS calculation. This starting point may be directly compared with shift average values. Evaluation of the according MOS values has to account for the following aspects: (a) study duration was 6 hours compared to occupational exposure of 8 hours, (b) physiological differences between humans at rest and workers account for a factor of 1.5. A further adjustment factor for remaining differences and uncertainties is not applied because the reported irritation was slight and because the NOAEC is based on a repeated toxicity study (9 days). The minimal MOS thus calculates to 2 (8/6 x 1.5), the according critical air concentration (8h) is identified as 615 mg/m^3 (1,230 mg/m^3 / 2).

There is no clear guidance for the extrapolation of acutely toxic effects to shorter durations of exposure (e.g. from 6 hours to short-term levels of about 30 minutes). Available evidence suggests that there is a relevant influence of the duration of exposure on effect size for cytotoxic irritants as well. With reference to the NRC “standing operation procedures for developing acute exposure guideline levels for hazardous chemicals” (www.epa.gov/oppt/aegl/sop.htm) the relationship C^n x t = k with n from 1 to 3 is used to extrapolate acute toxicity of different types of chemicals (including irritants) for exposure durations between about 30 minutes and 8 hours. To be at the side of caution a value of n = 3 is used when extrapolating from longer to shorter durations. Using the latter approach, and directly starting with a NOAEC of 1,230 mg/m³ for 6 hours, a NOAEC of 2,800 mg/m³ for 30 minutes is calculated. Available short-term levels are not higher than the latter NOAEC; no further concern for available short-term exposure levels is indicated.

Risk assessment for acute respiratory tract irritation is based on an NOAEC of 1,230 mg/m^3 (6 h/d, 9 days of exposure). For repeated dose toxicity there is the need for further testing (90-day rat inhalation study). The result of the required study might influence the outcome of this risk assessment for acute respiratory tract irritation.

Table 4.1.3.2.C: Summary of local effects by inhalation

	Acute exposure, respiratory depression	Acute exposure, other local effects (8 hours)	Repeated exposure, local effects
Starting point for MOS calculation	900 mg/m^3	1,230 mg/m^3	1,230 mg/m^3

Minimal MOS	1						2			12		
Critical exposure level	900 mg/m^3						615 mg/m^3			103 mg/m^3		
	short term			shift average			shift average			shift average		
	Exposure (mg/m^3)	MOS	Conclusion	Exposure (mg/m^3)	MOS	Conclusion	Exposure (mg/m^3)	MOS	Conclusion	Exposure (mg/m^3)	MOS	Conclusion
1. Production and further processing	550	1.6	ii	4.1	220	ii	4.1	300	ii	4.1	300	ii
2. Formulation of products	132	6.8	ii	15.5	58	ii	15.5	79	ii	15.5	79	ii
3. Use of paints	2,204	0.4	iii	494	1.8	ii	494	2.5	ii	494	2.5	iii
4a. Use of cleaning formulations (- LEV)	>900	<1	iii	446	2	ii	446	2.8	ii	446	2.8	iii
4b. Use of cleaning formulations (+ LEV)	-	-	-	72	13	ii	72	17	ii	72	17	ii
5. Use of printing inks	>900	<1	iii	109	8	ii	109	11	ii	109	11	iii
6. Use of disinfectants	61	14.8	ii	44	20	ii	44	28	ii	44	28	ii

For respiratory depression concern is indicated for scenarios 4a and 5. This conclusion is based on short-term levels, because this type of effect has been basically proven to be dependent on exposure concentration. Risk assessment for other local effects (8 hours) is based on comparison of available toxicity data with shift-average levels. No further concern is derived. Based on the NRC approach, a NAEC for a 30-minutes exposure is calculated and compared with short-term exposure levels. Again, no further concern is derived.

Conclusion: iii

Sensitisation

Dermal contact

Animal and human data demonstrate that propan-1-ol has no skin sensitising properties. There is no concern from skin sensitisation at the workplace.

Conclusion: ii

Inhalation

No information on respiratory sensitisation is available. Propan-1-ol is not suspected to be a potent respiratory sensitiser in humans according to the fact that during all the years of use no notice of specific case reports has been given. There is no concern from respiratory sensitisation at the workplace.

Conclusion: ii

Repeated dose toxicity

Local effects, inhalation

From a study of limited validity with inhalative exposure of rats for 9 exposure days, 6 hours per day, a NOAEC for irritation is recorded to be 1,230 mg/m^3 (see *Irritation, inhalation*). This value is used as starting point for MOS calculation. It is recognized that the quality of the study design is rather limited. Thus, risk characterisation for local effects by repeated inhalation contains substantial uncertainties.

Factors to be additionally taken into account during MOS evaluation are: (i) study duration was 6 hours compared to occupational exposure of 8 hours, (ii) physiological differences between humans at rest and workers account for a factor of 1.5, (iii) for duration adjustment of short-term data to chronic exposure situations a factor of 6 will be used. Altogether the minimal MOS calculates to 12 (8/6 x 1.5 x 6). The critical air concentration at the workplace is identified as 103 mg/m^3 (1,230 mg/m^3 / 12).

At the moment there is no clear guidance as to an intraspecies variation factor for local effects. Additional implementation of an intraspecies variation factor of 5, which is normally used for systemic effects, would result in a critical exposure level of about 20 mg/m³. Against the background of many national current OELs of about 500 mg/m³ it is not considered reasonable to use a minimal MOS that is essentially greater than calculated.

The highest shift average value for inhalation is reported as 494 mg/m^3 for use of paints (scenario 3). The according MOS value calculates to 2.5 which is clearly below the minimal MOS of 12. In addition, concern is expressed for scenarios 4a (use of cleaning formulations without LEV) and for scenario 5 (use of printing inks).

Risk assessment for respiratory tract irritation following repeated exposure is based on an NOAEC of 1,230 mg/m^3 (6 h/d, 9 days of exposure). For repeated dose toxicity (systemic effects) there is the need for further testing (90-day rat inhalation study). The result of the required study might influence the outcome of this risk assessment for chronic respiratory tract irritation.

Conclusion: iii

Local effects, dermal

Propan-1-ol is not classified as a skin irritant. Human data do not indicate acute skin irritation at untreated forearms. Following repeated dermal exposure under rigorous exposure conditions (rubbing of up to 5 ml of a 50% preparation into the hands, 15 times a day, 5 days a week, for two weeks) the appearance, intactness and turgor of the skin was slightly but significantly affected. With reference to table 4.1.1.2.7B dermal contact to propan-1-ol preparations is described to occur daily. Contact levels are assumed to be intermittent. Dermal

exposure is estimated to be up to 1,625 mg/person/day. For these estimated exposure conditions (EASE) slight chronic effects to the human skin cannot be excluded. Conclusion iii is drawn for all occupational exposure scenarios except for scenario 1 (use of gloves in the production scenario).

Conclusion: iii

Systemic effects by inhalation, dermal contact and combined exposure

Inhalative data concerning this endpoint are not available. From oral studies for up to four months in rats an overall NOAEL of 3,000 mg/kg/day is reported. All available information is insufficient with regard to current requirements. The chosen oral NOAEL refers to a drinking water study without any histopathological investigations except for the liver. Other toxicological studies indicate that liver effects occur at higher doses. It should be recognized that lethal effects (LD50) have been reported for the range of 6,500 to 8,000 mg/kg for adult rats. For very young rats an LD50 of about 2,000 mg/kg was observed.

Based on these repeated dose toxicity data which are of rather limited reliability, a scientifically sound health risk assessment cannot be derived. However, a provisional risk assessment nevertheless might be helpful to give some guidance as to the necessity and priority of getting robust toxicological data for worker risk assessment. The following provisional risk assessment is based on the assumption of an oral NOAEL of 3,000 mg/kg/day and an overall adjustment factor that to some degree takes account of the rather limited reliability of the data.

Because of the assumption of 100% oral absorption the internal NAEL is chosen to be 3,000 mg/kg/day and thus is identical to the oral NOAEL. The internal starting point, which is used for assessment of combined exposure, is 210,000 mg/person/day (3,000 mg/kg/day x 70 kg).

For dermal risk assessment, a 100% dermal absorption is used. Therefore, the external starting point for exposure to the skin is 210,000 mg/person/day as well.

For inhalation, a 75% absorption percentage is taken forward. The starting point for repeated dose toxicity by inhalation is calculated as 28,000 mg/m^3 (210,000 mg/person / 10 m^3 / 0.75).

For evaluation of MOS values the following aspects have to be considered: (a) metabolic rate scaling from rats to humans yields a factor of 4 , (b) for remaining interspecies differences an additional default factor of 2.5 is used, (c) intraspecies variation in humans is accounted for by a factor of 5, (d) duration adjustment from a subchronic to a chronic study design should be accounted for with a factor of 2, (e) for uncertainties as to the insufficient study design an additional adjustment factor is considered necessary; without specific justification at least a factor of 2 should be used.

Altogether the minimal MOS for systemic effects after repeated exposure (dermal contact and combined exposure) calculates to 200 (4 x 2.5 x 5 x 2 x 2). The according critical exposure level is 1,050 mg/person/d (210,000 / 200). For inhalation exposure, the corresponding critical inhalation exposure results in 140 mg/m³ (28,000 / 200).

Based on this orienting risk assessment, for specific occupational scenarios there might be concern for inhalation exposure and concern for dermal contact. This provisional risk assessment indicates that health risks for workers cannot be excluded.

However, in order to sufficiently justify possible risk management measures further toxicological information is needed. Overall, results from a repeated inhalation toxicity study (e.g. 90-day) are particularly suitable for a relevant occupational risk assessment.

Conclusion: i

Table 4.1.3.2.D: Repeated dose toxicity, systemic effects (provisional assessment*)

	Inhalation			Dermal			Combined		
Starting point for MOS calculation	28,000 mg/m^3			210,000 mg/p/d			210,000 mg/p/d		
Minimal MOS	200			200			200		
Critical exposure level	140 mg/m^3			1,050 mg/p/d			1,050 mg/p/d		
	Exposure (mg/m^3)	MOS	Conclusion	Exposure (mg/p/d)	MOS	Conclusion	Internal body burden (mg/p/d)	MOS	Conclusion
1. Production and further processing	4.1	6,830	(ii)	21	10,000	(ii)	52	4,038	(ii)
2. Formulation of products	15.5	1,806	(ii)	420	500	(ii)	536	392	(ii)
3. Use of paints	494	57	(iii)	1,625	129	(iii)	5,330	39	(iii)**
4a. Use of cleaning formulations (- LEV)	446	63	(iii)	1,260	167	(iii)	4,605	46	(iii)**
4b. Use of cleaning formulations (+ LEV)	72	389	(ii)	1,260	167	(iii)	1,800	117	(iii)**
5. Use of printing inks	109	257	(ii)	357	588	(ii)	1,175	179	(iii)
6. Use of disinfectants	44	636	(ii)	630	333	(ii)	960	219	(ii)

* Table 4.1.3.2.D contains the results of the provisional risk assessment. Because this provisional assessment is not considered sufficiently valid, but is only used to give some indication of possible risks, there is no formal conclusion ii oder iii. For repeated dose toxicity (systemic effects) conclusion i is drawn (see main text)

** conclusion iii already results from dermal and/or inhalation exposure, therefore no specific concern for the combined exposure scenario is indicated

Mutagenicity

Available results from mutagenicity testing do suggest that propan-1-ol is not to be considered a mutagen. For all occupational exposure scenarios conclusion ii is drawn.

Conclusion: ii

Carcinogenicity

There is no valid carcinogenicity study for propan-1-ol. Thus, a specific risk assessment for carcinogenicity can not be performed. However, taking into account the negative mutagenicity data, for carcinogenicity conclusion ii is drawn for all occupational exposure scenarios.

Conclusion: ii

Reproductive toxicity

Fertility impairment, inhalation

In an inhalation study, focusing on behavioral teratogenicity (6 weeks, 7h per day), 14 out of 16 male rats showed infertility at air concentrations of 17,469 mg/m^3. At 8,730 mg/m^3 no fertility impairment was detected. No adverse effects on reproductive capacity was observed for females. The study is of limited validity, there is no information on maternal toxicity. However, its results will be used for preliminary risk assessment since no other information is available. As starting point for MOS calculation the NOAEC for male rats of 8,730 mg/m^3 is chosen.

Evaluation of the MOS values has to account for various aspects: (a) there is the possibility that male rat infertility is caused by toxic effects to the testis; in rats LOAELs for testis histopathology frequently are lower than the corresponding LOAELs for infertility; based on the publication by Mangelsdorf et al. (2003) a factor of 3 is used, (b) for remaining interspecies differences an additional default factor of 2.5 is used, (c) for intraspecies variation in humans a factor of 5 is used, (d) study duration was 7 hours compared to occupational exposure of 8 hours, (e) physiological differences between humans at rest and workers account for a factor of 1.5. Altogether the minimal MOS calculates to 65 (3 x 2.5 x 5 x 8/7 x 1.5). Because this assessment of male fertility impairment is already based on possible reproductive organ damage, which is considered more sensitive than infertility parameters, no further adjustment factor is used. The critical air concentration at the workplace is identified as 134 mg/m^3 (8,730 mg/m^3 / 65).

The highest shift average values for inhalation are reported as 494 mg/m^3 for the use of paints (scenario 3) and 446 mg/m^3 for use of cleaning formulations without LEV (scenario 4a). The according MOS values calculate to 18 (8,730 / 494) and 20 (8,730 / 446). Because of a minimal MOS of 65, both scenarios are considered to be of concern (table 4.1.3.2.E).

Risk assessment for male fertility impairment is partly based on the assumption of testis toxicity (see first adjustment factor). For repeated dose toxicity there is the need for further

testing (90-day rat inhalation study). The result of the required study (testis toxicity?) might influence the outcome of this risk assessment for male fertility impairment.

Conclusion: iii

Fertility impairment, dermal contact

Risk assessment concerning dermal exposure will be based on the experimental no effect concentration of 8,730 mg/m^3 as outlined under *fertility impairment, inhalation.*

This air concentration is converted in an equivalent dose scaled per bodyweight for the rat, taking into account the amount inhaled by the rat during 7 hours of exposure and an inhalative absorption rate of 75 %. As starting point for MOS calculation a dose of 2,192 mg/kg (8.7 mg/l x 0.8 l/min/kg x 60 min/h x 7 h x 0.75) is identified as no effect level. The according dose for humans calculates to 153,440 mg/person (2,192 mg/kg x 70 kg/person), which likewise resembles the relevant internal dose for combined assessment, assuming 100 % dermal absorption.

Evaluation of the MOS values has to account for the following aspects: (a) for species extrapolation a scaling factor of 4 is introduced, (b) for remaining interspecies differences an additional default factor of 2.5 is used, (c) there is the possibility that male rat infertility is caused by toxic effects to the testis; in rats LOAELs for testis histopathology frequently are lower than the corresponding LOAELs for infertility; based on the publication by Mangelsdorf et al. a factor of 3 is used, (d) for intraspecies variation in humans a factor of 5 is used. Because this assessment of male fertility impairment is already based on possible reproductive organ damage, which is considered more sensitive than infertility parameters, no further adjustment factor is used. Altogether the minimal MOS calculates to 150 (4 x 2.5 x 3 x 5). The critical exposure level is identified as 1,023 mg/person/day (153,440 / 150).

Concern is indicated for scenario 3 (use of paints) and for scenarios 4a/4b (use of cleaning formulations). This risk characterisation is based on the assumption of a 100% dermal absorption.

Risk assessment for male fertility impairment is partly based on the assumption of testis toxicity (see first adjustment factor). For repeated dose toxicity there is the need for further testing (90-day rat inhalation study). The result of the required study (testis toxicity?) might influence the outcome of this risk assessment for male fertility impairment.

Conclusion: iii

Table 4.1.3.2.E: Fertility impairment

	Inhalation			Dermal			Combined		
Starting point for MOS calculation	8,730 mg/m^3			153,440 mg/p/d			153,440 mg/p/d		
Minimal MOS	65			150			150		
Critical exposure level	134 mg/m^3			1,023 mg/p/d			1,023 mg/p/d		
	Exposure (mg/m^3)	MOS	Conclusion	Exposure (mg/p/d)	MOS	Conclusion	Internal body burden (mg/p/d)	MOS	Conclusion
1. Production and further processing	4.1	2,130	ii	21	7,306	ii	52	2,950	ii
2. Formulation of products	15.5	560	ii	420	365	ii	536	286	ii
3. Use of paints	494	18	iii	1,625	94	iii	5,330	29	iii*
4a. Use of cleaning formulations (- LEV)	446	20	iii	1,260	121	iii	4,605	33	iii*
4b. Use of cleaning formulations (+ LEV)	72	121	ii	1,260	121	iii	1,800	85	iii*
5. Use of printing inks	109	80	ii	357	430	ii	1,175	130	iii
6. Use of disinfectants	44	198	ii	630	244	ii	960	160	ii

* conclusion iii already results from inhalative or dermal exposure, therefore no specific concern for the combined exposure scenario is indicated

Fertility impairment, combined exposure

For combined exposure the essential parameters for risk assessment and MOS calculation are the same as identified according to the procedure outlined under *fertility impairment, dermal contact.* For details see table 4.1.3.2.E.

For scenario 3 and 4a concern was already drawn for inhalation exposure. Consequently, these occupational scenarios for combined exposure are of concern as well. Additionally, concern is indicated for scenario 4b (mainly based on dermal exposure). There is specific concern for combined exposure for scenario 5 (use of printing inks).

Conclusion: iii

Developmental toxicity

Inhalation

In an inhalation study in rats for 7 hours per day, propan-1-ol has proven to be embryotoxic and teratogenic at concentrations which did not cause marked maternal toxicity. At 17,460 mg/m^3 embryos showed body weight reduction and sceletal malformations, at 24,940 mg/m^3

57 % of the implants were resorbed and one third of the remaining embryos showed cardiovascular, urinary and other malformations besides the skeletal defects. The embryonal NOAEC was 8,730 mg/m^3.

Starting point for MOS calculation will be the embryonal NOAEC of 8,730 mg/m^3. This NOAEC is identical to the NOAEC for fertility impairment.

Evaluation of MOS values has to account for various aspects: (a) for remaining interspecies differences a default factor of 2.5 is used, (b) for intraspecies variation in humans a factor of 5 is used, (c) study duration was 7 hours compared to occupational exposure of 8 hours, (d) physiological differences between humans at rest and workers account for a factor of 1.5. Altogether the minimal MOS for developmental toxicity calculates to 21 (2.5 x 5 x 8/7 x 1.5). The critical air concentration at the workplace is identified as 416 mg/m^3 (8,730 mg/m^3 / 21).

The highest shift average values for inhalation are reported as 494 mg/m^3 for the use of paints (scenario 3) and 446 mg/m^3 for use of cleaning formulations without LEV (scenario 4a). The according MOS values calculate to 18 (8,730 / 494) and 20 (8,730 / 446). With a minimal MOS of 21, both scenarios reach borderline. Because of remaining uncertainties, (e.g. a steep dose-response curve between NOAEC and a significant irreversible increase of malformations, both scenarios are considered to be of concern (see table 4.1.3.2.F).

Conclusion: iii

Dermal contact

Risk assessment concerning dermal exposure will be based on the experimental no effect concentration of 8,730 mg/m^3 as outlined under *developmental toxicity, inhalation.*

This air concentration is converted in an equivalent dose scaled per bodyweight for the rat, taking into account the amount inhaled by the rat during 7 hours of exposure and an inhalative absorption rate of 75 %. As starting point for MOS calculation a dose of 2,192 mg/kg (8.7 mg/l x 0.8 l/min/kg x 60 min/h x 7 h x 0.75) is identified as no effect level. The according dose for humans calculates to 153,440 mg/person (2,192 mg/kg x 70 kg/person), which likewise resembles the relevant internal dose for combined assessment, assuming 100 % dermal absorption.

Evaluation of the MOS values has to account for the following aspects: (a) for species extrapolation a scaling factor of 4 is introduced, (b) for remaining interspecies differences an additional default factor of 2.5 is used, (c) for intraspecies variation in humans a factor of 5 is used. Because this assessment of male fertility impairment is already based on possible reproductive organ damage, which is considered more sensitive than infertility parameters, no further adjustment factor is used. Altogether the minimal MOS calculates to 50 (4 x 2.5 x 5). The critical exposure level is identified as 3,069 mg/person/day (153,440 / 50).

There is no concern indicated for the described scenarios with respect of developmental toxicity after dermal conctact.

Conclusion: ii

Combined exposure

For combined exposure the essential parameters for risk assessment and MOS calculation are the same as identified according to the procedure outlined under *developmental toxicity, dermal contact.* For details see table 4.1.3.2.F.

For scenario 3 and 4a concern was already drawn for inhalation exposure. Consequently, these occupational scenarios for combined exposure are of concern as well.

Conclusion: iii

Table 4.1.3.2.F: Developmental toxicity

	Inhalation			Dermal			Combined		
Starting point for MOS calculation	8,730 mg/m^3			153,440 mg/p/d			153,440 mg/p/d		
Minimal MOS	21			50			50		
Critical exposure level	416 mg/m^3			3,069 mg/p/d			3,069 mg/p/d		
	Exposure (mg/m^3)	MOS	Conclusion	Exposure (mg/p/d)	MOS	Conclusion	Internal body burden (mg/p/d)	MOS	Conclusion
1. Production and further processing	4.1	2,130	ii	21	7,306	ii	52	2,950	ii
2. Formulation of products	15.5	560	ii	420	365	ii	536	286	ii
3. Use of paints	494	18	iii	1,625	94	ii	5,330	29	iii*
4a. Use of cleaning formulations (- LEV)	446	20	iii	1,260	121	ii	4,605	33	iii*
4b. Use of cleaning formulations (+ LEV)	72	121	ii	1,260	121	ii	1,800	85	ii
5. Use of printing inks	109	80	ii	357	430	ii	1,175	130	ii
6. Use of disinfectants	44	198	ii	630	244	ii	960	160	ii

*conclusion iii already results from inhalative exposure, therefore no specific concern for the combined exposure scenario is indicated

4.1.3.2.3 Summary on occupational risk assessment

Occupational risk assessment for propan-1-ol is summarized in table 4.1.3.2.F. This table contains the endpoint-specific overall conclusions.

With reference to the hazard assessment, it has to be recognized, that experimental animal data for repeated dose toxicity are of rather limited reliability. Because of the lack of valid data for propan-1-ol, for workers a scientifically sound risk assessment for repeated dose toxicity cannot be derived. There is the need for further information and/or testing (90-day rat inhalation study).

Propan-1-ol is also notified as an active substance within the scope of the Biocide Directive 98/8/EC. The necessary data on repeated dose toxicity is existing, but is owned by a company who wishes to use it in the framework of other EU regulation (the Biocides Directive). The company is so far not willing to make the study available to support risk assessment in the context of the Existing Substances Regulation, and there are no provisions in the Biocides Directive that would force them to share the data with other companies. The information on repeated dose toxicity is requested for reasons of human health. For the sake of animal protection it is hoped that the companies involved will be able to negotiate and share the data.

Results from a 90-day rat inhalation study, in connection with corresponding dose finding studies, may have an impact on the risk assessment of other toxicological endpoints.

Table 4.1.3.2.F: Endpoint-specific overall conclusions

Toxicological endpoints		General conclusion
Acute toxicity	systemic, inhalation	ii
	systemic, dermal	ii
	systemic, combined	ii
Irritation/ Corrosivity	dermal	ii
	eye	ii
	inhalation, acute	ii
	respiratory depression	iii
Sensitisation	skin	ii
	respiratory	ii
Repeated dose toxicity	local, inhalation	iii
	local, dermal	iii
	systemic, inhalation	**i**
	systemic, dermal	**i**
	systemic, combined	**i**
Mutagenicity		ii
Carcinogenicity		ii
Fertility impairment	inhalation	iii
	dermal	iii
	combined	iii
Developmental toxicity	inhalation	iii
	dermal	ii
	combined	iii

The toxicological endpoints of concern are respiratory depression, respiratory tract irritation by repeated exposure and reprotoxicity (fertility impairment and developmental toxicity). No concern is expressed for eye irritation although risks at the workplace cannot be excluded. Measures applied already because of classification and labelling with Xi, R 41 are judged to be sufficient for risk reduction. Slight effects to the skin following repeated dermal exposure cannot be excluded. For repeated dose toxicity (systemic effects) further toxicological data is needed in order to perform a robust risk assessment for workers.

4.1.3.3 Consumers

Exposure values to be taken for risk characterisation

For acute toxicity the highest amount of exposure should be taken which is 2290 mg/m³ resulting from the scenario for the use of disinfectants.

For chronic inhalation toxicity an exposure value of 30 mg/m³ is proposed to be taken forward to the risk characterisation which resulted from calculations of the aggregated yearly exposure via non-user scenarios to disinfectants, all-purpose cleaners, and kitchen cleaners (cf. table 4.1.1.3.4). This results in an exposure of 5.1 mg/kg bw/day. Dermal exposure of consumers originates mainly from the use of cosmetics, amounting to 2.6 mg/kg bw/d. Additional dermal exposure through disinfectants and kitchen cleaners can be considered negligible for risk characterisation due to >10-fold lower exposure values and in view of the lower use frequency (weekly). Oral exposure by mouth hygiene products may lead to a daily dose of 19.3 mg/kg bw. Taking all paths into consideration a total body burden of 27 mg/kg bw/day may arise under reasonable worst case conditions.

Acute toxicity

LD_{50} values of propanol were determined in oral and dermal acute toxicity studies as 5467 mg/kg bw (oral, mouse) and 4052 mg/kg bw (dermal, rabbit). Expected acute consumer exposures are far below these values, with 2.6 mg/kg bw for the dermal route and 19.3 mg/kg bw via oral uptake. The LC_{50} value after inhalation was determined as 42000 mg/m^3 in rats. At 12960 mg/m^3 signs of toxicity were occular and nasal irritation and hyperactivity, no mortality occurred. The exposure duration in this study was 4 h. The highest acute exposure of consumers from inhalation is estimated to be 2290 mg/m^3. The corresponding scenario (use of disinfectants) foresees an exposure duration of 0.5 h. In view of this short exposure time, the margin of safety is judged to be sufficient.

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

Irritation/Corrosivity

From human and rabbit data propan-1-ol has shown the potential to cause serious damage to the eyes. The concentration of the substance in some final products for consumers is above the concentration limit which leads in these cases to classification and labelling of the preparation with R 41.

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

Sensory irritation

Propan-1-ol shows weak sensory irritation (cf. 4.1.2.3). The majority of RD50-values is in the range of 30,000 mg/m^3. No information on an exposure level without reduced respiratory rate is available. Alarie introduced the air concentration of 0.03 x RD50 as prediction of an exposure level with a minimal or low degree of sensory irritation in humans (cf. 4.1.3.2). The

according air concentration for propan-1-ol is estimated to be 900 mg/m^3 and will be used as starting point for risk characterisation purposes.

Sensory effects are thought to be dependent on the air concentration of a substance. They do not depend on the duration of exposure. The short-term inhalation exposure of consumers due to application of disinfectants, hardener solutions, and wall paper removers (cf. 4.1.1.3) may result in propan-1-ol concentrations of 2290, 1611, and 1520 mg/m^3 which are higher than 900 mg/m^3. Thus, a concern for respiratory depression in relation to a few consumer exposure scenarios is derived.

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

Sensitization

Animal and human data demonstrate that propan-1-ol has no skin sensitising properties. Propan-1-ol is not suspected to be a potent respiratory sensitiser in humans according to the fact that during all the years of use no notice of specific case reports has been given. There is no concern for consumers.

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

Repeated dose toxicity

In an oral repeated dose study on male rats (drinking water/4 months) a nominal dose of 3000 mg/kg did not induce relevant toxic effects. However, investigated parameters were restricted to body weight, food and water consumption and alcohol uptake as well as liver weight and histology. The NOAEL is established at 3000 mg/kg bw/day.

Chronic oral exposure of consumers arises from the use of mouth hygiene products and amounts to 19.3 mg/kg bw/d.

For the decision on the appropriateness of MOS, the following aspects have been considered and taken into account:

- overall confidence in the database

No study strictly according to current guidelines is available. The available studies in rats and mice, covering application periods between five days and four months identify mainly the liver as the target organ of toxicity.

The data taken into account for performing the risk characterisation have been evaluated with regard to their reliability, relevance and completeness according to section 3.2 of the TGD. The data were published in peer reviewed journals, but the studies were not in accordance with GLP.

Based on the available repeated dose toxicity data which are considered to be of limited reliability, a scientifically sound health risk assessment cannot be performed. There is a need for further information and/or testing, i.e. formally conclusion (i) with the proposal to perform a 90-day rat inhalation study. However, a provisional risk characterisation can be based on the

oral NOAEL of 3000 mg/kg bw/d. The uncertainties in the database have to be considered in assessing the margin of safety.

- uncertainty arising from the variability in the experimental data

From the studies cited in 4.1.2.6 only one study allows to derive a NOAEL for oral application. The findings of all studies are not contradictory so that judgement can be based on this study.

- intra- and interspecies variation

Specific investigations about toxicokinetic behaviour and metabolism are available. All studies show comparable results. Propan-1-ol is readily metabolized via the aldehyde to propionic acid which can then be converted by a number of pathways (e.g., the citrate cycle).

With regard to intraspecies variability it is known that a functional polymorphism of the aldehyddehydrogenase enzyme AldDH2 exists which is involved in the metabolism of ethanol. But no data are available on the involvement of AldDH2 in the reaction in which propionaldehyde is converted to propionic acid. In a structure-activity assessment, however, it seems justified to make the assumption that this enzyme is involved and that the polymorphism plays a role in determining the internal exposure (AUC) to propionaldehyde. Taking into account that this polymorphism is confined to individuals of Asian origin in the European population it is concluded that people of the AldDH2 genotype may be at a higher risk.

- the nature and severity of the effect

The effects described are effects on the liver. These effects are not considered as serious health effects. No exposure related deaths occurred.

There are no reasons to assume that the effects shown in the animal experiments are limited to the species tested, thus being not of relevance for humans.

- dose-response relationship

The NOAEL for systemic effects was 3000 mg/kg bw/day. In a further study designed for research purposes rats received a dose of 25720 mg/kg bw/day propan-1-ol over a period lasted from one week up to 3 months. Electron microscopic studies of the liver showed a mixed population of small and enlarged mitochondria. The number of enlarged mitochondria per hepatocyte increased with prolongation of the duration of the experiment and more than 1 month was required to induce those changes. There are no indications for a steep dose-response relationship.

- differences in exposure (route, duration, frequency and pattern)

The estimated total chronic body burden with an assumed totally oral absorption is compared with a NOAEL of 3000 mg/kg bw/day in a drinking water study. The exposure estimates for

oral exposure to propan-1-ol is based on the assumption of daily use of a mouth-hygiene product in a worst case scenario. There are no reasons to assume that special concern can be derived from this procedure.

- the human population to which the quantitative and/or qualitative information on exposure applies

There are no substance-specific data which allow to quantify possible sensitivity differences among consumers. Following the exposure scenario there is no reason to assume a special risk for elderly, children or other people suffering from special diseases (besides liver diseases at a final stage).

other factors

There are no other factors known requiring a peculiar margin of safety.

MOS for oral exposure of the consumer:

Daily use of products will result in a propan-1-ol exposure (reasonable worst case) of 19.3 mg/kg bw/day.

The margin of safety between the

exposure estimate	19.3 mg/kg bw/day
and the	_____
NOAEL	3000 mg/kg bw/day

- is judged to be not sufficient taking into account the insufficiencies in the database.

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

This conclusion is drawn with reservation as there is the need for further testing for repeated dose toxicity (90-day rat inhalation study). The results of the required study may influence the outcome of the provisional risk characterisation for this endpoint.

MOS for dermal exposure of the consumer:

For the MOS calculation via dermal uptake the above described data regarding oral repeated dose study are sufficient. The estimated total chronic body burden with an assumed dermal absorption of 100% is compared with a NOAEL of 3000 mg/kg bw/day in a drinking water study. The exposure estimates of dermal exposure to propan-1-ol is based on the assumption of daily uses of cosmetics and reached 2.6 mg/kg bw/day exposure in reasonable worst case scenarios.

The margin of safety between the

exposure estimate	2.6 mg/kg bw/day
and the	
NOAEL	3000 mg/kg bw/day

is judged to be sufficient, even taking into account the insufficiencies in the database.

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

Inhalation exposure of the consumer:

For the MOS calculation via inhalation route one inhalation rat study is available with 9 exposure days. A $NOAEC_{local}$ of 500 ppm (1230 mg/m^3) for respiratory irritative effects can be derived from this study. The data seems not sufficient for the derivation of an inhalative $NOAEC_{sys}$. Therefore, the provisional oral $NOAEL_{sys}$ of 3000 mg/kg bw/d will be used for the risk characterisation.

For the decision on the appropriateness of MOS, the following aspects have been considered and taken into account:

- overall confidence in the database

The data taken into account for performing the risk characterisation have been evaluated with regard to their reliability, relevance and completeness according to section 3.2 of the TGD. Regarding irritative respiratory toxicity technical guidelines are not available. The chosen study in rats covers an application period of 9 days. The data were published in a peer reviewed journal. There are no reasons to assume limited confidence in the data which are chosen as the basis of the risk characterisation. For discussion of the oral data see above.

- uncertainty arising from the variability in the experimental data

From the study cited in 4.1.2.6 a NOAEC of irritation effects after inhalative application is available. However, there is a conclusion i, i.e. the need for further information and/or testing (90-day rat inhalation study).

- intra- and interspecies variation

Specific investigations about toxicokinetic behaviour and metabolism after inhalation are not available. For a discussion of general toxicokinetic data see above.

- the nature and severity of the effect

The systemic effects described are effects on the liver. These effects are not considered as serious health effects. No exposure related deaths occurred. Local irritative effects on the respiratory tract were also described. These are reversible.

There are no reasons to assume that the effects shown in the animal experiments are limited to the species tested, thus being not of relevance for humans.

- dose-response relationship

There are no indications for a steep dose-response relationship.

- differences in exposure (route, duration, frequency and pattern)

The estimated aggregated chronic exposure after inhalation is compared with a $NOAEC_{sys}$ derived from an oral drinking water study and a $NOAEC_{local}$ derived from a subchronic inhalation study. The exposure estimate is based on the combination of the non-user scenarios for disinfectants, general cleaning products, and kitchen cleaners, with a sum of 30 mg/m^3. There are no reasons to assume that special concern can be derived from this procedure.

- the human population to which the quantitative and/or qualitative information on exposure applies.

There are no substance-specific data which allow to quantify possible sensitivity differences among consumers. Following the exposure scenario there is no reason to assume a special risk for elderly, children or other people suffering from special diseases.

- other factors

There are no other factors known requiring a peculiar margin of safety.

MOS for inhalation exposure of the consumer, systemic effects:

The use of different consumer products will result in an aggregated external exposure via inhalation of 30 mg/m^3, corrresponding to an internal exposure of 5.1 mg/kg bw/d.

The margin of safety between the

exposure estimate	5.1 mg/kg bw/d
and the	
$NOAEL_{sys}$	3000 mg/kg bw/d

is judged to be sufficient.

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

This conclusion is drawn with reservation as there is the need for further testing for repeated dose toxicity (90-day rat inhalation study). The results of the required study may influence the outcome of the provisional risk characterisation for this endpoint.

MOS for inhalation exposure of the consumer, local effects:

The use of different consumer products will result in an aggregated external exposure via inhalation of 30 mg/m^3, corrresponding to an internal exposure of 5.1 mg/kg bw/d.

The margin of safety between the

exposure estimate	30 mg/m^3
and the	
$NOAEC_{local}$	1230 mg/m^3

is judged to be not sufficient.

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

MOS for the total exposure (dermal, oral and inhalation) of the consumer:

The total internal exposure has been calculated to be 27 mg/kg bw/day. The margin of safety between the

exposure estimate	27 mg/kg bw/day
and the	
$NOAEL_{sys}$	3000 mg/kg bw/day

is judged to be not sufficient taking into account the insufficiencies in the database.

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

This conclusion is drawn with reservation as there is the need for further testing for repeated dose toxicity (90-day rat inhalation study). The results of the required study may influence the outcome of the provisional risk characterisation for this endpoint.

Mutagenicity

There is no relevant concern with respect to mutagenicity. Propan-1-ol should not be classified as a mutagen.

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

Cancerogenicity

There is no valid carcinogenicity study available. Thus, a risk assessment for carcinogenicity can not be performed. However, taking into account the negative mutagenicity data it is concluded that carcinogenicity should not be an endpoint of concern.

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

Reproductive Toxicity

Fertility Impairment

In an inhalation study on reproductive toxicity, propan-1-ol did not induce detectable effects on the fertility of males or female rats at an exposure level of 3500 ppm, nor in females at 7000 ppm. At 7000 ppm, reduced fertility was observed in males. The NOAEC/fertility is established at 3500 ppm (8730 mg/m^3) corresponding to an internal exposure of approximately 2190 mg/kg bw/day.

For the decision on the appropriateness of MOS, the following aspects regarding the critical effect as well as exposure have been considered and taken into account:

- overall confidence in the database

The data taken into account for performing the risk characterization have been evaluated with regard to their reliability, relevance and completeness according to section 3.2 of the TGD. The data were submitted to the Competent Authority in private reports being adequately detailed and in accordance with internationally recognized guidelines and to GLP. There are no reasons to assume limited confidence.

- uncertainty arising from the variability in the experimental data

The findings of all studies are not contradictory so that the judgement can be based on the database (cf. 4.1.2.9).

- intra- and interspecies variation

There are no indications to limit the findings to a single species. The higher sensitivity of men compared to rats because of lower sperm reserve has to be taken into account.

- the nature and severity of the effect

The fertility effects observed in male rats revealed to be reversible within 13 weeks. There is the possibility that male rat infertility is caused by toxic effects to the testis which may also occur at lower doses but remained undetected. In rats, LOAEL values for testis histopathology are frequently lower than the corresponding values for infertility (cf. 4.1.3.2). There are no reasons to assume that the effects shown in the animal experiments are limited to the species tested, thus being not of relevance for humans.

- dose-response relationship

The effects of the male fertility were observed at high doses (7000 ppm / 17460 mg/m³), equivalent to an internal oral dose level 4.38 g/kg bw/day .

- differences in exposure (route, duration, frequency and pattern)

Following the exposure assessment, the consumer may be exposed to propan-1-ol via different routes. The systemic NOAEL was derived from an inhalation NOAEC. The resulting internal intake can be used for the risk characterisation of the other exposure routes as corrrections for differing bioavailability can be made (cf. 4.1.3.3).

MOS for inhalation exposure of the consumer:

Daily use of products will result in a propan-1-ol exposure concentration (reasonable worst case) of 30 mg/m³. The margin of safety between the

exposure estimate	30 mg/m³
and the	_____
NOAEC/fertility of	8730 mg/m³

is judged to be sufficient, taking into account the overestimated daily exposure figures.

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

MOS for oral exposure of the consumer:

Daily use of mouth hygiene products will result in an exposure of 19.3 mg/kg bw/day. The margin of safety between the

exposure estimate	19.3 mg/kg bw/day
and the	_____
NOAEL/fertility of	2190 mg/kg bw/day

is judged to be not sufficient taking into account a possible relation of the observed male infertility and testis toxicity that may occur at lower doses but remained undetected.

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

MOS for dermal exposure of the consumer:

Daily use of cosmetics will result in an exposure of 2.6 mg/kg bw/day. The margin of safety between the

exposure estimate	2.6 mg/kg bw/day
and the	_____
NOAEL/fertility of	2190 mg/kg bw/day

is judged to be sufficient.

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

MOS for the total exposure (dermal, oral and inhalation) of the consumer:

The total internal exposure has been calculated to be 27 mg/kg bw/day. The margin of safety between the

exposure estimate	27 mg/kg bw/day
and the	_____
NOAEL/fertility of	2190 mg/kg bw/day

is judged to be not sufficient taking into account a possible relation of the observed male infertility and testis toxicity that may occur at lower doses but remained undetected.

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

The concern arises mainly from the contribution of oral exposure to the combined body burden.

Developmental toxicity

Propan-1-ol did not induce detectable developmental effects in the conceptuses of dams exposed to 3500 ppm during the whole period of gestation. At maternally toxic concentrations of 7000 and 10000 ppm, embryotoxic, fetotoxic and teratogenic effects were observed. Based on the findings of reduced fetal body weights and higher incidences of rudimentary cervical ribs at gestational exposures of 7000 ppm, a NOAEC/developmental toxicity of 3500 ppm (8730 mg/m^3) is established from this study. This corresponds to an internal exposure of approximately 2190 mg/kg bw/day considering an inhalation absorption of 75%.

For the decision on the appropriateness of MOS, the following aspects regarding the critical effect as well as exposure have been considered and taken into account:

- overall confidence in the database

The data taken into account for performing the risk characterization have been evaluated with regard to their reliability, relevance and completeness according to section 3.2 of the TGD. The data were submitted to the Competent Authority in private reports being adequately detailed and in accordance with internationally recognized guidelines and to GLP.

There are no reasons to assume limited confidence.

- uncertainty arising from the variability in the experimental data

The findings of all studies are not contradictory so that the judgement can be based on the database (cf. 4.1.2.9).

-intra- and interspecies variation.

There are no indications to limit the findings to a single species. The effects are expected to be based on the chemical structure. Also ethanol shows similar effects.

- the nature and severity of the effect

The developmental effects are considered to be severe health effects per se. They were observed essentially at high exposure concentrations of 10000 ppm leading to maternal toxicity. There are no reasons to assume that the effects shown in the animal experiments are limited to the species tested, thus being not of relevance for humans.

- dose-response relationship

The mentioned effects were only observed at high maternally toxic doses (7000 and 10000 ppm; equivalent oral dose level 5800 and 8300 mg/kg bw/day.)

- differences in exposure (route, duration, frequency and pattern)

Following the exposure assessment, the consumer may be exposed to propan-1-ol via different routes. The systemic NOAEL was derived from an inhalation NOAEC. The resulting internal intake can be used for the risk characterisation of the other exposure routes as corrrections for differing bioavailability can be made (cf. 4.1.3.3)

MOS for inhalation exposure of the consumer:

Daily use of products will result in a propan-1-ol exposure concentration (reasonable worst case) of 30 mg/m³. The margin of safety between the

exposure estimate	30 mg/m³
and the	_____
NOAEC/dev.tox. of	8730 mg/m³

is judged to be sufficient, taking into account the nature and severity of the effect and the overestimated daily exposure figures.

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

MOS for oral exposure of the consumer:

Daily use of mouth hygiene products will result in a propan-1-ol exposure (reasonable worst case) of 19.3 mg/kg bw/day. The margin of safety between the

exposure estimate	19.3 mg/kg bw/day
and the	_____
NOAEL/dev.tox. of	2190 mg/kg bw/day

is judged to be sufficient, taking into account the nature and severity of the effect.

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

MOS for dermal exposure of the consumer:

Daily use of cosmetics will result in a propan-1-ol exposure (reasonable worst case) of 2.6 mg/kg bw/day.The margin of safety between the

exposure estimate	2.6 mg/kg bw/day
and the	_____

NOAEL/dev.tox. of 2190 mg/kg bw/day

is judged to be sufficient.

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

MOS for the total exposure (dermal, oral and inhalation) of the consumer:

The total internal exposure has been calculated to be 27 mg/kg bw/day. The margin of safety between the

exposure estimate 27 mg/kg bw/day

and the _____

NOAEL/dev.tox. of 2190 mg/kg bw/day

is judged to be not sufficient.

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

The concern arises mainly from the contribution of oral exposure to the combined body burden.

4.1.3.4 Man exposed indirectly via the environment

a) Local approach

The local model indicates consumption of leaf crops and inhalation as main routes for indirect exposure. Model calculations for the local scenario resulted in a total daily dose in the range of 0.036 mg/kg bw/day (cf. 4.1.1.4).

Repeated dose toxicity

From a repeated dose toxicity study (4 months) with rats a NOAEL of 3000 mg/kg bw/day was derived.

Comparison indirect exposure -NOAEL

Indirect exposure (local) 0.036 mg/kg bw/day

_____ = _____

NOAEL 3000 mg/kg bw/day

The margin of safety between the calculated exposure for the indirect exposure in the local approach and the NOAEL is judged to be sufficient even taking into account the insufficiencies in the database (cf. 4.1.3.3). Thus, the substance is of no concern for possible health effects risks in this provisional risk assessment in relation to local indirect exposure via the environment.

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

For repeated dose toxicity there is the need for further testing (90-day rat inhalation study). However, it might be expected that the results of this study will not influence the outcome of this risk characterisation.

Reproductive toxicity

Fertility

Data from an inhalation study on rats with propan-1-ol did not give evidence for adverse effects on reproductive performance and outcome for doses of up to 3500 ppm (resp. 2190 mg/kg bw/day) (cf. 4.1.3.3).

Comparison indirect exposure - NOAEL

$$\frac{\text{Indirect exposure (local)}}{\text{NOAEL}} = \frac{0.036 \text{ mg/kg bw/day}}{2190 \text{ mg/kg bw/day}}$$

The margin of safety between the calculated exposure for indirect local exposure source and the NOAEL is judged to be sufficient. Thus, regarding adverse effects on reproductive performance the substance is of no concern in relation to indirect exposure via the environment.

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

Developmental toxicity

Fetotoxic and teratogenic effects in rats occur at propan-1-ol exposure levels of approximately 7500 ppm and above. From this inhalation study a NOAEL of 3500 ppm (resp. 2190 mg/kg bw/day) was derived (cf. 4.1.3.3).

Comparison indirect exposure - NOAEL

Indirect exposure (local) 0.036 mg/kg bw/day

_____ = _____

NOAEL 2190 mg/kg bw/day

The margin of safety between the calculated exposure indirect exposure and the NOAEL is judged to be sufficient. Thus, regarding fetotoxic and teratogenic effects the substance is of no concern in relation to indirect exposure via the environment.

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

b) Regional approach

Using the regional approach the only significant indirect exposure occurs via drinking water. Model calculations for the regional scenario for propan-1-ol resulted in a total daily dose $3.12*10^{-4}$ mg/kg bw/d (cf. 4.1.1.4).

Repeated dose toxicity

From a repeated dose toxicity study (4 months) with rats a NOAEL of 3000 mg/kg bw/day was derived.

Comparison indirect exposure -NOAEL

Indirect exposure (regional) $3*10^{-4}$ mg/kg bw/day

_____ = _____

NOAEL 3000 mg/kg bw/day

The margin of safety between the calculated exposure for the only significant indirect exposure source drinking water in the regional approach and the NOAEL is judged to be sufficient even taking into account the insufficiencies in the database (cf. 4.1.3.3). Thus, the substance is of no concern for health risks in relation to indirect exposure via the environment in this provisional risk assessment.

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

For repeated dose toxicity there is the need for further testing (90-day rat inhalation study). However, it is expected that the results of this study will not influence the outcome of this risk characterisation.

Reproductive toxicity

Fertility

Data from a inhalation study on rats with propan-1-ol did not give evidence for adverse effects on reproductive performance and outcome for doses of up to 3500 ppm (resp. 2190 mg/kg bw/day).

Comparison indirect exposure - NOAEL

Indirect exposure (regional)		$3*10^{-4}$ mg/kg bw/day
_____	=	_____
NOAEL		2190 mg/kg bw/day

The margin of safety between the calculated exposure via drinking water and the NOAEL is judged to be sufficient. Thus, regarding adverse effects on reproductive performance the substance is of no concern in relation to indirect exposure via the environment.

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

Developmental toxicity

Fetotoxic and teratogenic effects have been observed in rats at propan-1-ol exposure levels of approximately 7500 ppm and above. From the inhalation study a NOAEC of 3500 ppm (resp. NOAEL of 2190 mg/kg bw/day) was derived.

Comparison indirect exposure (regional) - NOAEL

Indirect exposure (regional)		$3*10^{-4}$ mg/kg bw/day
_____	=	_____
NOAEL		2190 mg/kg bw/day

The margin of safety between the calculated exposure via drinking water and the NOAEL is judged to be sufficient. Thus, regarding fetotoxic and teratogenic effects the substance is of no concern in relation to indirect exposure via the environment.

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

4.1.3.5 (Combined exposure)

It is possible for an individual to receive exposure to propan-1-ol at work, from consumer products and indirectly via the environment. The levels that would be received indirectly from environmental sources are so low that they will not significantly add to the daily body burden received at work and at home. However, the levels of propan-1-ol in consumer products are not negligible so that they should add to the daily body burden received at work.Therefore the conclusions reached for workers apply to combined exposure.

4.2 HUMAN HEALTH (PHYSICO-CHEMICAL PROPERTIES)

4.2.1 Exposure assessment

4.2.1.1 Occupational exposure

See chapter 4.1.1.1

4.2.1.2 Consumer exposure

4.2.1.3 Indirect exposure via the environment

4.2.2 Effects assessment: Hazard identification and Dose (concentration) - response (effect) assessment

4.2.2.1 Explosivity

Propan-1-ol is not explosive.

4.2.2.2 Flammability

Propan-1-ol is flammable.

4.2.2.3 Oxidising potential

Due to its chemical structure, propan-1-ol is not expected to possess any oxidizing properties.

4.2.3 Risk characterisation

4.2.3.1 Workers

Propan-1-ol is flammable. Adequate worker protection measures are requested. Risk reduction measures beyond those which are beeing applied already are not considered necessary.

Conclusion: ii

4.2.3.2 Consumers

4.2.3.3 Man exposed indirectly via the environment

5 CONCLUSIONS / RESULTS

Environment

From the intrinsic properties it is expected that propan-1-ol is of low concern for the environment. Therefore, a targeted environmental risk assessment was performed. using conservative estimates based on worst-case assumptions at the exposure and effects side. The targeted risk assessment results in the following conclusion:

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

Based on the currently available data, propan-1-ol represents no risk to the environment for the area of production, processing, formulation and use (see chapter 3.3).

Although the exposure calculation is based on conservative „worst case“ assumptions the calculated environmental concentrations remain clearly under the predicted no effect concentrations.

Human Health

Workers

i) There is need for further information and/or testing

Further toxicological data (90-day rat inhalation study) is needed in order to perform a robust occupational risk assessment for repeated dose toxicity (systemic effects).

Propan-1-ol is also notified as an active substance within the scope of the Biocide Directive 98/8/EC. The necessary data on repeated dose toxicity is existing, but is owned by a company who wishes to use it in the framework of other EU regulation (the Biocides Directive). The company is so far not willing to make the study available to support risk assessment in the context of the Existing Substances Regulation, and there are no provisions in the Biocides Directive that would force them to share the data with other companies. The information on repeated dose toxicity is requested for reasons of human health. For the sake of animal protection it is hoped that the companies involved will be able to negotiate and share the data.

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

Occupational risk assessment reveals that for certain exposure scenarios there is a need for limiting the risks. The toxicological endpoints of concern especially are reprotoxicity (fertility impairment and developmental toxicity) and local effects by inhalation (acute sensory irritation and respiratory tract irritation by repeated exposure). Sensory irritation may occur following high short-term exposure levels. Results from the required 90-day rat inhalation study may have an impact on the risk assessment for fertility impairment and respiratory tract irritation by repeated exposure.

Slight effects to the skin following repeated dermal exposure cannot be excluded. No concern is expressed for eye irritation; it is assumed that corresponding classification of propan-1-ol will result in necessary risk reduction measures.

Concern especially concentrates on the use of paints (scenario 3) and on the use of cleaning formulations (scenario 4a).

Consumers

i) There is need for further information and/or testing

Further toxicological data is needed in order to perform a sound risk assessment for repeated dose toxicity (cf. conclusion i for workers).

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

The risk characterisation for the oral exposure scenario via mouth hygiene products and the aggregated exposure scenario of consumers via mouth hygiene products, cosmetics, disinfectants and general cleaning products leads to conclusion iii) because of concern for men with respect to fertility impairment.

The risk characterisation for the oral exposure scenario via mouth hygiene products and the aggregated exposure scenario of consumers via mouth hygiene products, cosmetics, disinfectants and general cleaning products leads to conclusion iii) because of concern with respect to developmental toxicity.

Sensory irritation may occur following short-term inhalation exposure during the application of propan-1-ol containing disinfectants, hardener solutions, and wall aper removers.

The provisional risk characterisation for repeated dose toxicity reveals that there may be a need for limiting the risks due to the oral exposure scenario via mouth hygiene products and the aggregated exposure scenario of consumers via mouth hygiene products, cosmetics, disinfectants and general cleaning products.. The results of the required inhalation study may influence the outcome of the risk characterisation for consumers.

Man exposed indirectly via the environment

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

Further toxicological data is needed in order to perform a sound risk assessment for repeated dose toxicity (cf. conclusion i for workers).

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

Conclusion (ii) is reached for the provisional risk characterisation for repeated dose toxicity and for all other toxicological endpoints for the local and the regional scenario.

6 REFERENCES

Abraham, M.H., Chadha, H.S., Whiting, G.S., Mitchell, R.C. (1994): J. Pharm. Sci., **83**: 1085-1100

Ahrens, W., Jöckel, K-H. (1996): Stoffbelastung in der Papierindustrie. Schriftenreihe der Bundesanstalt für Arbeitsschutz – GA 48, S.68-71, Wirtschaftsverlag NW, Bremerhaven

Alarie, Y. (1981): Dose response analysis in animal studies: Prediction of human responses. Env Health Perspect, **42**: 9-13

Ariel (2000): Ariel Insight 2.0: For survival in a world of expanding chemical regulations, April 2000

Altschuh, J., Brüggemann, R., Santl, H., Eichinger, G., Piringer, O. G. (1999): Henry's law constants for a diverse set of organic chemicals: experimental determination and comparison of estimation methods. Chemosphere, **39**: 1871-1887

Auty, R.M., Branch R.A. (1976): The elimination of ethyl, n-propyl, n-butyl and iso-amyl alcohols by the isolated perfused rat liver. J Pharmacol Exp Ther, **197**, 669-674

Babeu, L., Vaishnav, D.D. (1987): Prediction of biodegradability for selected organic chemicals. J. Ind. Microbiology, **2**, 107-115

Bariljak, I.P., Kozachuk, C.J. (1988): Untersuchung der cytogenetischen Wirkung einer Reihe einwertiger Alkohole auf Zellen des Knochenmarks von Ratten. Citologija i genetika, **22**: 49-52

BASF AG (1974): Unpublished report XXIV 522;

BASF AG (1978): Bestimmung der biologischen Abbaubarkeit von n-Propanol - Prüfergebnis, BASF AG DUU/OM - Z 570, 16.3.1978, unpublished.

BASF AG (1980): unpublished results; 78/647

BASF AG (1989): Labor fuer Umweltanalytik; unveroeffentlichte Untersuchung (09.01.1989)

BASF AG (1994): unpublished information from 19.01.1994

BASF AG (1995): Safety data sheet "n-Propanol" (23.05.1995)

BASF AG (1996): unpublished information from 19.04.1996

BASF AG (1999): unpublished information from 18.10.1999

BASF AG (2003): Report. In vitro chromosome aberration assay with n-Propanol in V79 cells. Project No.: 32M0469/024111

BAU (1994): Neue Stoffe am Arbeitsplatz: Ein Bewertungskonzept, Amtliche Mitteilungen der Bundesanstalt für Arbeitsschutz, Sonderdruck März 1994, Dortmund, Germany

Baumann, W., Muth, A. (1997): Farben und Lacke 1 - Daten und Fakten zum Umweltschutz, S. 143-146, Springer Verlag, Berlin

Baumann, W., Herberg-Liedtke, B. (1991): Druckereichemikalien - Daten und Fakten zum Umweltschutz, Springer Verlag, Berlin

Beaugé, F., Clément, M., Nordmann, J., Nordmann, R. (1979): Comparative effects of ethanol, n-propanol and isopropanol on lipid disposal by rat liver. Chem Biol Interact, **26**: 155-166

Bengtsson, B.-E., Renberg, L., Tarkpea, M. (1984): Molecular structure and aquatic toxicity - an example with C1-C13 aliphatic alcohols. Chemosphere, 13: 613-622.

Betterton, E.A.: Henry's law constants of soluble and moderately soluble organic gases: effects on aqueous phase chemistry. In: Nriagu, J. O. (1992): Gaseous pollutants, Wiley Series: Advances in Environmental Science and Technology, Vol. 24, John Wiley & Sons, New York

BGAA (1997): Altstoffe – Exposition am Arbeitsplatz – 1-Propanol In: Berufsgenossenschaftlicher Arbeitskreis Altstoffe (BGAA) Report 1/99, Hauptverband der gewerblichen Berufsgenossenschaften, Sankt Augustin

Bilzer, N., Penners, B.-M. (1985): Zur Frage der Abbau- und Ausscheidungsgeschwindigkeit der Begleitstoffe Propanol-1 und Isobutanol nach Genuß von Whisky der Marke "Chivas Regal". Blutalkohol, **22**: 140-145

Bilzer, N., Schmutte, P., Jehs, M., Penners, B.-M. (1990): Kinetik aliphatischer Alkohole (Methanol, Propanol-1 und Isobutanol) bei Anwesenheit von Äthanol im menschlichen Körper. Blutalkohol, **27**: 385-409

Blum, D.J.W., Speece, R.E. (1991): A database of chemical toxicity to environmental bacteria and its use in interspecies comparison and correlations. Res. J. Water Poll. Contr. Fed. 63 (3), 198-207

Bonte, W., Rüdell, E., Sprung, R., Frauenrath, C., Blanke, E., Kupilas, G., Wochnik, J., Zäh, G. (1881a): Experimentelle Untersuchungen zum Nachweis geringer Dosen höherer aliphatischer Alkohole im Urin von Versuchsteilnehmern. Blutalkohol, **18**: 399-411

Bonte, W., Sprung, R., Rüdell, E., Frauenrath, C., Blanke, E., Kupilas, G., Wochnik, J., Zäh, G. (1981b): Experimentelle Untersuchungen zum Nachweis geringer Dosen höherer aliphatischer Alkohole im Urin von Versuchsteilnehmern. Blutalkohol, **18**: 412-426

Bos, P.M.J., Zwart, A., Reuzel, P.G.J., Bragt, P.C. (1992): Evaluation of the sensory irritation test for the assessment of occupational health risk. Crit Rev Toxicol, **21**: 423-450

Boublik, T., Fried, V., Hala, E. (1984): The vapour pressure of pure substances. 2nd ed., Elsevier, Amsterdam

Bremmer, H.J., Veen, M.P. van (2000): Factsheet algemeen. RIVM report 612810 009

Bringmann, G. (1975): Bestimmung der biologischen Schadwirkung wassergefährdender Stoffe aus der Hemmung der Zellvermehrung der Blaualge Microcystis. Gesundheitsingenieur 96, 238-241

Bringmann, G. (1978): Bestimmung der biologischen Schadwirkung wassergefährdender Stoffe gegen Protozoen. Mitt. I. Bakterienfressende Flagellaten. Z. Wasser Abwasser Forsch. 11, 210-215

Bringmann, G., Kühn, R. (1977a): Befunde der Schadwirkung wassergefährdender Stoffe gegen Daphnia magna. Z. Wasser Abwasser Forsch. 10, 161-166

Bringmann, G., Kühn, R. (1977b): Grenzwerte der Schadwirkung wassergefährdender Stoffe gegen Bakterien (Pseudomonas putida) und Grünalgen (Scenedesmus quadricauda) im Zellvermehrungshemmtest. Z. Wasser Abwasser Forsch. 10, 87-98

Bringmann, G., Kühn, R. (1980): Bestimmung der biologischen Schadwirkung wassergefährdender Stoffe gegen Protozoen. Mitt. II. Bakterienfressende Ciliaten. Z. Wasser Abwasser Forsch. 13, 26-31

Bringmann, G., Kühn, R., Winter, A. (1980): Bestimmung der biologischen Schadwirkung wassergefährdender Stoffe gegen Protozoen. Mitt. III. Saprozoische Flagellaten. Z. Wasser Abwasser Forsch. 13, 170-173

Bringmann, G., Kühn, R. (1982): Ergebnisse der Schadwirkung wassergefährdender Stoffe gegen Daphnia magna in einem weiterentwickelten standardisierten Testverfahren. Z. Wasser Abwasser Forsch. 15, 1-6

Brooke, L.T., Call, D.J., Geiger, D.L., Northcott, C.E. (1984): Acute toxicities of organic chemicals to fathead minnows (Pimephales promelas). Vol. 1, pp 3, 5-16, 65-68. Center for Laker Superior Environmental Studies, University of Wisconsin-Superior.

Bushy Run Research Center (1979): Evaluation of the dermal carcinogenic potential of n-propanol

Bushy Run Research Center (1980): Evaluation of the dermal cancerogenic potential of n-propanol. Project Report 42-96; March 14, 1980, cited in EUCLID Data Sheet Union Carbide Benelux N V; 07.12.1995

Bushy Run Research Center (1992): n-Propyl Alcohol (n-Propanol): Nine-Day-Vapor Inhalation in Rats. Project Report 54-87; March 5

Carlson, G.P. (1993): Formation of fatty acid propyl esters in liver, lung and pancreas of rats administered propan-1-ol. Res Commun Chem Pathol Pharmacol, **81**, 121-124

CEH (1995): Oxochemicals. In: Chemical Economics Handbook, S. 682.7002S, 682.7002T, 684.7001C; SRI International

CHEMSAFE: national database for safety data of the Physikalisch-technische Bundesanstalt Braunschweig, established by expert judgement

Cometto-Muniz, J.E., Cain, W.S. (1990): Thresholds for odor and pungency. Physiology and Behavior, **48**: 719-725

CRC handbook of chemistry and physics, 72nd ed., 1991-1992, CRC Press

Crebelli, R., Conti, G., Carere, A. (1989): A comparative study on ethanol and acetaldehyde as inducers of chromosome malsegregation in aspergillus nidulans. Mutation Res, **215**: 187-195

Delogu, B. (2000): Understanding the Precautionary Principle. Presentation at the International Conference on Chemical Control Regulations as representative of EU DG SANCO, 10/12 May 2000, Documentation ChemCon 2000, Austrian Federal Economic Chamber, Salzburg, Austria

Dürwald, W., Degen, W. (1956): Eine tödliche Vergiftung mit n-Propylakohol. Arch Toxikol, **16**: 84-88

ECB4/TR2/98: Technical Recommendation "The use of the 10 % rule in emission estimations", ECB, Joint Research Centre, 1998.

EHC (1990): International Programme on Chemical Safety (IPCW), Environmental Health Criteria 102, propan-1-ol, World Health Organization, Geneva, 98 pages

Ehrig. T., Bohren, K.M., Wermuth, B., von Wartburg, J.P. (1988): Degradation of aliphatic alcohols by human liver alcohol dehydrogenase: Effect of ethanol and pharmacokinetic implications. Alcoholism : Clin Exp Res, **12**: 789-794

Fahelbum, I.M.S., James, S.P. (1979): Absorption, distribution and metabolism of propyl anthranilate. Toxicology, **12**: 75-87

Falbe J. et al. (1980): Propanol. In: Ullmanns Enzyklopädie der technischen Chemie, 4. Auflage, Bd.19, 443-451, Verlag Chemie, Weinheim - Deerfield Beach – Basel

Fiserova-Bergerova, V. (1985): Toxicokinetics of organic solvents. Scand J Work Environ Health, suppl **1**: 7-21

Fiserova-Bergerova, V., Diaz, M.L. (1986): Determination and prediction of tissue-gas partition coefficients. Int Arch Occup Environ Health, **58**: 75-87

Gad, S.C., Dunn, B.J., Dobbs, D.W., Reilly, C., Walsh, R.D. (1986): Development and validation of an alternative dermal sensitization test: The mouse ear swelling test (MEST). Toxicol Appl Pharmacol, **84**: 93-114

Gargas, M.L., Medinsky, M.A., Andersen, M.E. (1993): Advances in physiological modelling. Approaches for understanding the disposition of inhaled vapors. In Toxicology of the lung. Edited by D.E. Gardner et al., 2nd ed.. Raven Press, Ltd., New York, 461-483

GDCh (1997): BUA Report No. 190, 1-propanol, S. Hirzel Wissenschaftliche Verlagsgesellschaft

Gerner, Muhl, Rühl, Teich, Waßmann (1997): Beschichtungsarbeiten, BAU BG Hamburg, 1997

Gibel, W., Lohs, K., Wildner, G.P. (1975): Experimental study on the cancerogenic activity of propanol-1, 2-methylpropanol-1 and 3-methylbutanol. I. Arch. Geschwulstforsch., **45**: 19-24

Ginsberg, G., Smolenski, S., Hattis, D., Sonawane, B. (2002): Population distribution of aldehyde dehydrogenase-2 genetic polymorphism: implications for risk assessment. Regul. Toxicol. Pharmacol., **36**: 297-309

Grant, K.A., Samson, H.H. (1984): n-Propanol induced microcephaly in neonatal rat. Neurobehav Toxicol Teratol, **6**: 165-169

Gulati, A., Nath, C., Shanker, K., Srimal, R.C., Dhawan, K.N., Bhargava, K.P. (1985): Effect of alcohols on the permeability of blood-brain barrier. Pharmacol Res Commun, **17**: 85-93

Guo, Z., Sparks, L.E., Bero, M.R. (1995): Air exchange rate measurements in an IAQ test house. Engeneering Solutions to Indoor Air Quality Problems, Research Triangle Park, 498-510

Haddock, N.F., Wilkin, J.K. (1982): Cutaneous reactions to lower aliphatic alcohols before and during disulfiram therapy. Arch Dermatol, **118**: 157-159

Halarnkar, P.P., Blomquist, G.J. (1989): Comparative aspects of propionate metabolism. Comp Biochem Physiol, **92B**: 227-231

Hansch, C., Anderson, S.M. (1967) J. Org. Chem., 32, 2583

Harke, P. (1998): Disinfectants – Uses. In: Ullmann's Encylopedia of Industrial Chemistry, Sixth Ed., Edition Release, Wiley VCH, Weinheim

Hayes, S., Hayes, C., Duncan, D., Bennett, V., Blushke J. (1990): Stimulation of mutations suppressing the loss of replication control by small alcohols. Mutation Res, **231:** 151-163

Hein, P.M., Magerl, H., Schulz, E. (1989): Detection of alcohols in saliva. J Clin Chem Clin Biochem, **27**: 231

Heydenreich, A. (1966): Chemisch-toxische Schäden der Augen (Vergiftungen, Berufskrankheiten) Monatsblätter für Augenheilkunde, **149**: 145-165

Hiaki, T., Takahashi, K., Tsuji, T., Hongo, M., Kojima, K. (1994), J. Chem. Eng. Data, 39, 602-604

Hillbom, M.E., Franssila, K., Forsander, O.A. (1974): Effects of chronic ingestion of some lower aliphatic alcohols in rats. Res Comm Pathol Pharmacol, **9**: 177-180

Hilscher, H., Geissler, E., Lohs, K., Gibel, W. (1969): Untersuchungen zur Toxizität und Mutagenität einzelner Fuselöl-Komponenten an E.coli. Acta biol med germ, **23**: 843-852

Hoechst AG (1994): unpublished information from 14.06.1994

Hoechst AG (1995a): Safety data sheet "n-Propanol" (20.07.1995)

Hoechst AG (1995b): unpublished information from 15.12.1995

Hoechst AG (1997): unpublished information from 20.03.1997

Iffland, R., Balling, P., Oehmichen, M., Lieder, F., Norpoth, Th. (1989): Methanol, Isopropanol, n-Propanol - endogene Bildung unter Äthanoleinfluß? Blutalkohol, **26**: 87-97

INRS (2000): Results of occupational exposure measurements to 1-propanol, COLCHIC database, No. 38/2000

Johanson, G. (1991): Modelling of respiratory exchange of polar solvents. Ann Occup Hyg, **35**: 323-339

Juhnke, I., Lüdemann, D. (1978): Ergebnisse der Untersuchung von 200 chemischen Verbindungen auf akute Fischtoxizität mit dem Goldorfentest. Z. Wasser Abwasser Forsch. 5, 161-164

Kalberlah, F. et al. (1999): Zeitextrapolation und Interspeziesextrapolation bei lokal wirksamen Stoffen mit begrenzter Datenlage, Endbericht des Forschungsvorhabens F1719 der BauA, Bundesanstalt für Arbeitsschutz und Arbeitsmedizin, Schriftenreihe -Forschung- Fb 862

Kalberlah, F., Schneider, K. (1998): Quantifizierung von Extrapolationsfaktoren, Endbericht des Forschungsvorhabens Nr. 116 06 113 des Umweltbundesamtes, Bundesanstalt für Arbeitsschutz und Arbeitsmedizin, Schriftenreihe -Forschung- Fb 796

Kamil, I.A., Smith, J.N., Wiliams, R.T. (1953): Studies in detoxication. 46. The metabolism of aliphatic alcohols. The glucuronic acid conjugation of acyclic aliphatic alcohols. Biochem J, **53**: 129-137

Kane, L.E., Dombroke, R., Alarie, Y. (1980): Evaluation of sensory irritation from some common industrial solvents. Am Ind Hyg Ass, **41**: 451-455

Kaneko, T., Wang, P.-Y., Sato, A. (1994): Partition coefficients of some acetate esters and alcohols in water, blood, olive oil, and rat tissues. Occupat Environ Med, **51**: 68-72

Kennedy, G.L., Graepel, G.J. (1991): Acute toxicity in the rat following either oral or inhalation exposure. Toxicol Letters, **56**: 317-326

Khudoley et al. (1987): The study of mutagenic activity of carcinogens and other chemical agents with Salmonella typhimurium assays: Testing of 126 compounds. Arch f Geschwulstforschung, **57**: 453-462

Klecka, G.M., Land, L.P., Bodner, K.M. (1985): Evaluation of the OECD activated sludge respiration inhibition test. Chemosphere, **14:** 1239-1251

Kruhoffer, P.W. (1983): Handling of inspired vaporized ethanol in the airways and lungs (with comments on forensic aspects). Forensic Science International, 21; 1-17

Kristiansen, U., Hansen, L., Nielsen, G.D., Holst, E. (1986): Sensory irritation and pulmonary irritation of cumene and n-propanol: Mechanisms of receptor activation and desentitization. Acta pharmacol et toxicol, **59**: 60-72

Kühn, R., Pattard, M., Pernak, K.-D., Winter, A. (1989): Results of the harmful effects of selected water pollutants (anilines, phenols, aliphatic compounds) to Daphnia magna. Water Res., **23:** 495-499

Kühnholz, B., Wehner, H.D., Bonte, W. (1984): In-vitro-Untersuchungen zur Löslichkeit aliphatischer Alkohole in Körpergeweben. Blutalkohol, **21**: 308-318

Lasne et al. (1984): The in vitro micronucleus for detection of cytogenetic effects induced by mutagen-arcinogens:comperison with in vitro sister-chromatid exchange assay. Mutation Res, **130**: 273-282

Ludwig, E., Hausen, B.M. (1977): Sensitivity to isopropyl alcohol. Contact Dermatitis, **3**: 240-244

Maickel, R.P., Nash, J.F. Jr. (1985): Differing effects of short-chain alcohols on body temperature and coordinated muscular activity in mice. Neuropharmacology, **24**: 83-89

Morgan, E.T., Koop, D.R., Coon, M.J. (1982): Catalytic activity of cytochrome P-450 isozyme 3a isolated from liver microsomes of ethanol-treated rabbits. J Biol Chem, **257**: 13951-13957

Morris, J.B., Cavanagh, D.G. (1987): Metabolism and deposition of propanol and acetone vapors in the upper respiratory tract of the hamster. Fund Appl Toxicol, **9**: 34-40

Munch, J.C. (1972): Aliphatic alcohols and alkyl esters: narcotic and lethal potencies to tadpoles and rabbits. Indust Med, **41**: 31-33

Nelson, B.K., Brightwell, W.S., Taylor and Burg, J.R. (1985): Comparison of behavioral teratogenic effects of ethanol and N-propanol administered by inhalation to rats. Neurobehavioral Toxicology and Teratology, **7**: 779-783

Nelson, B.K., Brightwell, W.S., MacKenzie-Taylor, D.R., Khan, A., Burg, J.R., Weigel, W.W. (1988): Teratogenicity of n-propanol and isopropanol administered at high inhalation concentration to rats. Fd Chem Toxicol, **26**: 247-254

Nelson, B.K., Brightwell, W.S., Taylor, B.J., Khan, A., Burg, J.R., Krieg, E.F., JR and Massari, V.J. (1989): Behavioral teratology investigation of propan-1-ol administered by inhalation to rats. Neurotoxicology and Teratology, **11**: 153-159

Nelson, B.K., Brightwell, W.S., Krieg, E.F. (1990): Developmental toxicology of industrial alcohols: a summary of 13 alcohols administered by inhalation to rats. Toxicol Industrial Health, **6**: 373-387

Nelson, B.K., Brightwell, W.S., Krieg, E.F. (1996): Developmental toxicology of industrial alcohols: a summary of 13 alcohols administered by inhalation to rats. Int J Occup Med Immunol Toxicol, **5**: 29-42

Nielsen, G.D., Bakbo, J.C. (1985): Exposure limits for irritants. Ann Am Conf Ind Hyg, **12**: 119-133

NO_NL (1999): Guidelines for quantitative risk characterisation of non-threshold carcinogens in the framework of existing chemicals following Council Regulation (EEC) 793/93, Draft 09.08.99, Commission Working Group on the Technical Meetings for Risk Assessment for Existing Substances, NO_NL/01/99

Nordman, R. (1980): Metabolism of some higher alcohols. INSERM (Les Colloques de l`INSERM: Alcohol and the gastrointestinal tract), **95**: 187-205

Obe, G., Ristow, H. (1977): Acetaldehyde, but not ethanol, induces sister chromatid exchanges in Chinese hamster cells in vitro. Mutation Res, **56**: 211-213

Pedersen, L.M. (1987): Biological Studies in human exposure to and poisoning with organic solvents: Kinetics, haemotology, and serum chemistry. Phamacol Toxicol, **61**, Suppl. III: 1-38

Peschel, O., Bauer, M.F., Gilg, T., v. Meyer, L. (1992): Veränderung von Begleitstoffanalysen durch percutane Resorption propanolhaltiger Antiseptika. Blutalkohol, **29**: 172-184

Petrasol B.V. Gorinchem, HSDB (Hazardous Substances Data Bank), on-line via STN

Pitter, P. (1976): Determination of biological degradability of organic substances. Water Res. **10**, 231-235

Price, K.S., Waggy, G.T., Conway, R.A. (1974): Brine shrimp bioassay and seawater BOD of petrochemicals. J. Water Pollut. Contr. Fed. **46**, 63-77

Rietbrock, N., Abshagen, U. (1971): Pharmakokinetik und Stoffwechsel aliphatischer Alkohole. Arzneimittelforsch, **21**: 1309-1319

Robra, K.H. (1979): Akute Bakterientoxizität: Auswertung von Ringversuchen mit einer Reinkultur im Vergleich zu Untersuchungen an Mischpopulationen. Vom Wasser 53, 267-282

Rotter, M.L., Koller, W., Neumann, R. (1991): The influence of cosmetic additives on the acceptability of alcohol-based hand disinfectants. J Hospital Inf, **18**: 57-63

Savini, E.C. (1968): Estimation of the LD50 in Mol/kg. Proceedings of the European Society for the Study of Drug. Toxicity, **9**: 276-278

Schaper, M. (1993): Development of a database for sensory irritants and its use in establishing occupational exposure limits. Am Ind Hyg Assoc, **54**: 488-495

Scheuplein, R.J., Blank, I.H. (1973): Mechanism of percutaneous absorption. IV. Penetration of nonelectrolytes (alcohols) from aqueous solutions and from pure liquids. J Invest Dermatol, **60**: 286-296

Schmutte, P., Bilzer, N., Penners, B.M. (1988): Zur Nüchternkinetik der Begleitalkohole Methanol und Propanol-1. Blutalkohol, **25**: 137-142

Shehata, M., Saad, S. (1978): The effect of aliphatic alcohols on certain vitamins of the B-complex group in the liver of the rat. Pol J Pharmacol Pharm, **30**: 35-39

Siegel, I.A., Izutsu, K.T., Watson, E. (1981): Mechanisms of non-electrolyte penetration across dog and rabbit oral mucosa in vitro. Arch Oral Biol, **26**: 357-361

Sinclair, J., Lambrecht, L., Smith, E.L. (1990): Hepatic alcohol dehydrogenase activity in chick hepatocytes towards the major alcohols present in commercial alcoholic beverages: Comparison with activities in rat and human liver. Comp Biochem Physiol, **96B**: 677-682

Slooff, W. (1983): Benthic macroinvertebrates and water quality assessment: some toxical considerations. Aquatic Toxicol. **4**, 73-82

Slooff, W., Baerselman, R. (1980): Comparison of the usefulness of the mexican Axolotl (Ambystoma mexicanum) and the Clawed Toad (Xenopus laevis) in toxicological bioassays. Bull. Environ. Contam. Toxicol. **24**, 439-443

Slooff, W., Canton, J.H., Hermens, J.L.M. (1983): Comparison of the susceptibility of 22 freshwater species to 15 chemical compounds. I. (Sub)acute toxicity tests. Aquatic Toxicol. **4**, 113-128

Smyth, H.F., Carpenter, C.P., Weil, C.S., Pozzani, U.C. (1954): Range-finding toxicity data, List V. Arch Indust Hyg Occup Med, **10**: 61-68

Stolzenberg, S.J., Hine, C.H. (1979): Mutagenicity of halogenated and oxygenated three-carbon compounds. J Toxicol Environ Health, **5**: 1149-1158

Stoye, D., Funke, W., Hoppe, L., Hasselkus, J., Curtis, L., Hoehne, K., Zech, H.-J., Heiling, P., Yamabe, M. (1998): Paints and Coatings – Production Technology. In: Ullmann's Encylopedia of Industrial Chemistry, Sixth Ed., Edition Release, Wiley VCH, Weinheim

Taylor, J.M., Jenner, P.N., Jones, W.I. (1964): A comparison of the toxicity of some allyl, propenyl, and propyl compounds in the rat. Toxicol Appl Pharmacol, **6**: 378-387

Union Carbide Corporation (1991), Safety data sheet "n-Propanol"

Union Carbide Corp. (1992): N-propyl alcohol (n-propanol): acute inhalation toxicity in rats. Bushy Run Research Center; Project Report 54-48 (BRRC No. 90-13-40281)

Vaishnav, D.D., Boethling, R.S., Babeu, L. (1987): Quantitative structure-biodegradability relationships for alcohols, ketones and alicyclic compounds. Chemosphere 16/4, 695-703

van Veen, M.P., Fortezza, F., Bloemens, H.J.Th., Kliest, J.J. (1999): Indoor air exposure to volatile compounds emitted by paints: Model and experiment. J. Expo Anal Epidem **9**, 569-574

von der Hude, W., Scheutwinkel, M., Gramlich, U., Fissler, B., Basler, A. (1987): Genotoxicity of three-carbon compounds evaluated in the SCE test in vitro. Environ Mutagen, **9**: 401-410

von der Hude, W., Behm, C., Gürtler, R., Basler, A. (1988): Evaluation of the SOS chromotest. Mutation Res, **203**: 81-94

Wakabayashi, K.A., Kayo, A., Popinigis, J. (1991): Effects of alkyl alcohols and related chemicals in rat liver structure and function. Acta Pathol Jap, **41**: 405-413

Wallington T.J. and Kurylo M.J. (1987): The gas phase reaction of hydroxyl radicals with a series of aliphatic alcohols over the temperature range 240 - 440 K. Intern. J. Chem. Kinet. 19, 1015 - 1023

Wehner, H.D., Schieffer, M.C. (1989): Eliminationseigenschaften des Begleitstoffes n-Propanol. Blutalkohol, **26**: 28-41

Weissermel K., Arpe H.-J. (1988): Synthesen mit Kohlenmonoxid. In: Industrielle organische Chemie, bedeutende Vor- und Zwischenprodukte. 3. Auflage, 133-145, VCH Verlagsgesellschaft mbH, Weinheim

Welke, B., Ettlinger, K., Riederer, M. (1998): Sorption of Volatile Organic Chemicals in Plant Surfaces. Environ. Sci. Technol. **32**: 1099 – 1104

Wilhoit, R.C., Zwolinski, B.J. (1973), J. Phys. Chem. Reference Data 2 (Suppl.1) 1-1, 1-5, 1-66, 1-68 bis 1-72, 1-76, 1-389 bis 1-408

Yaws, C.L., Yang, H-C., Hopper, J.R., Hansen, K.C. (1990), Chemical Enginieering, 7, 116

The report provides the comprehensive risk assessment of the substance Propan-1-ol. It has been prepared by Germany in the frame of Council Regulation (EEC) No. 793/93 on the evaluation and control of the risks of existing substances, following the principles for assessment of the risks to man and the environment, laid down in Commission Regulation (EC) No. 1488/94.

The evaluation considers the emissions and the resulting exposure to the environment and to human populations in all life cycle steps. The scenarios for occupational exposure, consumer exposure and humans exposed via the environment have been examined and the possible risks have been identified.

While there is no concern for any environmental endpoint, further testing is required for all categories under Human Health, including for humans exposed via the environment. Besides, there is concern for both workers and consumers.