European Chemicals Bureau

Institute for Health and Consumer Protection

European Chemicals Bureau

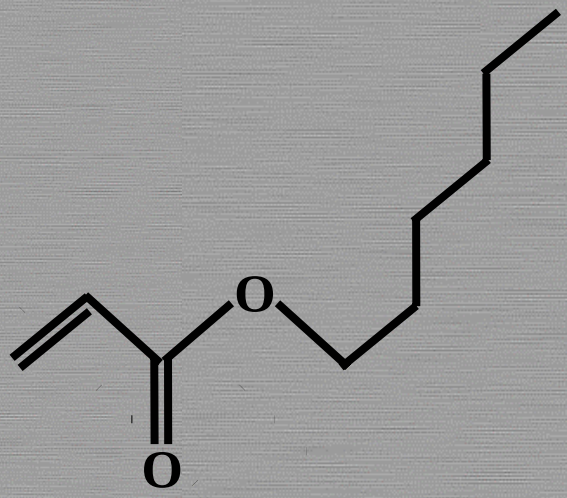
Existing Substances

European Union Risk Assessment Report

CAS No: 103-11-7

EINECS No: 203-080-7

2-ethylhexyl acrylate



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European Union Risk Assessment Report

2-ETHYLHEXYL ACRYLATE

CAS No: 103-11-7

EINECS No: 203-080-7

RISK ASSESSMENT

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RISK ASSESSMENT

Final Report, 2005

Germany

The risk assessment of edetic acid has been prepared by Germany on behalf of the European Union.

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This document is the revised draft of the Comprehensive Risk Assessment Report **2-ethylhexyl acrylate**, a substance chosen from the EU 1st Priority List in 1994.

Calculations are elaborated in Appendices A1 - A10.

Date of Last Literature Search:	**2003**
Review of report by MS Technical Experts finalised:	**2002**
Final report:	**2004**

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.

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
Acting Director-General
DG Joint Research Centre

Catherine Day
Director-General
DG Environment

[1] O.J. No l

[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: 103-11-7
EINECS No: 203-080-7
IUPAC Name: 2-ethylhexyl acrylate

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.

2-ethylhexyl acrylate represents, based on the present data configuration, no risk to the environment.

There is therefore at present no need for further testing or gathering of exposure information.

Human Health

Human Health (toxicity)

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.

Workers

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

The risk assessment reveals concern with regard to local effects after repeated inhalation for the formulation of preparations (Scenario 2).

Skin sensitisation gives rise to concern for dermal exposure during production and polymerisation (Scenario 1), the formulation of preparations (Scenario 2) and the use of formulations containing monomeric 2-EHA in the building trade (Scenario 3)

Humans exposed 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.

Human Health (risks from physico-chemical properties)

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

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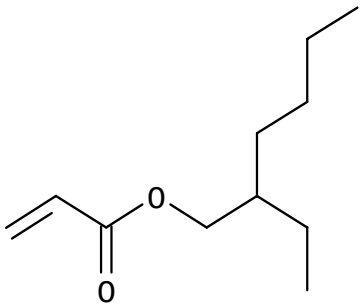
1 GENERAL SUBSTANCE INFORMATION

1.1 IDENTIFICATION OF THE SUBSTANCE

CAS No:	103-11-7
EINECS No:	203-080-7
IUPAC Name:	2-ethylhexyl acrylate
Synonyms:	Acrylic acid 2-ethylhexyl ester, 2-Ethylhexylprop-2-enoate, 2-Propenoic acid 2-ethylhexylester
Empirical formula:	$C_{11}H_{20}O_2$
Molecular weight:	184.28 g/mol
Structural formula:	



1.2 PURITY/IMPURITIES, ADDITIVES

Commercial 2-Ethylhexylacrylate has a purity of > 99%.

The following impurities are possible:

2-Ethylhexylacetate
2-Ethylhexylpropionate
2-Ethylhexanol
2-Methylstyrol
Styrol
n-Butylmethacrylate
n-Butylacrylate
Methylmethacrylate
Ethylacrylate
Methacrylate
2-Ethyl-4-methylpentylacrylate
2-Ethylhexylbutyrate
2-Ethylhexylcrotonate
2-Ethylhexylether
2-Ethylhexene
n-Hexylacetate
p-Methoxyphenol
2-Ethylhexyl 3-acryloxypropionate
2-Ethylhexyl 3-(2-ethylhexoxy) propionate
Acrylic acid
Water

Table 1.1 Physico-chemical properties

Parameter	Value	Reference
Physical state	liquid at 20°C	
Melting point	-90°C	Gerhartz, 1987
Boiling point	216°C at 1,013 hPa	Stull, 1947
	134°C at 80 hPa	
Relative Density	0.887 at 20°C	Gerhartz, 1987
Vapour Pressure	533.3 hPa at 192.2°C	Stull, 1947
	133 Pa at 50°C	
	17.1 Pa at 20°C	
	12 Pa at 20°C	BASF AG, 1995b
Surface Tension	69.2 mN/m at 20°C	BASF AG, 1995a
Water Solubility	9.6 mg/l at 25°C	BASF AG, 1996
Partition Coefficient	3.67	Fujisawa and Masuhara, 1981
(logPow-value)	4.6	BASF AG, 1988
	3.9	BASF AG, 1988
	4.09	BAuA, 1997
Flash Point	82°C	Chemsafe, 1994
Auto Flammability	245°C (DIN 51 794)	Chemsafe, 1994
Flammability	non flammable	Chemsafe, 1994
Explosive Properties	not explosive	no test conducted because of structural reasons
Oxidising properties	no oxidising properties	no test conducted because of structural reasons

Remarks:

boiling point: both data are literature values; 216°C is the boiling temperature under normal pressure and 134°C is the boiling temperature under reduced pressure at 80 hPa

vapour pressure: the values at 50°C and 192.2°C are literature values; the vapour pressure at 20°C was extrapolated from these data the value of 12 Pa was used for environment section of the risk assessment

surface tension: experimental value, using OECD guideline 115 (ring method); the concentration of the used test solution was approximately 90 mg/l

water solubility: valid experimental value based on column elution analysis

partition coefficient: 3.67 is a literature value on the basis of a HPLC-method

4.6 is an experimental value, using the OECD guideline 107 (shake flask method)

3.9 is an experimental value and has been used for the calculations in the environmental section of the risk assessment

4.09 was calculated by the computer programme KOWWIN for Microsoft Windows 3.1 of the company Syracuse Research Corporation

All four values have been assessed as correctly conducted; although the logPow of 4.6 is assumed to be a runaway

1.3 CLASSIFICATION

Classification and labelling according to the 29th ATP of directive 67/548/EEC[1]:

Classification

R 37/38 Irritating to respiratory system and to skin.

R 43 May cause sensitisation by skin contact.

According to the data presented below and the criteria of Directive 67/548/EEC, 2-ethylhexyl acrylate has not to be classified as dangerous to the environment.

Concentration limits:	$c \geq 20\%$	R37/38-43
	$1\ \% \leq c < 20\%$	R 43

Labelling

S: (2-) 36/37-46

[1] Commission Directive 2004/73/EC of 29 April 2004, adapting to technical progress for the 29th 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 216, 16.06.04, p.34.

2 GENERAL INFORMATION ON EXPOSURE

2-Ethylhexyl acrylate is produced from 2-ethyl hexanol and acrylic acid by catalytic dehydratisation in a continuous process. The spent lye of the aqueous work-up is treated in a waste water treatment plant.

6 companies are known to produce or import 2-ethylhexyl acrylate within the European Union. In 1999 the total EU production volume was 70,000 tonnes/annum, the import volume was approximately 30,000 tonnes/annum and 10,000 tonnes/annum were exported.

From the actual figures available for 1999, a total amount of 90,000 tonnes/annum is estimated to be available on the European market, 32,000 tonnes of that are used as an internal intermediate and 58,000 tonnes are sold to external processing sites. Recent information obtained from industry confirmed that no significant changes of the tonnages have to be expected for 2000 and 2001.

2.1 USE

2-Ethylhexyl acrylate is used as a monomer in the chemical industry for the production of polymers and copolymers, which are mainly processed further to aqueous polymer dispersions. The polymers and polymer dispersions are used in adhesives and as binders for paints. Other applications include coatings raw materials and uses in the plastics and textiles industries.

In addition, 2-ethylhexyl acrylate is used as a monomer in construction-industry chemicals (e.g. floor coatings, road-marking substances) in concentrations between 0.1-21%.

A quantitative breakdown of the use pattern is available for Western Europe for the year 1988 (BUA Report No 88).

Assuming no significant changes in the use pattern, with a total amount of approximately 90,000 tonnes of 2-ethylhexyl acrylate available on the European market in 1999, the following application amounts are estimated:

Table 2.1 Quantitative breakdown of the use pattern of 2-ethylhexyl acrylate

Type of use	Approximate % in this application	Estimated amount in this application
adhesives raw materials	60%	54,000 tonne/annum
binders for paints	25%	22,500 tonne/annum
coatings raw materials	5%	4,500 tonne/annum
plastics industries	5%	4,500 tonne/annum
textiles industries	5%	4,500 tonne/annum

A summary of the content of 2-ethylhexyl acrylate in different products as actually presented in the Danish Product Register (no production in Denmark):

Table 2.2 Danish Product Register data recorded in March 2002

Content of 2-ethylhexyl acrylate in the product	Number of products	Quantity [tonnes/annum]
0-2%	410	< 1
2-20%	10	2
20-50%	6	13
total	426	16

The main application areas recorded are adhesives, binding agents, construction materials, surface treatment, paints, lacquers and varnishes, reprographic agents, corrosion inhibitors and fillers.

From the Norwegian Product Register it can be seen that the number of products but not the quantities of 2-ethylhexyl acrylate contained in the products had significantly increased since 1993.

Table 2.3 Norwegian Product Register data recorded from 1993 until 2000

	1993	1994	1995	1996	1997	1998	1999	2000
number of products	77	112	221	298	308	257	233	456
quantity [t/a]	286	536	546	350	493	304	354	171

A summary of the actual content of 2-ethylhexyl acrylate in different products is presented in the Norwegian Product Register from 15 February 2002:

Table 2.4 Norwegian Product Register data recorded in February 2002

Content of 2-ethylhexyl acrylate in the product	Number of products	Quantity [tonnes/annym]
0-1%	565	0.5
1-10%	1	-
10-80%	7	45
80-100%	2	126
total	575	171

125 tonnes of the total amount are attributed to raw materials, 10 tonnes to binders for paints and paints and 6 tons to other binders.

In the Swedish Product Register, a total of 26 products containing a total quantity of 544-621 tonnes of 2-ethylhexyl acrylate were identified in 1993. Seven of those products (related to three functions) were available to consumers. The highest amounts of the substance are applied in the constructing industries, the plastics industries, the paint industries and as intermediates.

3 ENVIRONMENT

3.1 ENVIRONMENTAL EXPOSURE

3.1.1 General discussion

3.1.1.1 Release into the environment

Releases of 2-ethylhexyl acrylate into the environment are expected to occur mainly during production and processing with waste water and exhaust gases.

Further releases are expected through residual monomeric acrylate-contents in the polymeric products.

According to the producer, the aqueous polymer dispersions, as the main products, contain less than 200 mg monomeric 2-ethylhexyl acrylate per kg. In addition to this, a residual monomeric content of up to 800 ppm of 2-ethylhexyl acrylate in polymer dispersions is reported, but it was confirmed by the main producer that this value is not relevant for the current situation in Europe. Therefore, 200 ppm is considered to represent a realistic worst case and is used in the further assessment.

Through storage of the polymeric products the residual monomers may partly polymerise and quantification of the releases into the environment from polymeric products can be performed only roughly.

3.1.1.2 Degradation

Hydrolysis

There are no data available about hydrolysis of 2-ethylhexyl acrylate. However, acrylic acid esters are known to hydrolyse very slowly. For example, for ethyl acrylate a half life of approximately 3.5 ears at pH 7 and 25°C is reported (Mabey and Mill, 1978). Due to sterical reasons the half life of 2-ethylhexyl- acrylate is expected to be significantly longer. The HydroWin program (SRC) estimates a half life of 17 years at pH 7. Therefore, hydrolysis is not considered a relevant degradation pathway under environmental conditions.

Biodegradation

In a MITI-I test (OECD 301C) employing sludge from different sewage treatment plants, rivers, bays and a lake as inoculum biodegradation of 51% (on the upward trend) after 14 days was obtained. Biodegradation was measured as BOD (CITI 1992).

In a manometric respirometry test conducted according to OECD guideline 301 F biodegradation (related to BOD) of 2-ethylhexyl acrylate of 70% after 15 days and 75% after 28 days was found (BASF 1991a). The 10-day window criterion was fulfilled. As inoculum domestic activated sludge was used.

In a modified OECD screening test (OECD 301 E) using filtered effluent from a domestic sewage treatment plant as inoculum biodegradation of 2-ethylhexyl acrylate of 93% after 7 days and 99% after 14 days (measured as DOC) was observed (Amann/Steinhäuser 1986).

Price et al. (1974) tested the biodegradation of 2-ethylhexyl acrylate both in fresh and salt water. In the freshwater test settled domestic wastewater was used as inoculum (3 ml/bottle). The concentration of the test substance was 3, 7 or 10 mg/l. After 20 days a BOD/TOD ratio of 30% was achieved. The same test was then repeated with acclimated inoculum. An equal-volume mixture of 2 biologically treated petrochemical effluents, settled domestic wastewater, Kanawha river water (this river receives the waste effluent from numerous industrial and domestic sources) and soil in BOD dilution water was acclimated to 2-ethylhexyl acrylate for 45-60 days. With this acclimated inoculum a BOD/TOD ratio of 40% after 20 days was achieved. The saltwater test conducted in artificial seawater was performed in the same manner as the freshwater test with exception of the seed source. The seed used in the seawater test was developed in seawater taken from Lavaka Bay. This seed source was maintained by adding small amounts of settled raw waste water about every 3 to 4 days as a source of substrate, seed bacteria and growth factors. After 20 days a BOD/TOD ratio of 35% was achieved.

Regarding the available test results 2-ethylhexyl acrylate can be classified as readily biodegradable. Although the data reported by Price et al. (1974) do not point towards ready biodegradation the results from the standardised screening tests confirm the classification as readily biodegradable.

According to the available test results, a biodegradation rate in sewage treatment plants of 1 h^{-1} is assumed. Results from biodegradation simulation tests in surface water and soil are not available and have to be estimated based on the above described tests and the partition behaviour of 2-ethylhexyl acrylate (EC, 1995).

In Appendix A1, the respective calculations are presented.

Table 3.1 Biodegradation rate constants

Compartment / medium	Biodegradation rate
activated sludge (STP)	$K_{STP} = 1\ h^{-1}$
surface water	$K_{sw} = 0.047\ d^{-1}$
sediment	$K_{sed} = 0.002\ d^{-1}$
soil	$K_{soil} = 0.023\ d^{-1}$

Photo oxidation

In the atmosphere, 2-ethylhexyl acrylate will react with the photochemically produced hydroxyl radicals and with ozone.

Based upon atmospheric concentrations of $5 \cdot 10^5$ · OH/cm^3 and $7 \cdot 10^{11}$ O_3/cm^3, the atmospheric half-life of 2-ethylhexyl acrylate has been estimated to be about 19 hours (Atkinson, 1987).

3.1.1.3 Distribution

The Henry's law constant is estimated from the water solubility of 9.6 g/m^3 and the vapour pressure of 12 Pa. The value of H = 230 Pa.m^3/mol at 20-25°C indicates, that volatilisation from surface water is rapid.

The adsorption and desorption behaviour of 2-ethylhexyl acrylate was not investigated. According to the EU Technical Guidance Document (Chapter 4), from the experimentally determined logPow of 3.9 a Koc of 9, 14 l/kg is calculated.

From this value, the partition coefficients in the different compartments can be estimated using default organic carbon contents in the different compartments.

In Appendix A1, the calculations are presented.

Table 3.2 Partition coefficients

Compartment	Partition coefficient
soil-water	K_{p_soil} = 18 l/kg
sediment - water	K_{p_sed} = 91 l/kg
suspended matter - water	K_{p_susp} = 91 l/kg

Using the fugacity model of Mackay (level 1), the theoretical distribution at equilibrium can be estimated. About 97% of the total amount of 2-ethylhexyl acrylate is expected to be distributed to the atmosphere and about 1% is allocated to surface water. Less than 1% is expected to end up in each soil and sediment.

Based on the physical chemical properties of 2-ethylhexyl acrylate, the atmosphere is the main target compartment for distribution and only small amounts remain in the hydrosphere.

Elimination in the sewage treatment plants

Based on the above cited physical chemical properties (log H = 2.36 ; log Pow = 3.9), as well as the biodegradation rate of 1 h^{-1} in STP, the elimination through biodegradation and distribution can be estimated with the model SIMPLETREAT:

Table 3.3 Elimination and distribution in STPs

% to air	29.8
% to water	7.0
% to sludge	7.5
% degraded	55.8
% removal	93.0

From measurements of the influent and effluent concentration in an industrial sewage treatment plant, a similar elimination-rate can be estimated. Using the detection limit as effluent concentration because no 2-ethylhexyl acrylate was detected in the effluent and the 90 percentile of the measured influent concentrations, an elimination-rate of 90% is calculated (BASF, 2000). For further calculations, the removal rate estimated with the SIMPLETREAT model is used.

3.1.1.4 Accumulation

There are no experimental results on bioaccumulation available. The log Pow of 3.9 indicates a moderate potential for bioaccumulation though.

According to the Technical Guidance Documents (EC 1995), the BCF for fish can be estimated from the log Pow using the method developed by Veith et al. (1979). For 2-ethylhexyl acrylate a BCF of 4,12 $l/kg_{wet\ fish}$ is calculated.

The estimated Koc-value of 9,14 l/kg also indicates moderate potential for geoaccumulation. It is not expected, that considerable amounts of 2-ethylhexyl acrylate released to soil may leach with rain water to the groundwater.

3.1.2 Aquatic compartment

For the estimation of the local PECs, a total production volume of 70,000 tonnes/annum is assumed. With an import volume of approximately 30,000 tonnes/annum and approximately. 10,000 tonnes/annum that are exported a total amount of about 90,000 tonnes/annum is estimated to be available on the European market.

3.1.2.1 Estimation of PEC_{local}/Generic approach: production and processing

In the Technical Guidance Documents (EC 1995), a generic (i.e. non site-specific) exposure scenario ("emission scenario document") for the release into surface water of intermediates during production and processing is proposed. The following scenario reflects a worst case situation:

The total production quantity in the EU of 70,000 tonnes/annum and the total internal processing volume of 32,000 tonnes/annum is used for the generic calculation. For production an emission factor of 0.3% is proposed and for processing a generic release estimate of 0.001% for wet polymerisation process is used resulting in a worst case estimation of a PEC_{local} of 9.5 µg/l (for calculations see Appendix 2).

3.1.2.2 Estimation of PEC_{local} / Site-specific approach: production and processing

Using the available specific data for the production and processing sites, more precise PEC-estimations can be performed.

Table 3.4 Site specific release estimation

Company	$C_{local\ water}$ [µg/l]	Release [tonnes/annum]	Specific data
A	0.13	2.56	flow rate of receiving river, flow rate of STP; actual release estimated on the basis of effluent measurements;
B	0.005	0.007	processing volume, no further specific data;

From the confidential data provided to the rapporteur it is known that for site A a reliable PEC estimation based on site specific information was performed that is representative for production and internal processing in Europe.

Site B represents a realistic worst case situation for wet polymerisation of 2-ethylhexyl acrylate at external processing sites. The approximate number of external sites and the size of the biggest external sites are known to the rapporteur. From this a processing volume of 10,000 tonnes/annum is assumed and a default release estimate has been performed (see Appendix 3).

3.1.2.3 Estimation of PEC_{local}/Generic approach: use

a) Formulation of aqueous polymer dispersions

The release of monomeric 2-ethylhexyl acrylate is possible during formulation of adhesives, paints and other polymeric products. Especially for adhesives, generalising assumptions are difficult to make and there is no “emission scenario document” available at the moment for the mentioned applications.

Due to the lack of specific data it is assumed, that from the total amount of 90,000 tonnes 2-ethylhexyl acrylate aqueous polymer dispersions are obtained and formulated. The emissions are estimated with the “worst case” emission tables presented in Appendix I of the Technical Guidance Documents.

Based on information from industry it is assumed that from 90,000 tonnes/annum 2-ethylhexyl acrylate approximately 210,000 tonnes of aqueous based polymers are obtained containing 200 ppm (42 tonnes/annum) residual monomeric 2-ethylhexyl acrylate.

A generic exposure assessment for the formulation-stage is performed assuming a fraction of main source of 0.4 (Table B 2.3) and a release factor of 0.3% (Table A 2.1). From the calculation elaborated in Appendix 4 as a result a $C_{local\ water}$ of 0.6 µg/l is obtained for formulation.

This estimation is considered a worst case scenario. But as there are no information available indicating that the formulation of 2-ethylhexyl acrylate based polymer dispersions is wide disperse throughout Europe it is judged appropriate to use the TGD defaults.

b) Processing/use of water based adhesives and paints

Sufficient information for a reliable estimation of the releases of monomeric 2-ethylhexyl acrylate from the processing and use of adhesives raw materials is not available.

Aqueous polymer dispersions are understood to be the main product type for both, adhesives and paints. Therefore, in a first approach it is suggested, that the releases during processing and use can be estimated for both application areas accordingly.

Assuming that from approximately 76,500 tonnes 2-ethylhexyl acrylate (54,000 tonnes for adhesives and 22,500 tonnes for paints, see Section 2) approximately. 178,500 tonnes of water-based dispersions containing 200 ppm residual monomers are obtained, approximately 35.7 tonnes of monomeric 2-ethylhexyl acrylate are annually handled.

The estimation is performed using the A/B-tables for paints (IC 14, UC 10) proposed in Annex 1 of the Technical Guidance Documents. In Table A 3.15/A 4.5 a fraction of emission of 0.5% is

proposed and Table B 3.13/B 4.5 provides a fraction of main source of 0.05. The respective calculations elaborated in Appendix 5 result in a $C_{local\ water}$ of 0.1 µg/l.

The releases of 2-ethylhexyl acrylate into the aquatic environment through the private use of adhesives and paints are not relevant on a local scale due to the smaller amounts used.

c) Paper recycling

As 2-ethylhexyl acrylate-based polymers are used for coatings, paints and printing inks the residual monomers may be released during the paper recycling process.

According to the use pattern presented in Section 2, in a first approach it is assumed that 10% of the total amount available on the European market is processed to aqueous polymer dispersions used in the paper industry. An annual tonnage of 21,000 tonnes of those dispersions are assumed containing 200 ppm (4.2 tonnes) residual monomeric 2-ethylhexyl acrylate.

A PEC-estimation according to the "emission scenario document" proposed in the Technical Guidance Documents (EC, 1995) leads to a $C_{local\ water}$ of 0.5 µg/l (see Appendix 6).

3.1.2.4 Monitoring data

No relevant data on measured aquatic concentrations are available.

3.1.2.5 Sediment

Neither monitoring data on concentrations of 2-ethylhexyl acrylate in sediment nor experimental results with benthic organisms are available. A quantitative risk assessment using the equilibrium partitioning method proposed in the TGD seems not necessary for this substance as no information beyond those available for the water compartment can be obtained.

3.1.3 Atmosphere

3.1.3.1 Estimation of PEC_{local} at production and processing

No emission scenario document for the release into the atmosphere of intermediates during production and processing is available at the moment. The emissions can therefore be estimated with the emission tables presented in Appendix I of the Technical Guidance Documents.

However, specific data are available for the main production site from 1989, so that the PEC-calculation can be performed with these data. For external processing a default calculation is performed assuming a processing volume of 10,000 tonnes/annum and a fraction of emission of 0.1% (Table A 3.10). In a generic scenario for the formulation of aqueous polymer dispersions a fraction of emission of 0.5% (Table A 2.1) and a fraction of main source of 0.4 (Table B 2.3) are applied.

The calculations are presented in Appendices A7.1, A7.2 and A7.3 and the results are summarised in **Table 3.5**.

Table 3.5 Atmospheric release estimation

Scenario	Release[tonnes/annum]	PEC_{local} (air) [µg/m³]	$DEP_{total\ ann}$ [µg·m⁻²·d⁻¹]
production site, specific data	0.18 (direct) 10.9 (via STP)	8.3	9.1
external processor, default	10 (direct) 0.03 (via STP)	7.6	8.2
formulation of polymer dispersions	0.08 (direct) 0.1 (via STP)	0.1	0.2

Releases to the atmosphere during the use of the aqueous based polymeric products made from 2-ethylhexyl acrylate are significantly lower than the scenarios considered above.

3.1.4 Terrestrial compartment

The release of 2-ethylhexyl acrylate to soil is expected to occur through atmospheric deposition after local release to the atmosphere at the production and processing sites. The input through sludge application on agricultural soil is considered to be of minor relevance, because industrial sludge is incinerated and from the use pattern of the substance (predominantly polymeric material is handled containing only small amounts of residual monomers), considerable amounts of 2-ethylhexyl acrylate in municipal sewage sludge are not expected.

With the annual deposition rates calculated above, equilibrium soil concentrations in the vicinity of the plants are calculated according to the EU Technical Guidance Document (EC, 1995). The detailed calculation is presented in Appendices A8.1, A8.2 and A8.3:

Table 3.6 Local exposure of the soil compartment

	Exposure of the ecosystem	Exposure of grassland	Exposure of agricult. soil
site A: bulk soil and porewater concentration	PEC_{local} = 0.85 µg/kg ww $PEC_{local\ pw}$ = 0.05 µg/l	PEC_{local} = 1.34 µg/kg ww $PEC_{local\ pw}$ = 0.08 µg/l	PEC_{local} = 0.85 µg/kg ww $PEC_{local\ pw}$ = 0.05 µg/l
processing site: bulk soil and porewater concentration	PEC_{local} = 0.77 µg/kg ww $PEC_{local\ pw}$ = 0.05 µg/l	PEC_{local} = 1.21 µg/kg ww $PEC_{local\ pw}$ = 0.07 µg/l	PEC_{local} = 0.77 µg/kg ww $PEC_{local\ pw}$ = 0.05 µg/l
formulation site: bulk soil and porewater concentration	PEC_{local} = 0.014 µg/kg ww $PEC_{local\ pw}$ = 0.0009 µg/l	PEC_{local} = 0.022 µg/kg ww $PEC_{local\ pw}$ = 0.0014 µg/l	PEC_{local} = 0.014 µg/kg ww $PEC_{local\ pw}$ = 0.0009 µg/l

3.1.5 Non compartment specific exposure relevant to the food chain

3.1.5.1 Secondary poisoning

For 2-ethylhexyl acrylate a moderate bioaccumulation potential is expected. Therefore, an exposure assessment for secondary poisoning is required. Using the calculated BCF for fish (see Section 3.1.1) of 4,12 $l/kg_{wet\ fish}$ for the calculation and assuming that 50% of the diet comes from a source using the highest local concentration in surface water and 50% using $PEC_{regional}$, the following $PEC_{oral,\ fish}$ can be estimated:

$PEC_{oral\ fish} = 0.5 \cdot (0.6\ \mu g/l \cdot 4{,}12\ l/kg + 0.006\ \mu g/l \cdot 4{,}12\ l/kg) = 125\ \mu g/kg_{wet\ fish}$

3.1.6 Regional concentrations

For the estimation of the regional background concentrations, all releases, from diffuse as well as point sources should be taken into account. From the total release volume it is recommended to use 90% in the continental model and 10% in the defined EU-standard regional model. However, for 2-ethylhexyl acrylate it is known that the main fraction of production and internal processing and a considerable amount of external processing takes place within one region in Europe. Only approximately 48,000 tonnes/annum are available for external processing elsewhere in Europe. Therefore, for modelling purpose it is assumed that site A and site B are located in the same region and that an external processing volume of 48,000 tonnes/annum is allocated to the continent.

Point source releases to the aquatic compartment:

Based on the actual release data provided by the producers (see Section 3.1.2.2.) and the default releases estimated for the external processing sites (0.001% emission for wet polymerisation, 7% directed to surface water after elimination in STP), the total release amounts are summarised in the **Table 3.7**. The releases through the industrial use of products manufactured from 2-ethylhexyl acrylate are taken into account below (diffuse releases).

Table 3.7 Point sources releases to hydrosphere

Point source	Regional releases [tonnes/annum]		Continental releases [tonnes/annum]	
	To surface water	To STP	To surface water	To STP
site A	2.56	36.6	-	-
site B	0.007	0.1	-	-
other processing sites	-	-	0.03	0.48

Point source releases to air:

Using the same approach as described above for the aquatic compartment the releases to air from production (specific data) and external processing (default releases, 0.1% direct releases and 29.8% of the releases via STP) are estimated:

Table 3.8 Point sources releases to atmosphere

Point source	Rregional releases [tonnes/annum]		Continental releases [tonnes/annum]	
	Direct	Via STP	Direct	Via STP
site A	0.18	10.9	-	-
site B	10	0.03	-	-
other processing sites	-	-	48	0.14

Point source releases to soil:

No direct releases to soil from point sources were identified.

Diffuse releases:

Diffuse releases occur from residual 2-ethylhexyl acrylate in the polymeric products.

As the main product-type app. 210,000 tonnes/annum aqueous polymer dispersions are obtained containing about 200 ppm (42 tonnes/annum) residual monomeric 2-ethylhexyl acrylate.

As an initial worst case approach, it is assumed that 50% of the monomers (21 tonnes/annum) may leach into the hydrosphere during the whole life-cycle of the products. Diffuse releases via municipal STPs from professional and private use of the products are thought to be already included in this assumption, 30% of these releases are assumed to be directly to surface water, 70% via STP. 10% of the monomers (4.2 tonnes/annum) are assumed to be evaporated to the atmosphere during the whole life-cycle of the products. Through storage of the polymeric products the residual monomers may partly polymerise and quantification of the releases can only be regarded as a rough estimate.

Table 3.9 Diffuse releases

Diffuse releases	Regional releases [tonnes/annum]	Continental releases [tonnes/annum]
Directly to surface water	0.63	5.67
To STP	1.47	13.23
To air	0.42	3.78

The regional and continental PECs were calculated according to EUSES (see Appendix A 9). In **Table 3.10** an overview is given of all the releases considered as input for the model calculation.

Table 3.10 Total releases considered on regional and continental scale

Releases	Regional releases [tonnes/annum]	Continental releases [tonnes/annum]
Directly to surface water	0.63	5.67
To STP	38.17	13.71
Directly to air	10.6	51.78

The results of the calculations are compiled below :

$PECregional_{aquatic} = 5.8 \cdot 10^{-3}$ µg/l

$PECregional_{soil} = 8.1 \cdot 10^{-5}$ µg /kg ww

$PECregional_{air} = 7.9 \cdot 10^{-4}$ µg /m^3

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

3.2.1 Aquatic compartment (incl. sediment)

3.2.1.1 Toxicity test results

3.2.1.1.1 Fish

Juhnke and Lüdemann (1978) examined the effects of 2-ethylhexyl acrylate in fish in a short-term study. As test organism *Leuciscus idus* was used. The aim of the study was to compare the reproducibility of the standard test method DIN 38 412 L 15 that was new at that time. Fish were exposed in a static system for 48 hours. Apart from the shorter exposure time the method is comparable to the OECD Guideline 203. A 48-hour LC_{50}-value of 23 mg/l was found. The corresponding LC_0 and LC_{100} value was 9 mg/l and 45 mg/l. All values are nominal concentrations. As the effect concentrations exceed the water solubility of 9.6 mg/l for 2-ethylhexyl acrylate and as the possible decrease in test concentration by volatilisation of the substance was not considered, the test is regarded as invalid.

In a semi-static test BASF (1999) studied the acute toxicity of 2-ethylhexyl acrylate in the rainbow trout *Oncorhynchus mykiss.* The test was performed according to OECD Guideline 203 "Fish acute toxicity test" including the updated version of July 1992. Fish were exposed for 96 hours with daily replacement of the test water. Six test concentrations ranging nominally from 0.681 mg/l to 4.64 mg/l were used. The following effect values related to the analytically detected concentrations (mean values of the detected concentrations after 1, 24, 48 and 96 hours) are reported:

Table 3.11 Acute toxicity in rainbow trout

Effect concentration	Effect	Nominal concentration
96-hour LC_{50} = 1.8 mg/l*	mortality	2.15 mg/l < 96-hour LC_{50} < 3.16 mg/l
96-hour NOEC = 1.49 mg/l	mortality	2.15 mg/l

* Calculated as geometric mean from 1.49 mg/l < 96-hour LC_{50} < 2.19 mg/l

3.2.1.1.2 Invertebrates

In a short-term test with *Daphnia magna* conducted according to EEC guideline a 48-hour EC_{50} of 17 mg/l was found. The EC_{50}-value after 24 hours was 50 mg/l. Tween 80 was used as solubiliser (BASF 1989). The given effects values are related to nominal concentrations that significantly exceed the water solubility of 9.6 mg/l for 2-ethylhexyl acrylate. The possible decrease in test concentration by volatilisation of the substance was not considered and the test is regarded as invalid.

In addition, other static tests are available on *Daphnia magna* (Bringmann/Kühn 1982) and on the brine shrimp *Artemia salina* (Price et al. 1974) where only nominal concentrations are reported that are invalid and not suitable for risk assessment purpose.

BASF (2001) studied the effects of short-term exposure of *Daphnia magna* to 2-ethylhexyl acrylate according to OECD Guideline 202. The test was performed in a static and closed system in complete darkness. Darkness was chosen because of the instability of the pure substance against light (radical induced polymerisation). However, it is believed that this is not relevant for the aqueous solution and has not affected the test results. Six test concentrations ranging nominally from 3.13 mg/l to 100 mg/l were employed. The effect values were related to measured concentrations and are given below:

Table 3.12 Acute toxicity in Daphnia magna

Effects concentration	Nominal concentration
48-hour EC_0 = 0.7 mg/l	25 mg/l
48-hour EC_{50} = 1.3 mg/l	46.3 mg/l
48-hour EC_{100} = 2.8 mg/l	100 mg/l

3.2.1.1.3 Plants

In a test conducted according to DIN 38 412 L 9 the toxicity of 2-ethylhexyl acrylate to the green algae *Scenedesmus subspicatus* was examined (BASF 1990). Cremophor RH 40 was used as solubiliser. Test parameter was the growth inhibition of the algae measured as chlorophyll-a-fluorescence. The following effect values related to growth rate (R) and biomass (B) were found:

Table 3.13 Toxicity in algae (nominal concentrations only)

Effects on growth rate	Effects on biomass
72-hour E_RC_{10} = 30 mg/l	72-hour E_BC_{10} = 23 mg/l
72-hour E_RC_{50} = 67 mg/l	72-hour E_BC_{50} = 44 mg/l
96-hour E_RC_{10} = 40 mg/l	96-hour E_BC_{10} = 24 mg/l
96-hour E_RC_{50} = 67 mg/l	96-hour E_BC_{50} = 47 mg/l

Again, the nominal concentrations exceed significantly the water solubility and the possible decrease in test concentration by volatilisation of the substance was not considered. The test is therefore regarded as invalid.

In a 72-hour static test conducted according to EEC Directive 92/69/EEC and OECD Guideline 201 the acute toxicity of 2-ethylhexyl acrylate to the green algae *Desmodesmus subspicatus* was examined (BASF 2002). The test was performed in a closed system in the nominal concentration range between 3.13 and 100 mg/l. Growth inhibition of the algae was measured as chlorophyll-a-fluorescence. Effect data were related to the measured concentrations.

Table 3.14 Toxicity in algae (measured and nominal concentrations)

Effects on growth rate	Effects on biomass
72-hour E_RC_{10} = 0.8 mg/l (nominal: 11.1 mg/l)	72-hour E_BC_{10} = 0.55 mg/l (nominal: 7.62 mg/l)

72-hour E_RC_{50} = 1.71 mg/l (nominal: 23.7 mg/l)	72-hour E_BC_{50} = 1.17 mg/l (nominal: 16.3 mg/l)
72-hour E_RC_{90} = 2.91 mg/l (nominal: 40.4 mg/l)	72-hour E_BC_{90} = 2.38 mg/l (nominal: 33.1 mg/l)

In a growth inhibition test conducted with *Scenedesmus quadricauda* Bringmann and Kühn (1977, 1978) obtained an 8-day-TGK-value of > 1 mg/l. The TGK (toxic threshold concentration) corresponds to an EC_3.

With the blue-green algae *Microcystis aeruginosa* as test organism Bringmann and Kühn (1978) found in the growth inhibition test an 8-day-TGK of 0.06 mg/l. Also in this study the TGK was equivalent to an EC_3 and was based on nominal concentrations.

In both studies no analytical monitoring was performed and it has to be expected that the algae were not in the exponential growth phase during the whole test duration. Both studies are regarded as invalid.

3.2.1.1.4 Microorganisms

The toxicity of 2-ethylhexyl acrylate to different microorganisms was examined by Bringmann and co-workers using growth inhibition tests. The following test results were obtained:

Table 3.15 Toxicity in microorganisms

Pseudomonas putida	16-hour TGK(EC_3) > 1 mg/l	Bringmann/Kühn 1977
Entosiphon sulcatum	48-hour TGK (EC_5) > 10 mg/l	Bringmann 1978
Chilomonas paramaecium	48-hour TGK (EC_5) = 2.3 mg/l	Bringmann et al. 1980

In a test with domestic activated sludge the inhibition of oxygen uptake was examined according to guideline OECD 209 (BASF 1991b). After 30 minutes oxygen uptake was inhibited by 7% at 1,000 mg/l, the highest concentration tested. As no reference substance was tested and therefore, the sensitivity of the activated sludge is unknown, the test result should be used with care.

In a test according to DIN 38 412 L 27 the inhibition of oxygen uptake for *Pseudomonas putida* exposed to 2-ethylhexyl acrylate for 30 minutes was studied (BASF 1991c). Tween 80 was used as solubiliser. At the highest tested concentration of 10,000 mg/l no inhibition of oxygen uptake was found.

In addition to the data reported above, other test results are available, but due to missing information on test conditions they could not be checked on validity.

For microorganisms only nominal concentrations are reported and the possible decrease in test concentrations by volatilisation of the substance was not considered. In most studies the reported effect concentrations exceed significantly the water solubility of 2-ethylhexyl acrylate. Therefore, only the results reported for the protozoan species *Chilomonas paramaecium* may be considered suitable for risk assessment purpose.

3.2.1.1.5 Determination of $PNEC_{aqua}$

Due to the moderate volatility and low water solubility (9.6 mg/l) of 2-etylhexyl acrylate only effect values based on analytically measured concentrations should be used for the derivation of the PNEC. Such results are available from three acute tests conducted under standardised

conditions. The relevant LC_{50}-/EC_{50}-values range from 1.3 mg/l (daphnids) to 1.8 mg/l (fish). The most sensitive species was *Daphnia magna* showing a 48-hour EC_{50} of 1.3 mg/l.

Short-term tests with species from three trophic levels are available; therefore an assessment factor of 1,000 is applied to this value.

Therefore: $PNEC_{aqua}$ = 1.3 mg/l / 1,000 = 1.3 µg/l

3.2.1.1.6 Determination of $PNEC_{microorganisms}$

The most sensitive microorganism to 2-ethylhexyl acrylate was the protozoan *Chilomonas paramaecium* with a 48-hour TGK of 2.3 mg/l. Although this species does not influence the degradation processes itself, it is necessary for a proper function of a WWTP. For this kind of test result an assessment factor of 1 is proposed for the determination of $PNEC_{microorganism}$.

Therefore: $PNEC_{microorganism}$ = 2.3 mg/l / 1 = 2.3 mg/l

3.2.1.1.7 Sediment

There are no experimental results with benthic organisms available. The $PNEC_{sed}$ can be provisionally calculated using the equilibrium partitioning method. However, a quantitative risk assessment is not deemed necessary for 2-ethylhexyl acrylate as no information beyond those available for the water compartment can be obtained and the substance is neither released nor distributed to sediments in significant amounts.

3.2.2 Atmosphere

Data on biotic or abiotic effects in the atmosphere are not available. Because of the short half-life of 2-ethylhexyl acrylate in the atmosphere (about 19 hours) adverse effects are not to be expected.

3.2.3 Terrestrial compartment

Data on effects to terrestrial organisms are not available.

In an indicative risk assessment for the soil compartment, the aquatic PNEC will be used and compared to the concentration in soil pore water:

$PNEC_{soil}$ =1.3 µg/l (soil pore water)

3.2.4 Non compartment specific effects relevant to the food chain (secondary poisoning)

Because 2-ethylhexyl acrylate has a log Kow > 3 there is an indication of bioaccumulation potential. To evaluate whether the substance may cause toxic effects if accumulated in higher organisms the classification on the basis of mammalian toxicity data can be used.

2-Ethylhexyl acrylate is not classified as Very Toxic or Toxic or Harmful and there are no adequate data from dietary toxicity tests which can be used for the determination of $PNEC_{oral}$. Therefore a quantitative assessment of secondary poisoning can not be performed but improvement of the data basis is not considered to be of high priority for 2-ethylhexyl acrylate.

3.3 RISK CHARACTERISATION

3.3.1 Aquatic compartment (incl. sediment)

3.3.1.1 Water

3.3.1.1.1 Waste water treatment plants

An evaluation of the inhibition to microorganisms in WWTPs would seem most relevant for those situations where 2-ethylhexyl acrylate containing waste water is released to domestic treatment plants. Excluding therefore the production sites which are known to have their own industrial treatment plant, the effluent concentration calculated for the formulation of aqueous polymer dispersions is used for the initial assessment.

Therefore: $PEC_{microorganisms}$ = 6 µg/l

With a $PNEC_{microorganisms}$ of 2.3 mg/l, the PEC/PNEC ratio amounts to 0.003 and therefore a risk to microorganisms in WWTPs is not to be expected.

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.

3.3.1.1.2 Surface waters

In **Table 3.16** the comparison between PEC and PNEC (1.3 µg/l) for all relevant exposure scenarios are presented.

Table 3.16 PEC/PNEC ratios for surface water

Scenario	C_{local} + $PEC_{regional}$ = PEC_{local} µg/l]	PEC/PNEC
production and processing: site A site B	 0.13 + 0.006 = 0.14 0.005 + 0.006 = 0.01	 0.1 0.008
formulation of aqueous polymer dispersions	0.6 + 0.006 = 0.6	0.5
processing/use of water based adhesives and paints	0.1 + 0.006 = 0.1	0.08
paper recycling	0.5 + 0.006 = 0.5	0.4

As for all exposure scenarios PEC/PNEC < 1, a risk for the aquatic compartment of the environment is not deduced for the present data configuration.

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.

3.3.1.1.3 Sediment

Neither monitoring data on concentrations of 2-ethylhexyl acrylate in sediment nor experimental results with benthic organisms are available.

A quantitative risk assessment based on the equilibrium partitioning method on the effects and the exposure side is not necessary as no information beyond those available for the water compartment can be obtained.

From the results for the water phase it can be concluded that no further testing has to be recommended for the sediment compartment because 2-ethylhexyl acrylate is neither released nor distributed to sediments in significant amounts.

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.

3.3.2 Atmosphere

Due to the short atmospheric lifetime ($t_{1/2}$ = 19 hours), biotic or abiotic adverse effects upon the atmosphere are not expected from 2-ethylhexyl acrylate.

Therefore, qualitatively, no risk is deduced for this compartment.

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.

3.3.3 Terrestrial compartment

A site specific exposure scenario representing a worst case situation for production, processing and use of 2-ethylhexyl acrylate was used for a PEC calculation. Due to atmospheric deposition in the vicinity of this site, the concentration in the soil porewater is expected to be $PEClocal_{porewater}$ = 0.08 µg/l. The regional background concentration is considered to be negligible. An indicative risk assessment can be performed with the aquatic PNEC:

PEC/PNEC = 0.08 / 1.3 = 0.06

As PEC/PNEC < 1, a risk for the soil compartment is not identified.

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.

3.3.4 Non compartment specific effects relevant to the food chain (secondary poisoning)

As 2-ethylhexyl acrylate does present indications of a bioaccumulation potential, a risk characterisation for secondary poisoning seems opportune.

A $PEC_{oral,\ fish}$ of 0.1 $mg/kg_{wet\ fish}$ had been calculated (see Section 3.1.5). However, no adequate data from dietary toxicity tests for the determination of a PNEC are available.

2-Ethylhexyl acrylate is not classified as Very Toxic or Toxic or Harmful. Therefore, qualitatively no risk is identified for secondary poisoning and improvement of the data basis seems not of high priority.

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 HUMAN HEALTH

4.1 HUMAN HEALTH (TOXICITY)

4.1.1 Exposure assessment

4.1.1.1.1 General discussion

2-Ethylhexyl acrylate (2-EHA) is mainly used as a monomer in the chemical industry for the manufacture of polymeric chemicals, which are processed further to aqueous polymer dispersions (approximately 50% polymer). The polymers and polymer dispersions are used in different products e. g. in adhesives, in printing inks and as binders in paints (see Section 4.1.1.2).

According to information provided by the manufacturers, aqueous polymer dispersions may contain residual monomer contents of 0.02% 2-EHA (BUA, 1991). In latex coatings, for instance, residual 2-EHA concentrations are generally 0.08% or less (BAMM, 1993).

In addition, monomeric 2-EHA is an additive in preparations, which are applied in the building trade as floor coatings and road-marking materials. The concentration of monomeric 2-EHA amounts up to 21%.

For workers the inhalative and dermal exposure routes are the most likely.

According to the Swedish product register, 2-EHA is used e.g. in lubricants/greases. The consumer products are offered in wholesale and retail trade, e.g. in repair shops for cars and motor vehicles, as products for personal and household use (as per February 1995) and in agriculture.

Consumers use e.g. dispersion paints or lubricants and greases which may contain 2-EHA as a residual monomer. Thus, the consumer may be exposed to 2-EHA via the inhalatory and dermal routes.

4.1.1.2 Occupational exposure

The exposure assessment generally aims at assessing exposure levels representing the reasonable worst case situation. The reasonable worst case is regarded as the level of exposure which is exceeded in a small percentage of cases over the whole spectrum of likely circumstances of use for a specific scenario.

The assessment of inhalation exposure is mainly based on measured exposure levels from which, if possible, 90^{th} or 95^{th} percentiles 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 of exposure transparent.

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-member states, within the framework of existing substances, 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 importantly, 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 protective 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 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.

Industrial activities using monomeric 2-EHA present opportunities for exposure. Exposure ranges depend on the particular operation and the risk reduction measures in use. Concerning dermal exposure, on account of the highly irritative effect of 2-EHA and preparations containing > 21% of the substance (see Section 4.1.3.2), workers avoid immediate dermal contact to a large extent by using PPE (here gloves) and by applying appropriate working techniques.

There is only one occupational exposure limit for 2-EHA in Germany, amounting to about 82 mg/m^3 (10 ml/m^3), which may not be exceeded even short-term (15-minute average) (TRGS 900, 1996).

The odour threshold levels for monomeric 2-EHA which are described in the literature amount to between 0.55 mg/m^3 (0.07 ml/m^3) and 1.4 mg/m^3 (0.17 ml/m^3) (Brauer, 1992). It cannot be judged if this odour threshold provides an indicator for situations where industrial hygiene and/or engineering controls may need to be implemented.

The widespread industrial and skilled-trade applications of polymer dispersions containing residual 2-EHA monomer (< 0.08%) comprise uses in paints, lacquers, varnishes, moulding materials, impregnating agents and applications in adhesives and adhesive tapes. According to the Swedish product register 2-EHA is also used in lubricant/greases. In many cases, the polymeric dispersions are further processed to products, so that the concentration of the residual monomer decreases. However, the preparations are also directly used. Based on the low vapour pressure of the substance (12 Pa) and the low concentration of 2-EHA, the corresponding exposure scenario is expected to be of minor relevance for inhalation exposure. On account of the sensitising effect of the substance the scenario is described in view of dermal exposure.

Relevant occupational exposure scenarios are to be expected in the following areas:

- production of 2-EHA and polymerisation (Scenario 1),
- formulation of preparations containing up to 21% 2-EHA (Scenario 2),
- use of formulations containing monomeric 2-EHA in the building trade (Scenario 3),
- use of dispersions with residual monomeric 2-EHA (< 0.08%) (Scenario 4).

A decision on the importance of exposure scenarios is made in comparison with the “critical exposure level” derived on toxicological data. For 2-EHA, the critical exposure level amounts to

6.4 mg/m³. Within this occupational risk assessment, concern will be expressed for scenarios with exposure levels above this concentration. Therefore, exposure scenarios with anticipated exposure levels < 1 mg/m³, being considerably below this concentration, are regarded to be of minor relevance. These scenarios are therefore not described in detail and are not assessed quantitatively.

4.1.1.2.1 Production of 2-EHA and polymerisation (Scenario 1)

2-EHA is synthesised continuously in closed systems as a result of 2–ethyl hexanol and acrylic acid reacting in the boiling heat (acid-catalyzed esterification). Purification of the product is achieved via several stages of distillation (BUA, 1991).

2-EHA is transported in rail tank cars, tank trucks, barges and drums. In-company transportation mainly takes place in a closed system. Exposure associated with transport of this chemical would result from loading, unloading and drumming operations.

For the purpose of storage the substance is stabilised against spontaneous polymerisation using hydroquinone or hydroquinone monomethyl ether (BUA, 1991).

The monomer is processed further to polymers in closed systems, mainly to approximately 50% aqueous polymer dispersions. Typical specifications of residual monomer content of 2-EHA in these polymers are between 0.02-0.08% (BAMM, 1993, BUA 1991).

In the area of production and further processing of the substance exposure is possible during sampling, filling operations, reprocessing and cleaning, and maintenance works. Generally, it is to be assumed, that within the large-scale chemical industry high standards of control are practised even if the containment may be breached, e.g. during maintenance and the taking of process samples. Inhalation exposure in other areas is normally minimised by technical equipment (e.g. special designed filling stations, local exhaust ventilation).

Inhalation Exposure Workplace measurements

Table 4.1 2-EHA exposures (8-hour TWA) at workplaces during production and further processing (provided by 3 producers)

Job category / activities	Years of measurement	Number of samples [1)]	Range of measurement data [mg/m³]	95th percentile [mg/m³]
8-hour time weighted average				
Production operations	1997	28	< 2.9	1.3
Emulsion polymer plant	1993-1995	187	< 7.2	-
Drumming / loading	1993-1995	20	0.75-4.5	-
Maintenance	1997	20	< 7.6	2.8
Maintenance	1993-1995	63	0.02-3.5	
EP collection / disposal	1997	14	< 2.5	2
Quality assurance	1993-1995	9	< 0.-0.9	-
All workplaces described below	1995-2001	332 (27)	< 0.0038-8.4	0.48

Table 4.1 continued overleaf

Table 4.1 continued 2-EHA exposures (8-hour TWA) at workplaces during production and further processing (provided by 3 producers)

Job category / activities	Years of measurement	Number of samples [1)]	Range of measurement data [mg/m³]	95th percentile [mg/m³]
Production (closed system)	1995-2000	97 (3)	< 0.0084-8.4	0.54
Subsequent users (closed system)	1995-2000	173 (9)	< 0.0038-2.45	0.077
Laboratory (ventilation, exhaustion)	1995-2000	47 (8)	< 0.0076-2.22	0.3
Pilot plants (ventilation, exhaustion, closed system)	1995-2000	5 (2)	< 0.0076-0.29	-
Filling/Storage (ventilation, exhaustion)	1995-2000	4 (2)	0.05-3.06	-
Maintenance (ventilation, exhaustion)	1995-2000	2 (2)	0.023-0.092	-
Waste disposal (ventilation, exhaustion)	1995-2000	4 (1)	0.038-0.36	-

1) In brackets: number of plants

For the purpose of determining 2-EHA in the air at the workplace, the substance is adsorbed to activated charcoal and then desorbed using carbon disulphide and determined gas-chromatographically. The detection limit of the method amounts to 0.08 mg/m^3 (0.01 ml/m^3) (BASF, 1994). In most cases, no individual measurement results were provided by industry but pooled measurement data, in part only described as below a certain value (e.g. 1/10 of the OEL).

Due to the measurement method and the measurement strategy which were employed, the currently available measurement results are regarded as valid.

In the literature published personal monitoring data demonstrate, that on a routine basis, individuals involved in the production of 2-EHA are exposed to air concentrations that are generally lower than 8.3 mg/m^3 (1.1 ml/m^3) (BAMM, 1993).

On the basis of the presented measurement results (see **Table 4.1**) it is not possible to calculate a 90th percentile as the reasonable worst case representing all data collectives. The 95th percentiles provided for data collectives obtained 1995-2002 reveal that exposure levels have decreased. However, it is not known, whether the data from 1995-2000 is representative for all producers and users. Therefore, at present, the highest 95th percentile of 2.8 mg/m³ is taken as representing the reasonable worst case situation.

No information on short term exposure is available.

EASE estimation

EASE estimation for the production and further processing of monomeric 2-EHA:

- Input parameters: T = 20°C, closed system, significant breaching, LEV present, vapour pressure 12 Pa
- Exposure level: 4-8 mg/m^3 (0.5-1 ml/m^3)

Conclusion

The reasonable worst case of 2.8 mg/m^3 and the result of the estimation using the EASE model (4 - 8 mg/m^3, 0.5 - 1 ml/m^3) are in agreement, although the 95th percentile is slightly below the lower level of the assessed range. It has to be kept in mind, that the vapour pressure of 2-EHA is rather low. For the assessment of the risks of daily inhalation exposure 2.8 mg/m^3 (8-hour TWA) should be taken.

It is to be assumed that the substance is processed daily. Consequently, the duration and the frequency of exposure to 2-EHA are assumed to be daily and for the entire length of the shift.

Measurement values on short term exposure are not available. The assessment of short term exposure using the EASE model has some limitations. The model leads to exposure level of 4-8 mg/m³ for the time the activity under consideration is carried out, e.g. 1 hour. Based on the low vapour pressure of 2-EHA, the lower value seems to be reasonable. Since this level (4 mg/m³) is only slightly above the assessed 8-hour TWA, it does not provide useful information for the risk assessment.

Dermal exposure

When producing and further processing 2-EHA 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
- Exposure level: 0.1-1 mg/cm^2/day.

Considering an exposed area of 420 cm^2 (palms of hands) the model yields an exposure level of 42-420 mg/person/day.

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. Furthermore, on account of the highly irritative effect of pure 2-EHA as well as of preparations (> 21% 2-EHA) it is assumed that, as a rule, daily repeated immediate skin contact is avoided to a large extent by using personal protective equipment (here: gloves) and by applying appropriate working techniques. Therefore daily repeated actual dermal exposure is assessed as negligible. According to the discussion on the revision of the TGD (worker exposure) the experts concluded not to assess dermal exposure for the handling of corrosive or highly irritative formulations.

Single dermal contacts may occasionally occur during activities like drumming, filling, cleaning and maintenance. For this scenario, potential dermal exposure is assessed applying the EASE model:

- Input parameters: Direct handling, non dispersive use, incidental
- Exposure level: 0-0.1 mg/cm^2/day.

Because workers avoid contact with highly irritative substances it is to be assumed that rather small skin areas are exposed. Considering an exposed area of 105 cm^2 the exposure level amounts to 0-10.5 mg/person/day. This exposure level should be taken for assessing the risks of occasional but not daily dermal exposure.

In the case of occasional cleaning and maintenance of the plant (e.g. during "shut down" of the plant), larger skin areas than during usual daily work may be exposed and complex mixtures may be involved. If the highly irritative substance is handled, workers avoid immediate contact. This effect is not present if diluted solutions (concentration below 21% 2-EHA) are handled. An EASE estimation:

- Input parameters: Non dispersive use, direct handling, intermittent
- Level of exposure: 0.1-1 mg/cm²/day

leads under consideration of a skin area of 1,300 cm² (both hands and parts of the forearms) and a concentration of 21% 2-EHA to exposure levels of 27-270 mg/person/day for occasional (not daily) exposure during cleaning and maintenance activities.

Conclusions

Taking into account the high irritative effect of 2-EHA, daily exposure is assessed as negligible. Nevertheless, occasional exposure of 0 – 10.5 mg/person/day is possible (not daily).

In case of cleaning and maintenance activities (e.g. during shut down of a plant), higher dermal exposure of 27 - 270 mg/person/day are possible. It is to be assumed, that these exposure levels occur once a year for several days.

Exposure to the eyes is largely avoided by using eye protection.

4.1.1.2.2 Formulation of preparations containing up to 21% 2-EHA (Scenario 2)

According to information provided by one producer formulations on monomer basis used in the building trade (such as, for example, floor coatings, road-marking substances) may contain 2-EHA in concentrations up to 21%.

Formulating works of 2-EHA may occur in large scale chemical companies as well as in small and medium-sized formulating companies. A research project of BAuA revealed that in small and medium-sized companies beneath high level of protection also lower levels are observed, e.g. workplaces are not equipped with ventilation systems and workers not wearing gloves although both measures are required (Voullaire, Kliemt, 1995).

It is to be assumed, that floor coatings and road-marking agents are produced batch wise. In this, exposure relevant activities are performed not during the whole shift but for a limited duration.

Inhalation exposure

Workplace measurements

No data on exposure levels of 2-EHA at the workplace are available.

For the manufacture of formulations in the large scale chemical industry, exposure levels are regarded to be similar as those given for the production (see **Table 4.1**). Taking into account that exposure relevant activities are not performed during the whole shift (batch wise production), daily exposure is assumed to be lower. However, these results are not regarded to be representative for all formulators.

EASE estimation

EASE estimation for the further processing of monomeric 2-EHA without local exhaust ventilation (LEV):

- Input parameters: T = 20°C, non dispersive use, direct handling, dilution ventilation, vapour pressure 12 Pa
- Exposure level: 77-154 mg/m^3 (10-20 ml/m^3).

As described above it is assumed, that due to batch wise production exposure relevant activities are not performed during the whole shift. The duration and frequency of exposure are assumed to be daily and for 2 hours/day, thus reducing daily exposure to a shift average of 19-38.5 mg/m^3.

Conclusions

Since measurement results are not available, exposure levels predicted according to the EASE model should be used for risk assessment. Due to the low vapour pressure of the pure substance (12 Pa at 20°C) the actual levels of exposure can be expected to be located at the lower limits of the predicted exposure ranges.

Inhalation exposure of 77 mg/m³ (lower level of the assessed range) is assessed based on the EASE estimate. Taking into account a daily duration of 2 hours, the shift average is reduced to 19 mg/m³. This level should be taken for assessing the risks related to workplaces not equipped with LEV. Investigations of BAuA revealed, that these workplaces are frequently observed in small and medium sized companies.

The assessed exposure level is regarded to be an estimate for the reasonable worst case situation. Lower exposure levels are expected if workplaces are equipped with LEV. In this, for the collective of all formulators, the typical exposure level is assumed to be lower than the assessed level.

Dermal exposure

On account of the highly irritative effect of pure 2-EHA as well as of preparations (> 21% 2-EHA) it is assumed that, as a rule, daily repeated immediate skin contact is avoided to a large extent by using personal protective equipment (PPE, here: gloves) and by applying appropriate working techniques. It is assumed that worker avoid immediate dermal contact to a large extent even if besides the strongly irritating substance also irritating preparations are handled. Filling, drumming, cleaning and sampling are regarded to be relevant for exposure. Therefore daily repeated dermal exposure is assessed as negligible. According to the discussion on the revision of the TGD (worker exposure) the experts concluded not to assess dermal exposure for the handling of corrosive or highly irritative formulations.

A research project of BAuA (Voullaire, Kliemt, 1995) revealed that in small and medium sized companies, if exposure relevant activities are performed, personal protective equipment is only seldom used. Therefore, for the further processing of 2-EHA in small and medium sized chemical enterprises it cannot be excluded that gloves are not regularly worn and that single dermal contacts may occasionally occur during activities like drumming, filling, cleaning and maintenance. The corresponding exposure is assessed by the EASE model:

- Input parameters: Direct handling, non-dispersive use, incidental
- Exposure level: 0-0.1 mg/cm^2/day

Based on the highly irritative effect, it is to be assumed that rather small skin areas are exposed. Considering an exposed area of 105 cm^2 the exposure level amounts to 0-10.5 mg/person/day. The higher level (10.5 mg/cm^2/day) should be taken for assessing the risks of occasional but not daily dermal exposure.

4.1.1.2.3 Use of formulations containing monomeric 2-EHA in the building trade (Scenario 3)

2-EHA is a component of preparations used in the building trade, for example, coating agents for industrial flooring or road-marking agents. According to information provided by one manufacturer the monomer concentration amounts up to 21%.

The preparations containing 2-EHA are filled into relevant containers on site, if necessary, mixed and applied by hand using a smoothing blade or other appropriate tools. It is to be assumed that a certain part of 2-EHA evaporates during the hardening phase. Similar information was provided by the Federal Monitoring Authorities in Germany.

Information submitted with regard to the use of a 2-component floor-coating agent reveals that 2-EHA is contained as a monomer in the liquid component in addition to methyl methacrylate. The preparation is used in the building trade for coating floors in buildings which are still at the shell stage. Technical protective measures are not employed. The wearing of respiratory protection and protective clothing are mentioned as personal protective equipment. The use of the coatings is determined by the order situation and is stated to be infrequent.

Inhalative and dermal exposure of workers is possible during charging, mixing and coating work as well as during cleaning work. It is assumed that these works are performed during the whole shift, but not daily. In case of application of road marking agents works are performed outside.

Inhalation exposure

Workplace measurements

No measurement results are available.

Analogous data

The formulations often contain beneath 2-EHA methyl methacrylate as a copolymer. The concentration of methyl methacrylate is similar to the concentration of ethyl hexylacrylate: up to 20%. Therefore, exposure data of methyl methacrylate obtained during flooring works (cast coating, filling, sealing) are given (see RAR methyl methacrylate). Measurement values between 200-800 mg/m^3 (mean values, no TWA) were provided. In addition, 95th percentiles of 1,045 mg/m^3 (n = 78) for flooring works with ventilation systems being present and 625 mg/m^3 for workplaces without ventilation systems were given. Using the value of 1,045 mg/m^3 and taking into account the vapour pressures of the substances (3,870 Pa for methyl methacrylate and 12 Pa for ethyl hexylacrylate) a rough estimation leads to an exposure level of 3 mg/m^3 ethyl hexylacrylate (a linear relationship of vapour pressure of the pure substances and exposure levels is assumed).

Results of measurements of methyl methacrylate for reduced times of exposure (< 1 hour) are clustered with other activities than floor coating (n = 50, 50th percentile: 195 mg/m^3, 90th

percentile: 521, 95th percentile 683 mg/m³). It is stated that for the application of floor sealing agents, exposure levels were higher than the 50th percentile.

Even if it is assumed that during flooring works the 95th percentile is a measure for the reasonable worst case situation of short term exposure, this level is not higher than the highest 8-hour TWA. In this, assessing a short term value on this basis would not provide useful information.

Model estimations

The EASE estimation for the uses in the building trade (T = 20°C, wide dispersive use, direct handling, dilution ventilation, vapour pressure 12 Pa) leads to non-plausible exposure levels (765-1,071 mg/m^3), since they are higher than the saturation concentration of 2-EHA. Therefore, the model estimates cannot be used for assessing exposure levels.

Conclusions

For assessing the risk of inhalation exposure in the building trade an exposure level based on measurement results of methyl methacrylate is taken. For flooring works, the risks of not daily inhalation exposure should be based on an exposure level of 3 mg/m³. For works performed outside (here road marking activities) exposure is assumed to be lower because the air ventilation rate is higher than in rooms.

Short term exposure levels are not assessed. The available measurement results are below the shift average.

Dermal exposure

For the building trade it is to be assumed that protective gloves are not regularly worn. This assumption is also valid in case of handling preparations containing ≤ 21% 2 EHA, since these preparations are less irritative than the pure substance or concentrated preparations. Therefore dermal exposure is assessed according to the EASE model:

- Input parameters: Direct handling, wide dispersive use, intermittent
- Exposure level: 1-5 mg/cm^2/day.

Considering a 2-EHA content of 21% and an exposed area of 840 cm^2 (hands) an exposure level of 175-880 mg/person/day is obtained. The higher level (880 mg/person/day) should be taken for assessing the risks. Exposure is assumed to occur not daily.

4.1.1.2.4 Use of dispersions with residual monomeric 2-EHA (Scenario 4)

The widespread industrial and skilled-trade applications of polymer dispersions containing residual 2-EHA monomer (< 0.08%) comprise uses in paints, lacquers, varnishes, moulding materials, impregnating agents and applications in adhesives and adhesive tapes.

It is to be assumed, that the amount of residual monomeric 2-EHA decreases during the further processing of the dispersions to products and by further reactions of the monomer, e.g. hydrolysis. At present, it is not possible to quantify the extent of this decrease. Inhalation exposure during the application of the preparations is assumed to be negligible even if spray applications are performed (low concentration, low vapour pressure). Taking into account the

sensitising effect of the substance, dermal exposure is regarded to be of importance in spite of the low concentrations.

For an overall estimation of dermal exposure a 2-EHA concentration of 0.08% is assumed. Applying the EASE model with the following parameters:

- Input parameters: Direct handling, wide dispersive use, intermittent
- Estimated level of exposure: 1-5 mg/cm²/day

and considering an exposed area of 840 cm², dermal exposure levels of 1-3 mg/person/day are obtained. The higher level (3 mg/person/day) should be taken for assessing the risks. Exposure is assumed to occur not daily.

4.1.1.2.5 Summary

Based on the information available within the framework of this exposure assessment, ethylhexyl acrylate is mainly used as a monomer in the chemical industry for the manufacture of polymeric chemicals, which are processed further to aqueous polymer dispersions (approximately 50% polymer). The polymers and polymer dispersions are used in different products e. g. in adhesives, in printing inks and as binders in paints. In addition, monomeric 2-EHA is an additive in preparations, which are applied in the building trade as floor coatings and road-marking materials. The concentration of monomeric 2-EHA amounts up to 21%.

Exposure scenarios regarding the handling of monomeric 2-EHA present opportunities for exposure. The low vapour pressure of 2-EHA (12 Pa) leads to limited inhalation exposure levels. If the pure substance or preparations containing > 21% 2-EHA are handled it is to be assumed, that workers protect themselves against the highly irritative effect of the substance by using protective equipment (here gloves) and by applying appropriate working techniques.

Relevant occupational exposure scenarios are:

- production of 2-EHA and polymerisation (Scenario 1),
- formulation of preparations containing up to 21% 2-EHA (Scenarios 2),
- use of formulations containing monomeric 2-EHA in the building trade (Scenario 3),
- use of dispersions with residual monomeric 2-EHA (< 0.08%) (Scenario 4).

The inhalative and dermal exposure levels (reasonable worst case) are given in **Table 4.2** and **Table 4.3**.

For the large-scale chemical industry, it is assumed that the production and further processing of 2-EHA is mainly performed in closed systems. Exposure occurs during certain activities in the manufacturing and further processing of monomeric 2-EHA (Scenario 1, **Table 4.2** and **Table 4.3**).

Within the further processing industry and skilled-trade areas, lower levels of protection than in the large-scale chemical industry are to be assumed. Exposure occurs mainly during the manufacture of preparations containing up to 21% 2-EHA (Scenario 2, **Table 4.2** and **Table 4.3**) and their uses in the building trade (floor coating, road marking) (Scenario 3, **Table 4.2** and **Table 4.3**).

The widespread industrial and skilled-trade applications of polymer dispersions containing residual 2-EHA monomer (< 0.08%) comprise uses in paints, lacquers, varnishes, moulding

materials, impregnating agents and applications in adhesives and adhesive tapes. On account of the low concentration of 2-EHA and the low vapour pressure (12 Pa); inhalation exposure is regarded to be negligible compared to the critical exposure level of 6.4 mg/m³ (see Section 4.1.1.1.2, rough estimation, < 1 mg/m³). In view of the sensitising effect of the substance, dermal exposure is assessed although the concentrations of monomeric 2-EHA are very low (Scenario 4, **Table 4.3**).

Table 4.2 Summary of inhalation exposure data of 2-ethylhexyl acrylate which are relevant for occupational risk assessment

Inhalation exposure								
Area of production and use	Form of exposure	Activity	Duration	Frequency	Shift average [mg/m³]	Method	Shortened exposure [mg/m³]	Duration, Method
Production and further processing as a chemical intermediate								
1) Production of 2-EHA and polymerisation	vapour (liquid)	filling, sampling, cleaning, repair, maintenance	shift length	daily	2.8	95th percentile	1)	-
Further processing of monomeric 2-EHA to formulations (< 21% 2-EHA)								
2) Formulation of preparations containing up to 21% 2-EHA	vapour (liquid)	filling, sampling, cleaning, repair, maintenance	2 hours/day (assumed)	daily	19	EASE without LEV	77 (2 hours)	EASE
Use of formulations								
3) Use of formulations containing monomeric 2-EHA in the building trade (< 21% 2-EHA)	vapour (liquid)	floor coating, road marking	shift length	not daily	3	analogous data 2)	1)	-
4) Use of dispersions with residual 2-EHA (< 0.08%)	vapour (liquid)	different activities	-	-	negligible 3)	exp. judg.	negligible	exp. judg.

1) Short-term exposure levels are in the same range as the assessed shift average (see text)
2) Analogous data: methyl methacrylate is used, in part, in the same formulation
3) Exposure < 1 mg/m³

Table 4.3 Summary of dermal exposure data of 2-ethylhexyl acrylate which are relevant for occupational risk assessment

Dermal exposure								
Area of production and use	Form of exposure	Activity	Contact level [1]	Frequency	Level of exposure [mg/cm²/day]	Exposed area [cm²]	Shift average [mg/person/day]	Method
Production and further processing as a chemical intermediate								
1) Production of 2-EHA and polymerisation	liquid€	filling, sampling, cleaning, repair, maintenance	-- incidental	daily not daily	-- 0.1	-- 105	negligible 10.5 [2]	exp. judg. [3] EASE [3, 4]
Further processing of monomeric 2-EHA to formulations (< 21% 2-EHA)								
2) Formulation of preparations containing up to 21% 2-EHA	liquid	filling, sampling, cleaning, repair, maintenance	-- incidental	daily not daily	-- 0.1	-- 105	negligible 10.5	exp. judg. [3] EASE [3, 4]
Use of formulations								
3) Use of formulations containing monomeric 2-EHA in the building trade (< 21%)	liquid	floor coating, road marking	intermittent	not daily	1.05	840	880	EASE [5]
4) Use of dispersions with residual 2-EHA (< 0.08%)	liquid	different activities	intermittent	daily	0.004	840	3	EASE [5]

1) Contact level according to the EASE model
2) For cleaning and maintenance during shut down of a plant, exposure level of 27 – 270 should be taken (once a year, several days)
3) Highly irritative substance
4) Occasional exposure
5) Gloves are not regularly worn

4.1.1.3 Consumer exposure

As mentioned in Section 4.1.1, the Swedish Product Register lists a total number of seven products available for consumers and containing 2-EHA, which are referred to three product categories/functions. The respective codes of the product types are 1) lubricants/greases, 2) Agricultures/forestry and 3) products offered in wholesale and retail trade, repair shops for motor vehicles, -cycles and other household goods.

Because 2-EHA is also used in 4) paints and lacquers, as given in Section 4.1.1, it can be assumed that these products will reach the area of consumer use. 5) Because 2-EHA is used in the production of plastics, it can be expected that it is should appear as a residual monomer in plastic materials which may come into contact with food.

Although 2-EHA is also present as a residual monomer in floor coatings, the notifier has declared that these coatings are used only for industrial floors.

For consumer exposure, the following categories remain where knowledge of use is sufficient enough and paths of exposures are of interest for consumers. According to BAMM (1990; 1991; 1993) the residual monomer content accounts for 0.08% of the polymer.

Table 4.4 Summary of consumer product types

	Type of category of use	Path of exposure	Residual monomer content in product
1)	Lubricants and greases	dermal	0.08%
2)	Paints and lacquers	inhalation	0.08%
3)	Plastics	oral	unknown

Other uses have not been considered because of lack of information.

Agricultural and forestry uses are mentioned under “indirect exposure” via the environment.

Dermal exposure

Lubricants and greases used in cars or other vehicles may be exposed dermally to consumer for short periods of time during bringing up the grease. From this point of view, dermal exposure can occur via the use of consumer products, the quantification, however, is not possible because of lack of data. For a worst case estimate of dermal exposure the following assumptions were made: the weight fraction of residual monomer in grease is assumed to be similar to paints (0.08%) of the content of the polymer which is 10%. The volume of grease contacting the hands is 8.4 cm^3 (= 840 cm^2 [surface area] · 0.01 cm [thickness], TGD default, assumed density 1), then an amount of 0.672 mg/event of the residual monomer would lead to dermal contact (= 8.4 g · 10% · 0.08%). Assuming a body weight of 60 kg, the dermal exposure would result in 11.2 µg/kg bw per event.

For paints, the same scenario can be taken, however, taking a lower contact area set to 1 cm^2 for splashes of paints. Taking the weight fraction of the residual monomer in paints of 0.00048 (see Table), the dermal exposure to paints would reveal 1.3 µg/kg bw/event.

Inhalation exposure

For the estimation of the inhalatory exposure of the consumer, a computer simulation with the US-EPA model SCIES was used (comp. Technical Guidance Document p. 187-188) and using data given by BAMM for dispersion paints. All values together with SCIES default values are given in the table below. The amount of 2-EHApolymers in the paint is 60%, therefore the content of 2-EHA residual monomer is 0.048% (weight fraction of 2-EHA in paints 0.00012).

Consumer exposure with 2-EHA (dispersion paints)

Annual frequency of use	6	events/year
Mass of product	13,600	grams
Duration of use	4.9	hours
Volume of room of use (zone 1 volume)	40	m^3
Whole house volume	292	m^3
House air exchange rate	0.2	room air exchange/hr
User inhalation rate (during use)	1.3	m^3
Non-user inhalation rate	1.1	m^3
Molecular weight	184	g/mole
Vapour pressure	0.09	torr
Weight fraction	0.00048	residual monomer
Body weight	60	kg

The calculation reveals a peak room concentration during use of 22 mg/m^3 (= 2.9 ppm), the average concentration is 16 mg/m^3 (= 2.1 ppm). Measurements (BAMM) of 2-EHAresidual monomers after painting with paints containing 940 ppm (weight fraction 0.00094) and 2,000 ppm (weight fraction 0.002) a room with restricted ventilation revealed room air peak concentrations of 2.5 ppm and 8 ppm, which is in accordance to the estimated values. 2-EHA was not detectable 25 hours after painting.

For handicraftsmen, maximum air concentrations of < 1 ppm were measured during a monitoring programme by the notifier according to the TRG 402 which may be comparable to consumer use of paints.

For risk characterisation, the value of 1 ppm (and 0.0075 mg/l) of 2-EHA residual monomer in indoor air should be taken as a worst case value for short-term exposure scenarios. Taking into account the time of application of paints and that 2-EHA was not measured 25 hours after painting, chronic (long-term) exposure by inhalation is not given.

Oral exposure

Exposure to articles coming into contact with food

Plastic material that comes into contact with food is regulated by the EU directive 90/128/EEC, 28th of February 1990, “Directive of materials and articles intended to come in contact with food stuff”. In this regulation, 2-EHA has not been finally evaluated. Exposure data due to limitations given by the directive are therefore not available.

Due to other plastic material (e.g. MMA) the amounts of 2-EHA should be low and therefore be neglected.

4.1.1.4 Indirect exposure via the environment

According to Appendix VII of Chapter 2 of the TGD, the indirect exposure to humans via the environment, i.e. through food, drinking water and air is estimated.

Two local scenarios are calculated for comparison purpose. Site specific data for the main production site are used to represent worst case exposure of the soil and air compartment combined with a lower but realistic concentration in surface water. On the other hand, the scenario for the formulation of aqueous polymer dispersions is used representing the highest estimated concentration in surface water and comparably low exposure of the soil and air compartment.

In addition, the average human intake due to the regional background concentrations is calculated.

The input parameters are compiled in **Table 4.5**. The model calculations are presented in Appendix A10.1 and A10.2.

Table 4.5 Local and regional scenarios for indirect exposure

		Site specific, site A	Formulation of polymer dispersions	Regional
Concentration in surface water	PEC_{water_ann}	$1.2 \cdot 10^{-4}$ mg /l	$4.9 \cdot 10^{-4}$ mg /l	$5.8 \cdot 10^{-6}$ mg /l
Concentration in the atmosphere	PEC_{air_ann}	$8.3 \cdot 10^{-3}$ mg /m³	$7.7 \cdot 10^{-5}$ mg /m³	$7.9 \cdot 10^{-7}$ mg /m³
Concentration in grassland soil	$PEC_{grassland}$	$1.3 \cdot 10^{-3}$ mg /kg	$2.2 \cdot 10^{-5}$ mg /kg	$6.8 \cdot 10^{-5}$ mg /kg
Concentration in grassland porewater	$PEC_{grassland\_pw}$	$8.2 \cdot 10^{-5}$ mg /l	$1.4 \cdot 10^{-6}$ mg /l	$4.2 \cdot 10^{-6}$ mg /l
Concentration in groundwater:	PEC_{grw}	$5.2 \cdot 10^{-5}$ mg /l	$8.7 \cdot 10^{-7}$ mg /l	$4.2 \cdot 10^{-6}$ mg /l

The resulting total daily doses and the routes of exposure are displayed in **Table 4.6**:

Table 4.6 Total daily doses and contribution of the different routes of indirect exposure

Scenario	Site specific, site A	Formulation of polymer dispersions	Regional
total daily dose ($DOSE_{tot}$)	$2 \cdot 10^{-3}$ mg·kg $_{bw}^{-1}$·d^{-1}	$3.6 \cdot 10^{-4}$ mg·kg $_{bw}^{-1}$·d^{-1}	$6 \cdot 10^{-6}$ mg·kg $_{bw}^{-1}$·d^{-1}
% via drinking water	< 0.1	2	2
% via air	89.4	4.6	2.9
% via stem (leaf crops)	5.4	0.3	0.2
% via root crops	1.1	0.1	28.4
% via meat	< 0.1	< 0.1	< 0.1
% via milk	< 0.1	< 0.1	< 0.1
% via fish	3.9	93	66.6

The main route of indirect exposure in the local scenario is the intake via air (for site A) and via fish consumption (for formulation of polymer dispersions). Other routes of exposure do not comprise to a significant extent to the total daily dose. For the regional scenario 2/3 of the total

dose is attributed to the consumption of fish followed by nearly 30% uptake via root crops. Exposure via air is only of minor importance in the regional scenario.

4.1.2 Effects assessment: Hazard identification and Dose (concentration) - response (effect) assessment

4.1.2.1 Toxico-kinetics, metabolism and distribution

So far no specific studies have been carried out on the metabolism of 2-EHA (2-ethylhexyl acrylate). However a variety of studies on rats have indicated that short-chain-acrylates such as ethylacrylate undergoes the following metabolic reactions: carboxylesterase-catalyzed hydrolysis of the ester function to release acrylic acid and alcohol (Silver and Murphy, 1981; De Bethizy et al., 1987; Ghanayem et al., 1987; Vodicka et al., 1990; Frederick et al., 1992; Linhart et al., 1994; Frederick et al., 1994). The half-life of ethylacrylate-hydrolysis in rat liver (*in vitro*) was approximately 2 seconds. In 13 other tissues it was as much as 15 minutes (Frederick et al., 1992).

Using purified porcine liver carboxylesterase, the enzymatic hydrolysis of several acrylates and methacrylates was characterised to determine Km[5] and Vmax- values for each ester (McCarthy and Witz, 1997). α- Methylsubstitution had only a minor effect upon Km or Vmax, but the alcohol chain length significantly affected the Km values for enzymatic hydrolysis. Butyl acrylate had a Km value four times lower compared with that for ethyl acrylate. The Vmax for butyl acrylate was about six times slower than the Vmax of ethyl acrylate. Data on 2-EHA are not available.

Excretion balance studies were conducted with 2-ethylhexanol (2-EH) in female Fischer 344 rats following single high (500 mg/kg) and low (50 mg/kg) oral doses of 14C -2-EH, following repeated (14 day) oral dosing with unlabelled 2-EH at the low level, and following a 1 mg/kg i.v. dose of 14C-2-EH. All the oral doses were eliminated rapidly, predominantly in the urine during the first 24 hours. Urinary metabolites eliminated following the oral doses were predominantly glucuronides of oxidised metabolites of 2-EHA (2-ethyladipic acid, 2-ethylhexanoic acid, 5-hydroxy-2-ethylhexanoic acid and 6-hydroxy-2-ethylhexanoic acid) (Deisinger et al., 1994).

The acrylic acid is decarboxylated and degraded to carbon dioxide (Gut et al., 1988; Sapota, 1988). Only a part of 2-8% (vary with the route of administration) of 2-EHA is bound to glutathione and excreted as thioether (Gut et al., 1988; Vodicka et al., 1990; Linhart et al., 1994).

14C-2-EHA (labelled on the vinyl carbons) was administered p.o. or i.p. on rats (100 mg/kg bw) (Sapota, 1988). The highest specific radioactivity was found three hours after i.p. administration in liver and kidneys, followed by spleen, lungs, brain, adipose tissue and blood. The 14C tissue levels decreased continuously. One exception to this was the adipose tissue at the 100 mg/kg dose; in this tissue the 14C- level remained constant for 72 hours. After oral dosing about 50% of the radioactivity was eliminated via the expired air and about 38% via the urine within the first 24 hours. A small portion of 2-EHA (about 1% of the dose) was excreted via the faeces.

Toxicokinetics summary

One study in experimental animals by the oral route has shown that 2-EHA is rapidly and extensively absorbed, distributed and eliminated (about 90% during the first 24 hours). There are no specific toxicokinetic studies using dermal administration or inhalative exposure.

[5] Km: Michaelis Menten Constant, Vmax: maximal velocity

4.1.2.2 Acute toxicity

4.1.2.2.1 Studies in animals

Oral

Acute oral toxicity is characterised by LD_{50} values of 4,000-6,000 mg/kg. As clinical signs: scant droppings, wet yellow stained anogenital area, decreased spontaneous motoric activity and ataxia are mentioned:

Within a list of range finding toxicity data a short abstract of test results is given, stating that for 2-EHA (no data on purity) an oral LD_{50} of 6.50 (4.72-8.95) ml/kg (approximately 5,770 mg/kg) was detected in a test with male rats (no further data available, Carpenter et al., 1974).

In a second test with 2-EHA (stabilised with 0.05% hydroquinone, no data on purity) administration of 10% aqueous traganth solutions of the substance to rats resulted in an oral LD_{50} value of 5.0 ml/kg (approximately 4,430 mg/kg). Clinical signs observed were apathy, narcotic state, and diarrhoea; no histologic alterations were detected, no further data are given (BASF AG, unpublished report, 1958).

In a test with ten male mice/dose group (2,500 mg/kg and 5,000 mg/kg, vehicle corn oil), 2-EHA (purity > 99.5%, stabilised with 10-20 ppm MMHQ) caused no mortality after administration of 2,500 mg/kg, but 2/10 mice died within 24 hours after administration of 5,000 mg/kg. Surviving animals recovered within 3 days after substance application. Clinical signs observed were scant droppings, wet yellow stained anogenital area, decreased spontaneous motor activity, ataxia, and abdominal breathing. No gross changes were detected at necropsy (Rohm and Haas, unpublished report, 1982).

Inhalation

Valid data on acute inhalation toxicity tests are not available; but acute inhalation toxicity of 2-EHA seems to be low. Several reports on animal tests are mentioned: Within a list of range finding toxicity data is stated that no mortality was observed in rats after an 8-hours inhalation of “concentrated vapours” of 2-EHA (no data on purity), temperature of that atmosphere is not mentioned (Carpenter et al., 1974).

In a test with rats, after an 8-hour inhalation of an atmosphere saturated with 2-EHA at 20°C (stabilised with 0.05% hydroquinone, no data on purity) no mortality and no clinical signs were observed in 6 animals, no more details are given (BASF AG, unpublished report, 1958).

In a range-finding test on ethylhexyl acrylate, substantially saturated vapour was prepared by spreading 50 g of the chemical over 200 cm^2 area on shallow tray placed near the top of a 120 L glass chamber at room temperature which was then sealed for at least 16 hours, while an intermittently operated fan agitated the internal chamber atmosphere. Rats were then introduced in a cage designed and operated to minimise vapour loss. After an 8-hour inhalation of that saturated 2-EHA vapour (no data on purity) none of 6 rats died within the inhalation or within the 14-day observation period after the inhalation of 2-EHA vapours. Hyperactivity on removal from exposure chamber was the only clinical sign documented, gross pathology revealed nasal and ocular irritation (Mellon Institute of Industrial Research, unpublished report, 1950).

Citations from literature on reports of tests with rats and mice are given within a report on the toxicity of 2-EHA. No deaths were observed. All rats exposed to saturated atmospheres at 20°C over an exposure time of 8 hours survived; no deaths occurred when mice were exposed to an atmosphere saturated with the substance at 60°C (BUA, 1991).

Dermal

The acute dermal toxicity of 2-EHA is low. For rabbits, a dermal LD_{50} value >10,000 mg/kg is reported: Only a short abstract of test results is given within a table, stating that for 2-EHA (no data on purity) a skin penetration LD_{50} of 16.00 (4.48-57.2) ml/kg (approximately 14,180 mg/kg) was detected for rabbits (Carpenter et al., 1974).

4.1.2.2.2 Studies in humans

Human data on the acute toxicity of the substance are not available.

4.1.2.2.3 Conclusion

Human data on the acute toxicity of the substance are not available. In animal studies with rodents, 2-EHA possesses slight acute toxicity, the primary effect being local irritation or corrosion, as judged on the basis of the sparse information on clinical signs reported and on the results from local irritation/corrosion testing (see Section 4.1.2.3/4.1.2.4), systemic effects are non-specific and much less pronounced. The substance is not to be labelled because of acute toxic effects.

4.1.2.3 Irritation/Corrosion

4.1.2.3.1 Studies in animals

Skin

In a skin irritation test performed similar to OECD and EU test guidelines, 6 rabbits were exposed for 4 hours under occlusion to 2-ethylhexyl acrylate (no data on purity). All animals exhibited severe erythema (mean values for 24 hours/72 hours: 3.2/2.7) and oedema (mean values for 24 hours/72 hours: 2.7/1.2); the severity of the skin lesions enhanced in 1/6 animals during the 72 hours observation period and the skin of this animal demonstrated score 4 and superficial chemical burns 3 days after exposure when the test was terminated (Hoechst Celanese Corp. unpublished report, 1972). It remains unclear whether full thickness destruction would have been observed at later observation times. In a series of patch tests the test substance caused moderate erythema after a 1 minute and after a 5 minute exposure time which reversed within 8 days. After an exposure time of 15 minutes severe erythema with scaling after 8 days were observed and severe erythema and moderate edema appeared within 24 hours after a 20-hour exposure time. Eight days after application scaling was stated (BASF AG unpublished report, 1978).

In an occlusive patch test according to US Federal Register Guideline of 1964 four rabbits were tested with 0.5 ml of 2-EHA (no data on purity) each, using 24 hours occlusive exposure to intact

and abraded skin. Scores (24 hours/72 hours) for erythema of 1.75/2 and for oedema of 3.25/3.25 were observed for intact skin, for abraded skin the same scores were obtained. No indication of necrotic effects are mentioned both in intact and abraded skin (Consultox Laboratories, unpublished report, 1980).

A short summary of the skin responses in rabbits and guinea pigs after repeated uncovered contact with 2-EHA is given by Hunter et al.: After 12 daily applications of the material without covering foci of necrosis were apparent and the test was terminated due to severity of skin damage (Hunter et al. 1966).

In order to support the decision if the local lesions caused after skin contact are to be classified as corrosion or as severe irritation, BASF AG carried out an alternative to the Draize skin test which was developed in order to differentiate between irritation and corrosion (EU Guideline B.40). The so called EpiDerm^{TM} Skin Corrosivity Test was performed using 2-ethylhexyl acrylate, purity 99.7%. The potential of 2-EHA to cause dermal corrosion was assessed by a single topical application of 50 µl of the test substance to a reconstructed three dimensional human epidermal model (EpiDerm^{TM}). Dublicates of the EpiDerm^{TM} tissue were incubated with 2-EHA for 3 minutes and 1 hour, followed by a colorimetric determination of the possibly induced cytotoxic effects. Viability of the test substance treated tissues determined after an exposure period of 3 minutes was 99% and for the exposure period of 1 hour 104% (2-EHA is not able to directly reduce MTT, the indicator used for detection of cytotoxicity). It is demonstrated that 2-EHA reacts like the negative control, while a caustic compound used as positive control proved that this system is able to detect caustic chemicals. Based on the observed results and applying the evaluation criteria of the test, 2-EHA does not have a corrosive potential in this test under the conditions chosen (BASF AG, unpublished report, 2001).

Eye

Eye irritation is reported to be evident but less significant than local effects on the skin of rabbits. In a test according OECD test guideline 405 and performed under GLP, 2-EHA (purity 98%) caused mild eye irritation: 0.1 ml of the substance was instilled into the eyes of 3 Albino rabbits, the following mean scores are documented for the 24, 48 and 72 observation times: cornea 0/0/0.3, iris 0/0/0.3, conjunctival redness 0.3/0/0.3, conjunctival chemosis 0/0/0.3. All signs of irritation were reversible within 3 days (Koch et al., 1985). The other existing tests on eye irritation are poorly described. In most cases there exists a general statement with respect to the test as to be performed in general, but no information on the specific test carried out with the substance 2-EHA. Hence, it is very difficult to decide on the weight of evidence of “grades” or “scores” mentioned in the tables of results and on possible consequences for classification according to current EU regulations.

Carpenter and Smyth reported corneal injury grade 6 on a scale of 10 as result of the instillation of 0.005 ml of a 40% solution of 2-EHA into the eyes of 5 rabbits. 24 hours after instillation of the material into the eyes numerical assessment scores > 5 were determined (meaning of “score 5”: necrosis on 63-87% of cornea, visible after staining with fluorescein). This result yielded in an assessment grade 6 out of a scale of 10 for corneal lesions caused by 2-EHA (Carpenter and Smyth, 1946).

Only a short abstract of test results is given within a list of range finding toxicity data, stating that corneal injury grade 1 within a scale of 10 was detected for undiluted 2-EHA (no data on purity; grade 1 means that at most a very small area of necrosis resulted from the instillation of 0.5 ml of the test substance into the eyes of rabbits) (Carpenter et al., 1974). Moderate

conjunctival irritation, but no lesions on cornea or iris resulted in an ocular irritation test according to US Federal Register Guideline of 1964. Instillation of 0.1 ml of 2-EHA (no data on purity) into the eyes of 6 rabbits resulted in either slight or well-defined injection of the vessels of the conjunctivae, one example of slight swelling was exhibited (scores after 24, 48, and 72 hours are stated to exist, but no information on these scores is available). The observation period is not mentioned. No corneal or iris lesions were identified in any animal. Since only 1/6 rabbits displayed a reaction which would be considered to be positive according to US regulations of 1964, the test was regarded as being negative (Consultox Laboratories, unpublished report, 1980).

There exists no standard test method for the assessment of respiratory irritation. Thus, the labelling of 2-EHA with R 37 according to current EU regulations is not based on results of a specific respiratory irritation test, but on considerations on the general irritation potential of 2-EHA: Nasal and ocular irritation is noted in a test on acute inhalation toxicity with rats (see Section 4.1.2.2). The local irritation potential of 2-EHA is detected on the skin and on the conjunctivae of the eye; primary respiratory irritation may be one of the origins of the serious lesions seen after repeated inhalation of 2-EHA (see Section 4.1.2.6).

4.1.2.3.2 Studies in humans

No data available.

4.1.2.3.3 Conclusion

Information on human experience with local irritation/corrosion caused by 2-ethylhexyl acrylate is not available. In animal experiments 2-EHA caused serious lesions to the skin of rabbits which are assessed to be situated at the border between severe irritation and corrosion. Therefore, in 2001 BASF AG carried out an alternative to the Draize skin irritation test according to the new EU test guideline B.40. (Skin Corrosion). This alternative test method is developed for differentiation between irritation and corrosion. The result of the new study demonstrates that 2-EHA does not have a corrosive potential in this test, and hence, the current classification of 2-EHA as irritant and labelling with “R 38, Irritating to skin” is confirmed.

Most of the existing tests on eye irritation are poorly described. In the only test according to international guidelines, 2-EHA caused mild eye irritation with the following mean scores for the 24, 48 and 72 observation times: cornea 0/0/0.3, iris 0/0/0.3, conjunctival redness 0.3/0/0.3, conjunctival chemosis 0/0/0.3. All signs of irritation were reversible within 3 days (Koch et al., 1985).

On the basis of this test the eye irritating properties of 2-EHA does not warrant labelling with R 36.

There exists no standard test method for the assessment of respiratory irritation. Thus, the labelling of 2-EHA with “R 37, Irritating to respiratory tract” according to current EU regulations is not based on results of a specific respiratory irritation test, but on considerations on the general irritation potential of 2-EHA (nasal and ocular irritation noted in a test on acute inhalation toxicity with rats, severe local irritation potential detected on the skin and moderate irritation potential detected on the conjunctivae of rabbits; serious lesions as seen after repeated inhalation of 2-EHA may well be initiated i.a. by primary respiratory irritation). Labelling with

R 37 is confirmed on the basis of all of the respective data mentioned within this Risk Assessment Report.

4.1.2.4 Corrosivity

4.1.2.5 Sensitisation

4.1.2.5.1 Studies in animals

In various tests involving guinea pigs 2-EHA proved sensitising, with and without adjuvants.

2-EHA has shown strong sensitising effects in a Freund's Adjuvant test (FCA test) with 10 animals in the test and in control group. The test was carried out by injecting or applying a 0.1% aqueous suspension of 2-EHA to the shorn skin on the backs of the guinea pigs on three days per week for three weeks. Challenge was performed 10 days after the last induction treatment with a 0.1% aqueous suspension of 2-EHA. After topical application 10/10 and after intradermal injection 5-7/10 animals reacted after 24 hours and 5/10 after 48 hours. The treated animals showed intense redness and oedema (Hunter et al., 1966).

Strong sensitising effects were reported also in a second FCA test. Naive guinea pigs were treated with a series of 3 intradermal injections on days 0, 5 and 9, with FCA and a 3% or a 9% concentration of 2-EHA. Challenges were performed every two weeks from day 21 by open epicutaneous application of 0.025 ml 2-EHA (approximately 18%) until day 49 on the shaved flanks. With both induction concentrations, sensitisation of guinea pigs to 2-EHA was demonstrated. Up to 13/16 treated animals, using an induction concentration of 3% and up to 11/16 animals, using an induction concentration of 9% 2-EHA revealed a positive response. Positive skin reactions were observed until day 105 after rechallenge on day 77. Of the control animals, which were treated for the first time epicutaneously with the challenge solution on day 21, three out of ten reacted as early as day 35 and one out of ten on day 49. Cross reactions of animals sensitised to 2-EHA were: for ethylacrylate, three out of eight; for n-butylacrylate, seven out of eight; and for hexylacrylate, two out of eight. No cross reactions were observed for tert.-butyl acrylate, methyl methacrylate or hexyl methacrylate (Waegemaekers and van der Walle, 1983).

The sensitising potential of 2-EHA in the guinea pig could be demonstrated by the Polak method. Guinea pigs (6 per group) received 4 footpad injections of 0.1 ml of an emulsion containing 2 mg/ml of 2-EHA, in ethanol: saline (1:4) in Freund's Complete Adjuvans (FCA). Challenge was performed 7 days after the induction treatment with 0.02 ml of the solution of 2-EHA in acetone: olive oil (4:1). The animals showed positive skin reactions after treatment with 0.2% and 0.8% 2-EHA already 7 days after the last treatment (Parker and Turk, 1983).

There is no information available on the potential for 2-EHA to produce respiratory sensitisation in animals.

4.1.2.5.2 Studies in humans

Seven male volunteers developed an allergic contact dermatitis to an acrylic based adhesive. All subjects were strongly positive to 2-EHA and three of the subjects were also strongly positive to N-tert.-butyl maleamic acid. Test concentrations were 5% in olive oil and 1% of n-tert.-butyl maleamic acid in petrolatum (Jordan, 1975).

A 51-year-old engineer developed hand eczema after contact to products used as anaerobic sealants in metal manufacturing. Positive test reactions were seen with the main component polyethylenglycoldimethacrylate but also with several other acrylates and methyacrylates including ethylhexyl acrylate. The test concentration was 0.5% in ethanol (Senff et al., 1992).

Four patients developed dermatitis from working with UV-cured inks in printing plants. Patch tests with multifunctional acrylate monomers yielded positive results with various acrylates, including ethylhexyl acrylate in two patients (Björkner and Dahlquist, 1979).

Six patients developed contact dermatitis to various acrylates after exposure to tape or glues. All patients tested positive to various acrylates. One of these patients developed eczema after surgery. The wound had been dressed with a tape, and the eczema was strictly localised under the tape. Positive patch test reactions were seen for 2-ethylhexyl acrylate (2% in petrolatum) and two other acrylates (Daecke et al., 1994).

A 51-year-old man with a limb prothesis developed contact sensitivity in the area of the amputation stump and in other areas after readjustment of the prothesis to have it revarnished. He reacted positive to numerous (Meth)acrylates including 2-ethylhexyl acrylate (test concentration: 0.1% in petrolatum) (Romaguera et al., 1990).

The evaluation of health surveillance examinations since 01.01.1989 in about 900 employees potentially exposed to 2-EHA in 5 different plants did not show any cases of sensitisation or allergic contact dermatitis. This was explained by the fact that according to the hazardous properties of the substance technical measures and personal protective equipment were applied (BASF AG, 2001).

Among 13,833 patients suspected of contact dermatitis examined during the years 1978-1999 occupational contact allergy to (meth) acrylates was diagnosed in 31 patients. Contact allergy to 20 different (meth) acrylates was diagnosed. The three most common sensitisers were ethylenglycol dimethacrylate (17 positive patch tests), 2-hydroxyethyl methacrylate (14 positive patch tests) and triethyleneglycol dimethacrylate (6 positive patch tests). Based on these evaluations 2-ethylhexyl acrylate (test concentration 0.5% in petrolatum) was not listed as an important occupational contact allergen (Geukens and Gossens, 2001).

There is no information available on respiratory sensitisation.

4.1.2.5.3 Conclusion

The contact sensitisation potential of 2-EHA was demonstrated in various test models involving guinea pigs. It was concluded that 2-EHA has moderate sensitising potential in experimental animals, and sensitisation in humans has also been reported. Information on respiratory sensitisation is not available. According to the data 2-EHA is classified with R 43 (May cause sensitisation by skin contact).

4.1.2.6 Repeated dose toxicity

4.1.2.6.1 Studies in animals

Inhalation studies

In a valid 90-day inhalation study (BASF, 1989) Wistar rats were administered in a whole-body exposition on 6 hours per day, 5 days per week, to 2-EHA vapour at concentrations of 0 ppm, 10 ppm, 30 ppm or 100 ppm (approximately 0.075 mg/l, 0.225 mg/l or 0.750 mg/l for the treatment groups) (2-EHA purity 99.7%). The study design was conducted according to OECD 413 (1981). Compared to the actual version of the test guideline, validity is restricted in that food consumption was not recorded, lung tissues were not perfused, and laryngopharynx was not examined. Histopathologic examination was carried out on 31 organs/tissues of high dose and control animals. The lungs, nasal cavity, thyroid and parathyroid glands, trachea and liver from all animals/all test groups were subjected to histopathology examination.

There were no treatment-related premature deaths. During exposure period animals of the high and mid dose groups exhibited lethargy and ptosis. Body weight gain was lower in both sexes of the high dose during and at the end of the study. A transiently reduced body weight gain was observed in mid dose females. From day 21 onwards mean body weight (absolute) was lower in high dose males compared to the control group. This parameter was not significantly altered in any other group at any time point during the study. Activities of ALAT and alkaline phosphatase were elevated in high dose females. In high dose males and females lower levels of total protein, albumin and glucose were demonstrated. Reduced protein and albumin values were also seen in each sex of the mid dose groups.

Absolute liver weight was reduced in high dose males and relative adrenal weights were lower in high dose males and females compared to the control groups. The microscopic examination revealed no lesion other than a focal or diffuse degeneration of the olfactory epithelium of the cranial nasal cavity in animals of both sexes of the high and mid dose groups. All rats of the 100 ppm group showed degeneration of the olfactory mucosa in the anterior part of the nasal cavity. The incidence of degeneration of the olfactory mucosa but not the severity was increased in mid dose rats. No treatment-related lesion of the nasal cavity was diagnosed at the low dose level.

Degeneration of the olfactory epithelium was characterised by a reduction of cell layers, reduction or loss of apical cytoplasmic structures such as olfactory knobs and microvilli, and by necrosis. Identification of the remaining olfactory mucosa cells was not possible. Occasional mitosis were present.

In detail, degeneration of the olfactory mucosa was diagnosed in the anterior part of the nasal turbinates (level 1) in all high dose rats and in four males and four females of the mid dose group. At the high dose level, the degeneration affected the olfactory mucosa diffusely in the dorsal and dorsolateral area, and the severity was mainly moderate. Mid dose animals showed small areas of degeneration of the dorsolateral olfactory mucosa of minimal severity. At level 2 of the turbinates, degeneration was diagnosed in all high dose rats, one mid dose male, two female and one female of the low dose group, and in one control group male. In the high dose group, the degeneration was diffuse in the dorsal and dorsolateral region, whereas in the other groups the degeneration was focal. The severity was minimal to marked in the high dose group, and mainly minimal in the other groups. Slight degeneration of the olfactory mucosa of the level

3 was only diagnosed in one male and one female each of the high dose group. 2-EHA induced no lesions of the trachea and the lungs, data of the pharynx/larynx were not available.

Table 4.7 2-EHA induced olfactory degeneration in rats from a 90-day inhalation study (BASF, 1989)

	Dose group	Control		10 ppm		30 ppm		100 ppm	
	Sex	M	F	M	F	M	F	M	F
	No. of animals	10	10	10	10	10	10	10	10
Nasal cavity	Level 1 (anterior)					4	4	10	10
	Mean severity grade					1	1	2.9	3.1
	Level 2	1		2	1	1		10	10
	Mean severity grade	1		1	1	2		2.9	2.5
	Level 3							1	1
	Mean severity grade							2	2

Grading used 1 = minimal, 2 = slight, 3 = moderate, 4 = marked; M = male, F = female

Treatment-related microscopic lesions outside the respiratory tract were seen in the liver. Fatty change (at low and medium severity grades a common finding in well fed rats) occurred in rats of all dose groups and control groups. In high dose males, the severity of fatty change was less compared to other groups and the control. The mean severity grades of lipid accumulation in the periportal zone of the liver lobus decreased from 2.6 in control males to 1.0 in the high concentration males, a minimal change was also seen in high concentration females (1.0 versus 1.6 in controls). No indication of a peroxisomal proliferation was evident in electron microscopy.

No treatment-related effects were evident at the low dose group.

Reduced body weight gain, lower levels of parameters of the protein metabolism, the reduced serum glucose concentration and the reduced lipid accumulation in liver cells were assumed to be induced by a lower food consumption possibly resulting from the irritation effect on the respiratory tract of exposed animals. Similar findings were reported from repeated dose inhalation studies on acrylic acid (BASF, 1987). Although this study did not include food consumption measurement to verify this assumption, the above mentioned effects were not considered to represent relevant toxic effects. In high dose groups, a minimal liver damage was indicated by elevated activities of transaminase and alkaline phosphatase. This effect was considered to be the only systemic one of toxicological significance. In conclusion, the NOAEC for local effects on the respiratory tract was considered at 10 ppm, whereas the NOAEC for systemic toxic effects was 30 ppm.

In an early inhalation study (Gage, 1970) two male and female rats exposed on 13 days (6 hours/day) to saturated 2-EHA vapour (1 mg/l, 130 ppm) showed initial weight loss, lethargy and slight respiratory difficulty. Any abnormality was found in blood and urine tests, and at autopsy. No other details were available.

Dermal studies

In a less documented study on the skin effects of 2-EHA on two mice strains after 3-month epicutaneous application it was shown that skin irritation was more severe in C3H than in NMRI

mice (BASF, 1986). 10 male C3H mice and 5 male NMRI mice were administered to 25 µl 2-EHA solution (86.5% 2-EHA in acetone) on the clipped dorsal skin (approximately 1,081 mg/kg bw/day, based on mouse body weight of 20 g) at three days per week, additionally 5 NMRI mice were treated with a 21% solution of 2-EHA in acetone (approximately 262 mg/kg bw/day), 10 male NMRI mice treated with acetone served as controls. Clinical symptoms, mortality and body growth were recorded. Macroscopic skin effects were reported from all C3H mice and five of the NMRI mice of all other groups. Histopathological examinations were restricted to the skin of the application area and of tissues with macroscopic abnormalies of 5 animals of each group.

No other clinical abnormalities other than crust formation in 10/10 C3H mice and reddening in 2/5 NMRI mice at the application site at 2-EHA concentration of 86.5% were observed. Epidermal hyperplasia was found in some C3H mice but not in the NMRI strain at 2-EHA concentrations of 86.5%. A condensation of the subcutis was reported for both strain at this concentration. No clinical or microscopic lesions were observed in NMRI mice treated with 21% 2-EHA and in vehicle control mice. With respect to local effects on the skin, a NOAEL of 25 µl of a solution containing 21% 2-EHA in acetone (262 mg/kg bw/day) administered on three days/week during 3 months was delivered for the NMRI mice. No conclusion on a systemic NOAEL can be drawn from this study.

Chronic irritative skin damage was noted in mice of a carcinogenicity study treated by dermal application of 2-EHA in acetone on weekly clipped interscapular region (Wenzel-Hartung et al., 1989; Brune and Deutsch-Wenzel, 1986, see Section 4.1.2.8 and **Table 4.16**). No data on the size of treated area were reported, 10% of the total body surface area can be used as a default assumption. Groups of 80 C3H/HeJ mice received 25 µl 2-EHA solution with 2.5%, 21% and 86.5% (corresponding to 1,081, 262 or 31 mg/kg/treatment day, calculation basis: 20 g bw at study begin) on 3 times/week during life time or served as controls (untreated control and vehicle group). An additional group was treated with a 43% solution during 24 weeks and was observed until end of life (stop-test). Beginning within the first few weeks scaling, and/or scabbing were observed at all dose levels. Lesions observed in animals treated with 2.5% showed a trend to regression after weeks 4 and 5 of treatment. Whereas regression of the skins lesions occurred within 7 weeks after termination of the treatment with 43% 2-EHA solution, further skin lesions developed in the 21% and 86.5% groups. At the end of study, numbers of animals with histological findings at the application site were:

Table 4.8 Skin examinations of different groups of mice treated with a solution of 2-EHA

	2-EHA-dose					
	86.5%	43%*	21%	2.5%	Acetone control	Untreated control
Hyperplasia grade 1	35	5	21	6	-	-
Hyperplasia grade 2	23	1	25	-	-	-
Hyperplasia grade 3	6	-	6	-	-	-
Hyperkeratosis	66	4	54	7	1	-
Scabbing	23	5	41	11	1	-
Thickened subcutis	68	37	56	79	-	-
Pigmentation in subcutis	72	10	54	42	-	-

* Stop-test;
Grading used: 1 mild, 2 moderate, 3 severe.

There are no repeated dose studies with oral application.

4.1.2.6.2 Studies in humans

No data available.

Other information

Toxic effects of 2-EHA with repeated application via the inhalation route at doses > 30 ppm (0.225 mg/l) were comparable to the effects of acrylic acid in 90-day inhalation studies at doses >75 ppm (0.221 mg/l). Further information; see risk assessment report acrylic acid.

No-observed-adverse-effect-level (NOAEL)

NOAEC for local effects on the respiratory tract

10 ppm (and 0.075 mg/l), 90-day inhalation, rats (BASF, 1989)

NOAEC for systemic toxic effects

30 ppm (and 0.225 mg/l), 90-day inhalation, rats (BASF, 1989)

LOAEL for local effects on the skin

25 µl 2-EHA solution (2.5% 2-EHA in acetone) (31 mg/kg bw/day), dermal lifetime study (3 days/week), mice (Wenzel-Hartung, 1989)

4.1.2.6.3 Conclusion

The relevant toxic effect after 90-day inhalation exposure of rats to 2-EHA was dose-related increased degeneration of the olfactory epithelium at concentrations from 30 ppm and higher (0.225 mg/l). The NOAEC for local effects on the respiratory tract was 10 ppm (0.075 mg/l). Animals exposed to 2-EHA concentrations of 30 ppm or higher showed poor health condition (lethargy, ptosis) during exposure period and reduced body weight gain, but no toxic effect on internal organs was identified (NOAEC for systemic effects). Minimal liver damage was indicated by elevated liver enzyme activities at a concentration of 100 ppm (0.75 mg/l). Valid studies with dermal or oral application routes are not available. Cancer studies and less documented subchronic studies with dermal application revealed that 2-EHA causes skin irritation at concentrations $\geq$ 2.5% (LOAEL).

4.1.2.7 Mutagenicity

In vitro studies

Bacterial systems

Two bacterial gene mutation assays were negative in doses up to 10,000 µg/plate with and without S-9 mix in Salmonella typhimurium strains TA 98, TA 100, TA 1535 and TA 1537 (Scribner and O'Neill, 1979; Zeiger et al., 1985).

Table 4.9 Mutagenicity-*In vitro* tests: bacterial systems

	Concentration range				
Test system	With S-9 mix	Without S-9 mix	Result	Remarks	Rreference
Salm. Typh. TA98, TA100, TA1535, TA1537	up to 5 µl/plate	up to 5 µl/plate	neg		Scribner and O'Neill, 1979
Salm. Typh. TA98, TA100, TA1535, TA1537	up to 10'000 g/plate	up to 10,000 µg/plate	neg	rat and hamster liver S-9 mix	Zeiger et al., 1985

In vitro studies

Chromosomal aberration and micronucleus tests

In mouse lymphoma cells, parallel investigation of structural chromosomal aberrations and micronuclei was performed by Dearfield et al. (1989). Treatment with doses of 20, 25, 31 and 34 µg/ml 2-EHA was done for 4 hours, only without S-9 mix. All treatments resulted in strong cytotoxicity (27, 16, 12 and 12% relative survival).

For analysis of chromosomal aberrations, cultures were exposed to BrdUrd after treatment and were sampled 14 to 15 hours after start of treatment. This was to enable the selection of 1st-division mitoses for analysis; however, co-treatment with BrdUrd, which is a genotoxin, is not recommended by the guidelines and makes findings difficult to interpret. No more than 100 mitoses were analysed per experimental point. Aberration frequencies in treated cultures varied from 5 to 9% (negative control, 4%). Given the various methodological insufficiencies, the findings are evaluated as inconclusive.

For analysis of micronuclei, cultures were exposed to cytochalasin B (3 µg/ml) after treatment; sampling was 16 to 17 hours after start of treatment; 1,000 cells were analysed per experimental point. The micronucleus frequency was 1.2% in the negative control and varied from 0.8 to 1.1% in the treated cultures, i.e. the result was negative.

In conclusion, there is no relevant evidence for clastogenicity of 2-EHA; a fully reliable finding, however, is lacking.

Table 4.10 Mutagenicity-*In vitro* tests: chromosomal aberrations and micronuclei

	Concentration range					
Test system	With S-9 mix	Without S-9 mix	Result	Toxicity	Remarks	Reference
chrom. Ab. in mouse lymphoma cells		20-34 µg/ml	inconcl	strong toxicity at all tested doses	various methodological insufficiencies	Dearfield et al., 1989
Micronuclei in mouse lymphoma cells		20-34 µg/ml	negative	strong toxicity at all tested doses		Dearfield et al., 1989

In vitro studies

Mammalian cell gene mutation test

Two mouse lymphoma assays and two HPRT tests with CHO cells were performed.

Cifone and Myhr (1984) reported on a mouse lymphoma assay which was weakly positive at doses in the toxic range. Doses of 15.6 to 150 nl/ml (with S-9 mix from Aroclor-induced rat

livers) or 1.95 to 60 nl/ml (without S-9 mix) were tested in a 4-hour treatment. Effects were slightly more pronounced with S-9 mix. Here, combining the data from two experiments, more than a doubling of the mutation frequency was achieved at doses ranging from 70 to 150 nl/ml, increases were 2.2- to 4.6-fold and were accompanied by moderate to strong cytotoxicity (relative growth varied from 4.8 to 54.6% without clear dose-dependency).

Without S-9 mix, combining the data from 3 experiments, more than 2-fold increases in mutation frequencies were obtained for doses of 15.6 to 60 nl/ml, maximum increase was 2.9-fold at the highest dose with only 8.5% relative survival.

Another mouse lymphoma assay, with 4-hour treatment only without S-9 mix, was reported by Dearfield et al. (1989). Slightly positive effects were obtained in high doses with strong cytotoxicity. Doses of 30, 31, 32, 33 and 34 nl/ml were tested in three independent experiments; according to combined data, mutation frequencies increased by factors of 1.6 to 1.9 without dose-effect relationship; relative survival ranged from 11 to 20.3%.

Two investigations were done on induction of HPRT mutations in CHO cells.

According to Slesinski et al. (1980) 2-EHA does not induce HPRT mutations after 5-hour treatment with or without S-9 mix. Doses of 3.13 to 50 · 10^{-5}% (v/v; with S-9 mix) or 6.25 to 100 · 10^{-5}% (without S-9 mix) were used; the maximum doses correspond to 5 and 10 nl/ml; only minor toxicity was seen.

In a 2nd investigation, cells were treated with doses of 5 to 80 µg/ml (monolayer assay) or 14 to 26 µg/ml (suspension assay) in the absence of S-9 mix (Moore et al, 1991). Whereas the suspension assay was clearly negative for all tested doses up to extreme toxicity, sporadic increases in mutation frequency were seen in the monolayer assay. In a 1st experiment, weak effects were seen at doses of 35 and 40 µg/ml which led to less than 20% relative survival. In the 2nd experiment doses of 60 and 70 µg/ml resulted in increased mutation frequencies, toxicity was moderate (33% relative survival). Higher doses of 75 and 80 µg/ml were negative (40 and 7% relative survival).

In conclusion, 2-EHA seems to have a low potential for induction of gene mutations in mammalian cells. Since the genetic effects were limited to doses with strong cytotoxicity, the potential will probably not be expressed *in vivo*.

Table 4.11 Mutagenicity-*In vitro* tests: mammalian cell gene mutations

	Concentration range					
Test system	With S-9 mix	Without S-9 mix	Result	Toxicity	Remarks	Reference
Mouse lymphoma assay	15.6-150 nl/ml	1.96-60 nl/ml	weakly positive	strong toxicity at high doses	genetic effects were limited to doses with strong cytotoxicity	Cifone and Myhr, 1984
Mouse lymphoma assay		30-24 nl/ml	weakly positive	strong toxicity at all doses	genetic effects were limited to doses with strong cytotoxicity	Dearfield et al., 1989
HPRT test with CHO cells	up to 5 nl/ml	up to 10 nl/ml	negative	minor toxicity		Slesinski et al., 1980
HPRT test with CHO cells		5-80 µg/ml	weakly positive	strong toxicity at high doses	negative in a suspension assay; weakly positive in a monolayer assay for cytotoxic doses	Moore et al., 1991

In vitro studies

Mammalian cell indicator tests

Slesinski et al. (1980) reported on a weak positive effect in an SCE test with CHO cells after 2-hour treatment in the presence of S-9 mix; without S-9 mix a negative finding was described for 5-hour treatment. Doses ranging from 3.1 to 100 · 10^{-5} % (v/v; 0.31 to 10 nl/ml) were tested. Due to severe methodological insuffiencies, these results are not reliable (extremely high 'spontaneous' SCE frequencies in negative controls of 13 to 18 SCE per cell; only 15 cells per entry were analysed in a single experiment).

A test for induction of unscheduled DNA synthesis (UDS) with primary rat hepatocytes was performed with the 'liquid scintillation counting' methodology which is known to be quite insensitive (Slesinski et al., 1980). A single experiment was conducted. The overall result for this test was negative for doses up to 100 · 10^{-5} % (10 nl/ml).

In conclusion, the data from mammalian cell indicator tests do not add relevant information.

Table 4.12 Mutagenicity-*In vitro* tests: Mammalian cell indicator tests

	Concentration range				
Test system	With S-9 mix	Without S-9 mix	Result	Remarks	Reference
SCE test with CHO cells	0.31-10 nl/ml	0.31-10 nl/ml	weakly positive	weak effects with S-9 mix; severe methodological insuffiencies	Slesinski et al., 1980
UDS test with rat hepatocytes		up to 10 nl/ml	negative	insensitive LSC methodology	Slesinski et al., 1980

In vivo studies

Bone marrow chromosomal aberration test

The possible induction of chromosomal aberrations *in vivo* was investigated in bone marrow cells of male Charles River CD-1 mice (Sames et al., 1984). Oral doses of 2,500mg/kg bodyweight were given acute or repeatedly on 5 consecutive days; toxic signs were seen. Sampling was 6 hours, 24 hours and 48 hours after single treatments and 6 hours after the last repeated application; treatment groups consisted of 5 animals each. At the 24 hours sampling after acute treatment an aberration frequency of 2.2% was obtained which differed in a statistically significant manner from the concurrent negative control, but not from the historical negative control. Since no more than 272 metaphse cells were analysed in this group, a biologically meaningful conclusion cannot be drawn from this finding. Furthermore, the whole investigation suffers from the drawback that less than 50 cells were analysed for 24 out of 72 animals, indicating severe methodological problems. Therefore, the overall result is inconclusive.

Table 4.13 Mutagenicity-*In vivo* tests: bone marrow test

Test system	Doses	Expos. regimen	Sampl. Times	Result	Local cyto-tox.	General toxi-city	Remarks	Reference
chrom. ab. test with mice	2,500 mg/kg	a) acute; b) 5 daily administratios	a) 6, 24, 48 hours b) 6 hours after last administration.	inconcl	no	yes	severe methodological problems	Sames et al., 1984

Genotoxicity data on 2-EHA cleavage products

Acrylic acid (CAS no. 79-10-7) was negative in bacterial mutations tests (Cameron et al., 1991; Zeiger et al., 1987; BASF, 1977) and in an HPRT mammalian cell gene mutation test (McCarthy et al., 1992). Positive effects were obtained in mammalian cell chromosomal aberration tests (McCarthy et al., 1992; Moore et al., 1988; Ishidate, 1988) and in mouse lymphoma assays (with preferential induction of small colonies, indicating clastogenicity; Cameron et al., 1991; Moore et al., 1988). *In vivo*, negative results were reported for structural chromosomal aberrations in mouse bone marrow cells and for a mouse dominant lethal test (McCarthy et al., 1992).

2-Ethylhexanol (CAS no. 104-76-7) was negative in bacterial and mammalian cells assays (Kirby et al., 1983) and an *in vivo* bone marrow cytogenetic assay (Putman et al., 1983).

Table 4.14 Mutagenicity-OVERVIEW ON FINDINGS

Negative effects	Inconclusive	Positive effects
In vitro tests		
Bacterial mutations	chromosomal aberrations	
micronuclei	gene mutations in mammalian cells at cytotoxic doses	
	SCE, UDS	
in vivo tests		
	chromosomal aberrations	

4.1.2.7.1 Conclusion

2-EHA is negative in bacterial mutation tests. Data from mammalian cells give no relevant evidence for clastogenicity; however, a fully reliable study is lacking. 2-EHA seems to have a low potential for induction of gene mutations in mammalian cells. Since this effect is limited to doses with strong cytotoxicity, it is highly unlikely that this potential will be expressed *in vivo*. The data from mammalian cell indicator tests do not add relevant information. An *in vivo* cytogenetic assay was inconclusive (neither positive nor negative); due to severe methodological insuffiencies this study cannot be used for evaluation purposes. Cleavage products of 2-EHA were negative in *in vivo* mutagenicity tests.

Since a fully reliable *in vitro* chromosome abberration test is lacking, it might be argued that the minimum requirements for genotoxicity testing are not met. However, the number of studies available from various test systems and the negative data on 2-EHA cleavage products 2-ethylhexanol and acrylic acid are regarded as a sufficient substitute. From all these data there is no relevant evidence that 2-EHA might be an *in vivo* mutagen.

4.1.2.8 Carcinogenicity

There are dermal carcinogenicity studies in mice, summarised in **Table 4.16**, but no cancer studies with oral or inhalative application route.

In an early dermal life-time carcinogenicity study (DePass et al., 1985; DePass, 1982; Peterson, 1979; Slesinski et al., 1980) 40 male C3H/HeJ mice were treated 3 times/week with 2-EHA (75% w/v) dilution in acetone at an average dose of 20 µg 2-EHA/application (approximately 750 mg/kg bw/day). 6 of 40 treated males developed neoplastic skin lesions. Four males had squamous cell papillomas and two others had squamous cell carcinomas. One out of 40 animals of the vehicle control group developed a skin carcinoma near the eye. The authors concluded that 2-EHA is carcinogenic in C3H mice.

The study is less reliable due to the method defaults. Only 40 males were investigated, exclusively gross lesions of skin and internal organs were examined histologically. Several tumours were documented grossly and most of them were examined histologically. Peterson (1979) reported several cases of chronic nephritis in 13 of 19 kidneys examined and necropurulent nephritis in several cases. Survival rate after one year of treatment was reduced (75%); the first skin tumour was seen at 11 months of treatment. At 18 months only 15/40 male and at 24 months none of them were still alive. As skin tumours were seen in a total of 6/40 mice, the cause of premature deaths remained unclear. Furthermore there were no data on irritative skin effects due to the treatment, histopathology of the skin were reported from seven males only. Tumours in non-cutaneous tissues were reported to be comparable between treated and control groups, however tumour data in internal organs were insufficient.

In order to confirm the preliminary findings of the above cited study a further study with the same strain, sex and 2-EHA-concentration was done, two additional dose groups were tested (Wenzel-Hartung, 1989; Brune and Deutsch-Wenzel, 1986). In this carcinogenicity study 25µl of 2-EHA (86.5%, 21%, or 2.5% solution in acetone, approximately 1,081, 262, 31 mg/kg bw/day) was applied 3 times/week to the clipped dorsal skin of male C3H/HeJ mice (80 per group) over their lifetime. Another group was treated with a 43% 2-EHA solution for 24 weeks and thereafter observed for lifetime (stop-test). An untreated group and acetone group served as controls. Body weight, clinical symptoms, and skin irritation were recorded. Gross lesions and the dorsal skin were fixed. The skin tissue from the application site was the only tissue that was examined histologically. Body weight was increased in all dosed groups; the survival time was comparable to that of the control groups. Treatment-related scale and eschar formation indicative of skin irritation were found in all 2-EHA groups beginning after the first few weeks of treatment. The subcutis was thickened and sometimes pigmented. The cutis showed hyperkeratosis, hyperplasia and scabbing in the 86.5% and 21% dose groups and with smaller incidence in the 43% and 2.5% groups. The observed skin changes were reversible in the 2.5% group after the 11th week of treatment and in the 43% group of the stop test immediately after treatment was stopped. Only in the 86.5% and 21% test groups papillomas of the skin were found and in a large percentage of animals cornified squamous cell carcinomas, melanocarcinomas and fibrosarcomas were identified without any dose dependency. No skin tumours were found in the control groups, in the groups treated with 2.5% 2-EHA for lifetime or in the group treated with 43% 2-EHA for about 6 months and observed for lifetime. Hepatic tumours were found in more than half of the mice of each dose group and control groups without any relation to treatment. Histologic examination of other organs was less extensive. The authors concluded that irritative skin lesions were precursors of the neoplasia. From this dermal lifetime study a LOAEL for local nonneoplastic effects on the skin was 25 µl 2-EHA solution at a concentration of 2.5% 2-EHA in acetone was derived (31 mg/kg bw/day). Incidences for skin tumours in animals were as follows:

Table 4.15 Incidences for skin tumours in animals

Number of animals with skin tumours	2-EHA-dose					
Tumours	86.5%	43%*	21%	2.5%	Acetone control	Untreated control
Papilloma	8	-	4	-	-	-
Papilloma with strong cornification	2	-	1	-	-	-
Cutaneous horn	2	-	1	-	-	-
Haemangioma	1	-		-	-	-
Basal cell carcinoma		-	1	-	-	-
Corinified squamous-cell carcinoma	16	-	20	-	-	-
Malignant melanoma	9	-	7	-	-	-
Fibrosarcoma	-	-	5	-	-	-

* Stop-test

2-EHA was also tested for carcinogenicity in male NMRI mice exposed dermally 3 times/week to 25 µl of 21.5%, 43%, or 85% 2-EHA (w/w) diluted in acetone (approximately 269, 538 and 1,063 mg/kg bw/day). 39-40 of 80 males tested were treated with 2-EHA alone, acetone (solvent control) or 0.015% (w/w) of benzo(a)pyrene in acetone (positive control, results were not reported here) for up to 24 months (BASF, 1992). Two animals of each group were killed at week 13 to examine the skin lesions. Nearly half of the animals (30-39 males) of each dose and control groups were treated as above for 7 months, after a treatment-free period of two months animals were treated with a promoter, 12-O-tetradecanoylphorbol-13-acetate (TPA), for 20 weeks. Thereafter there was no further treatment until the end of the study. Histopathology data were reported on the treated skin of all test animals and on the untreated skin area of 5-10 animals per group. Other organs/tissues were not included in histopathology examinations.

Whereas premature deaths were seen in the benzo(a)pyrene control group no treatment-related effect on mortality was observed in the 2-EHA groups. Neither 2-EHA nor the promoter TPA caused clinical signs besides the skin effects. Treatment-related lesions of the treated skin region were observed after 13 weeks and after 24 months in all 2-EHA groups. Some findings as hyperkeratosis, hyperplasia, crust formation and ulceration increased in severity or incidence related to the doses of 2-EHA, others (lymphocyte/macrophage infiltration, dermal fibrosis) were observed without any relation to the treatment groups. Reddening and thickening of the skin and similar microscopic skin lesions were reported during the promoter-phase. The authors concluded an irritative effect of the promoter TPA itself.

6/41 animals of the acetone group showed mild clinical symptoms (hyperkeratosis, hyperplasia, ulceration or lymphocytic/macrophage infiltrations).

None of animals treated with 2-EHA for up to 24 months showed a neoplastic lesion of the skin. 2-EHA and TPA promotion caused a squamous cell papilloma in one animal in each dose groups (of 30, 39, and 36 animals). One squamous cell papilloma was found in untreated skin of one male out of 41 of the acetone group.

The authors concluded that these tumours were related to the irritative effects of TPA, not to the treatment with 2-EHA. The LOAEL for nonneoplastic toxic effects of 2-EHA on the skin was 25 µl 2-EHA solution at a concentration of 21.5% 2-EHA in acetone (269 mg/kg bw/day).

The findings did not indicate a carcinogenic potential of 2-EHA on the skin of male NMRI mice.

Other information

Carcinogenicity data from cleavage products, acrylic acid and 2-ethylhexanol, were supplemented:

Cancer data from one oral rat study and two dermal mice studies (without conformance to requirements of the actual carcinogenicity testing protocols) using acrylic acid as test substance were considered.

In a valid carcinogenicity study (BASF AG, 1989; Hellwig et al., 1993) Wistar rats were administered to doses of 120, 400 or 1,200 ppm (mean substance uptake 9, 31, or 88 mg/kg bw/day) acrylic acid (99%, stabilised with 200 ppm hydroquinone monomethylether) in the drinking water for 26 months (males) or 28 months (females). Except a slightly reduced water consumption of high dose males and females no treatment-related clinical, hematological or histopathological changes were detected in comparison with the controls. The incidence and organ distribution of tumours found in the groups treated with acrylic acid did not differ from those of the controls (**Table 4.17**).

In a dermal carcinogenicity study no tumour of the skin or subcutis were induced in treated mice or in the vehicle controls. (Intercompany Acrylate Study Group, 1982 (**Table 4.18**). A group of 40 C3H/HeJ male mice received 25 µl applications of acrylic acid as 1.0% (v/v) dilutions in acetone. A negative control group received acetone only. The substances were applied to the skin of the back three times weekly for the lifetime of the animals. Histologic examination was performed on the dorsal skin of all treated mice and on gross lesions. The mortality rate was not affected by treatment (mean survival time in the acrylic acid group 515 days, in the acetone group 484 days). No signs of skin irritation were observed. One male of the acrylic acid group had an epidermal hyperplasia.

In another dermal carcinogenicity study 25 or 100 µl of 1% (v/v) acrylic acid in acetone was administered to two strains of mice (C3H/HeN Hsd BR, Hsd:(ICR)BR) during 21 months (3 times/week). Histopathology was done on the skin, some internal organs and every unusual gross lesion. No treatment-related signs of skin irritation, toxicity, clinical signs or skin tumours were observed. There was no treatment-related effect on body weight gain or mortality rate. 7/50 female C3H-mice of the 100 µl acrylic acid treated group revealed a significant increased frequency of lymphosarcoma compared to the acetone control group (BAMM, 1990, 1991; TSCATS, 1990, 1992a, 1992b) but lymphosarcomas are commonly seen in most strains of mice which are 18-24 months of age (Frith and Wiley, 1981) and their relation to the treatment was considered to be uncertain.

2-Ethylhexanol is known as peroxisome proliferator in animals, however this mechanism of tumour growth is considered not to be significant for humans. Results from long-term studies in rats and mice (EPA, 1992a, b) did not indicate that 2-ethylhexanol is carcinogenic in animals. Arneson et al (1995) reported that the US-National Toxicology Program nominated 2-ethylhexanol for further cancer studies.

4.1.2.8.1 Summary and conclusion

The carcinogenic potential of 2-EHA was tested in skin painting studies on the shaved back skin of male mice of different strains. None of the studies was performed according to the current regulatory recommendations on the EEC methods B 32 or B 33. The main defaults were that exclusively male mice were tested and that the effects on internal organs were not or

insufficiently examined and documented resulting in an incomplete database on carcinogenic activities after absorption.

2-EHA induced skin irritation and in nearly half the male C3H/HeJ mice treated dermally with 2-EHA solutions > 21% benign and malignant skin tumours (Wenzel-Hartung et al., 1989; Brune and Deutsch-Wenzel, 1986). Tumour incidence showed no relation to the dosage group of 21% or 86.5%. The additional study where treatment of 43% of 2-EHA were stopped at week 24 did not reveal any skin tumour. The weight of evidence that the test substance is carcinogenic is limited by the occurrence of skin irritation assumed to represent the precursor lesion of tumour growth. Repeated regenerative or proliferative reactions to irritative substances are discussed to be strongly associated to tumour development (Hasegawa et al., 1989). Even chronic physical stimuli such as abrasion in untreated skin were demonstrated to play a role in skin tumour induction. In general, dermal carcinogenicity studies should use test substance concentrations which did not induce irritative effects. In the BASF study (1986), no tumours were seen in the low dose group applicating 2.5% 2-EHA, although transient mild skin irritation was seen up to the 11th week of treatment. Contrary to positive cancer studies, skin irritation but no skin tumour was found in male NMRI mice treated dermally with 21.5%, 43% or 85% of 2-EHA in acetone for 24 months (BASF, 1992).

Higher incidences of skin tumours were also evident in two other dermal studies in mice. However in first study only one dosage (75% 2-EHA in acetone) was tested in C3H/HeJ mice where the mortality of unknown cause was markedly increased (94-97) already at one year of the treatment period. No clear tumour response was demonstrated in the two-stage carcinogenicity model using 2-EHA as the initiator substance and TPA as promoter in the study on NMRI mice (BASF, 1992). The negative response in this study may be related to the different strain used in this study (NMRI mouse) compared to the C3H/HeJ80 mouse of earlier studies that were positive. One skin tumour bearing animal occurred at each dose after 7 months treatment to 2-EHA and an additional treatment period to the promoter TPA. 2-EHA as well as TPA was shown to be irritative to the skin. Both studies were considered to be inadequate to detect the presence of carcinogenic effects.

Skin tumours from spontaneous origin are known to be variable in different mouse strains. One out of 41 control animals of the study of BASF (1992) had a squamous cell papilloma at an untreated skin area. No other spontaneous skin tumour was reported in the control groups of treated and untreated skin areas of the above cited studies.

From oral (the only study with validity according to the cancerogenicity test guidelines) and dermal studies on acrylic acid, the hydrolysis product of 2-EHA, there is no evidence on carcinogenic properties. Also, there is no concern from cancer data on 2-ethylhexanol.

In conclusion, there are no data available to the carcinogenic effects with respect to oral or inhalative exposure routes.

Findings from the dermal mouse carcinogenicity study showed that 2-EHA induces skin tumours at concentrations which were highly irritative. It was concluded, that tumour growth is associated the highly irritative properties of 2-EHA. At a low concentration of 2.5% 2-EHA with transient irritation no tumour response of the skin was observed. Other long-term studies on different mouse strains did not confirm tumour induction of the mouse skin. Additionally, there is no concern from tumour data of acrylic acid and 2-ethylhexanol, the hydrolysis products of 2-EHA.

Taking into account the negative results from in-vivo genotoxicity testing, it is concluded that 2-EHA induces skin tumours by a non-genotoxic mechanisms. Irritative skin damage was

identified as presumed mode of tumourigenicity as to was associated with carcinogenic effect of 2-EHA. Due to the limited reliability of skin painting studies in mice as a tool to identify the carcinogenic potential of a test substance these studies give some concern but no clear evidence that 2-EHA has carcinogenic potential. Based on limited database from dermal studies and absence of carcinogenicity data for the oral and inhalation routes, no conclusion could be drawn about the carcinogenic potential of 2-EHA. However taking into account the negative experimental results from long term animal studies with the cleavage product acrylic acid after oral and dermal application (cf. EU Risk Assessment Report Acrylic acid) there are no reasons to assume that 2-EHA should be considered as a carcinogenic substance.

Table 4.16 Dermal carcinogenicity studies with 2-Ethylhexylacrylate (2-EHA)

Species/ Strain no. of animals/sex/ group	Exposure time	Treatment schedule	Mortality rate	Skin irritation	Skin hyperplasia	Skin tumours	Tumour response of internal organs	Study design according to the B32/B33 method	Reference
mice/ C3H/HeJ 40 males	life time 3x/week	20 µg of 2-EHA (75% in acetone)	increased	no data	no data	squamous cell papilloma/ carcinoma in 6/40 males	no (no exact data)	no	DePass et al.,1985 DePass ,1982 Peterson, 1979 Slesinski et al.,1980
mice/ C3H/HeJ 80 males/ group	life time 3x/week	25 µl of 2-EHA (2.5, 21, 86.5% in acetone)	Ø	yes, 2.5%: symptoms until week 11, other findings see 4.1.2.6	yes	squamous cell papilloma/ carcinoma, melanoma fibrosarcoma in 21% group: 39/80 males 86.5% group 38/80 males	no (no exact data)	no	Wenzel-Hartung et al., 1989 Brune and Deutsch-Wenzel, 1986
mice/ C3H/HeJ 80 males	24 week 3x/week, thereafter observation until death	25 µl of 2-EHA (43% in acetone)	Ø	yes, lesions reversible	no	0/80 males	no (no exact data)	no	Wenzel-Hartung et al., 1989 Brune and Deutsch-Wenzel, 1986

Table 4.16 continued overleaf

Table 4.16 continued Dermal carcinogenicity studies with 2-Ethylhexylacrylate (2-EHA)

Species/ Strain no. of animals/sex/ group	Exposure time	Treatment schedule	Mortality rate	Skin irritation	Skin hyperplasia	Skin tumours	Tumour response of internal organs	Study design according to the B32/B33 method	Reference
mice/NMRI 39-40 males/ group	Life time (max. 24 months)	25 µl of 21.5, 43, 85% 2-EHA in acetone	Ø	yes,all doses	yes, all doses	0/39-40 males of each group	no data	no	BASF 1992
mice/NMRI 30-39 males/ group	Life time 3x/week for 7 months, thereafter treatment*	25 µl of 21.5, 43, 85% 2-EHA in acetone + TPA*)	Ø	yes, all doses	yes, all doses	squamous cell papilloma in 21.5%+TPA 1/36 males 43% + TPA 1/39 males 85%+TPA: 1/30 males	no data	no	BASF 1992

* Treatment with the promoter O-tetradecanoylphorbol-13-acetate (TPA) for 20 weeks after a 2 months treatment - free period.
Ø No treatment-related effects on the mortality rate.

Table 4.17 Oral carcinogenicity study on Acrylic acid (AA)

Species/strain no. of animals/ sex/group	Exposure time	Treatment schedule	Mortality rate	Treatment- related tumour response	Study design according to the B32 / B33 method	Reference
rat/ Wistar, 50/sex/ group	26/28 months	120, 400, 1,200 ppm AA in drinking water	Ø	no	yes	BASF, 1989

Ø No treatment-related effects on the mortality rate and mean survival time

Table 4.18 Dermal carcinogenicity studies with Acrylic acid (AA)

Species/strain no. of animals/ sex/group	Exposure time	Treatment schedule	Mortality rate	Skin irritation	Skin hyper plasia	Skin tumours	Tumour response of internal organs	Study design according to the B32 / B33 method	Reference
Mouse/C3H/ HeJ 40males	life time	25 µl AA (1% v/v in acetone)	Ø	no	1/40	0/40	no	no	Intercompany Acrylate Study Group, 1982
Mouse/ C3H/ HeN Hsd BR, 50/sex/ group	life time	25 or 100 µl AA (1% v/v in acetone)	Ø	no	0/50 for each sex	0/50 for each sex	100µl AA: 7/50 females with lympho sarcoma	no	BAMM, 1990 BAMM ,1991
Mouse/Hsd: (ICR)BR 50/sex/ group	life time (86-92 weeks)	25 or 100 µl AA (1% v/v in acetone)	Ø	no	0/50 for each sex	0/50 for each sex	no	no	BAMM ,1990 BAMM, 1991

Ø No treatment-related effects on the mortality rate and mean survival time

4.1.2.9 Toxicity for reproduction

4.1.2.9.1 Studies in animas

Fertility impairment

There are no generation studies, and fertility studies on 2-EHA available.

Data on reproductive organ toxicity (testes weights as well as information on gross and microscopic pathology for testes, seminal vesicles, ovaries, and uteri can be derived from a 3 month inhalation study (10, 30, 100 ppm 2-EHA, 6 hours/day, 10 animals/sex/dose level) with Wistar rats according to OECD Guideline 413 (BASF, 1989). Respective results from animals exposed to the highest dose level of 100 ppm (approximately 0.750 mg/l) did not give evidence for any impairment of the investigated reproductive organs of both sexes.

Developmental toxicity

Developmental toxicity studies with the oral route of administration are not available.

2-EHA was investigated during a study on the relative developmental toxicities of a set of various acrylates (acrylic acid, methyl acrylate, butyl acrylate, hydroxyethyl acrylate, hydroxypropyl acrylate) in Sprague-Dawley rats (Saillenfarth et al., 1999). For the investigation with 2-EHA groups of 23 to 25 dams were exposed (6 hours/day, whole-body) to atmospheres containing 2-ethylhexyl acrylate (99.7% purity) at 0, 50, 75, and 100 ppm (approximately 0.375, 0.563, and 0.750 mg/l) during day 6 to day 20 of gestation. From preliminary level-setting studies (no details available) a level of 100 ppm 2-EHA had been reported to provide the highest reliable vapour concentration. Maternal food consumption was measured for the intervals of g.d. 6-13 and of g.d. 13-21. Maternal body weights were recorded on g.d. 0, 6, 13, and 21. Dams were sacrificed on day 21 of gestation and the uteri were removed and weighed. The number of implantation sites, resorptions, and dead and live fetuses were recorded. Uteri, which had no visible implantation sites, were stained with ammonium sulfite (10%) for the detection of early resorptions. At sacrifice live fetuses were weighed, sexed, and examined for external anomalies including those of the oral cavity. Half of live fetuses from each litter were examined for either internal soft tissue or for skeletal changes.

There were no maternal deaths in any of the treatment groups. Dams from the 100-ppm groups showed an absolute weight gain of 24±16 g through the period of exposure, which was lower and statistically significantly different from that of the concurrent control group (42±11 g). Also food intake of 24±3 g food/dam/day through the period of exposure of the 100-ppm group was somewhat lower and statistically significantly different in comparison to that of the concurrent control group (27±2 g food/dam/day). No adverse effects were observed on the mean number of implantation sites per litter and on the mean number of live fetuses per litter in any of the 2-EHA exposed groups. The incidences of non-live implants (3.7-6.4%) and of resorption sites per litter (3.7-6.1%) in the treated groups were lower than those of the concurrent control (both 10.1%). This observation, however, is not considered to be of toxicological significance. Mean fetal body weights were slightly lower in the treated groups, however not statistically significantly different from that of the concurrent control fetuses. Sex ratio was unaffected. No significant differences were observed between the control and the 2-EHA-treated groups in the incidences of gross anomalies or of visceral or skeletal malformations or variations.

In summary, no embryotoxic, teratogenic or fetotoxic properties of 2-EHA had been revealed from this study for concentrations of up to and including 100 ppm. Due to technical limitations exposure to higher concentrations could not be tested. Based on slightly reduced food intake and lower maternal weight gain at the higher exposure level a NOAEC/maternal toxicity of 75 ppm (approximately 0.563 mg/l) is derived from this study. No embryo-/fetotoxic effects were revealed even at the highest tested concentration at which some signs of maternal toxicity had been observed. Therefore, a NOAEC/developmental toxicity of 100 ppm (approximately 0.750 mg/l) is derived from this study.

4.1.2.9.2 Studies in humans

No data available.

4.1.2.9.3 Conclusion

There are no human data available on the reproductive toxicity of 2-EHA. The available data base for hazard assessment of toxicity for reproduction from animal testing consists of data from a 3 month repeated dose study (rats) and a developmental toxicity study (rats), both with the inhalatory route of administration. According to the TGD (Chapter 2, Section 3.12) these data should be considered a sufficient investigation of reproductive toxicity of 2-EHA for screening purposes. Evaluation of the available screening information so far does not provide evidence for significant reproductive toxicity of 2-EHA. For doses up to and including 100 ppm (approximately 0.75mg/l) so far no adverse effects on reproductive organs (organ weight, histopathology) and on embryo-/fetal development had been observed.

4.1.3 Risk characterisation

4.1.3.1 General aspects

2-Ethylhexyl acrylate (2-EHA) is rapidly and extensively absorbed, distributed and eliminated after oral administration. There are no specific toxicokinetic studies using dermal administration or exposure by inhalation available. Studies on rats have indicated that short-chain acrylates such as 2-EHA undergo carboxylesterase-catalysed hydrolysis to acrylic acid and 2-ethylhexanol.

Human data on the acute toxicity of 2-EHA are not available. In animal tests, single oral or dermal administration or inhalation of saturated atmospheres of 2-EHA demonstrated only low toxicity. Acute oral toxicity in rats is characterised by LD_{50} values of 4,000-6,000 mg/kg with slight toxic effects (scant droppings, wet yellow stained anogenital area, decreased spontaneous motoric activity and ataxia). For rabbits, a dermal LD_{50} value > 10,000 mg/kg is reported. Valid data on acute inhalation toxicity tests are not available. In a test with rats, after an 8-hour inhalation of an atmosphere saturated with EHA at 20°C no mortality and no clinical signs were observed. The substance is not to be labelled because of acute toxic effects.

Information on human experience with local irritation/corrosion caused by 2-ethylhexyl acrylate is not available. In animal experiments 2-EHA caused serious lesions to the skin of rabbits which are assessed to be situated at the border between severe irritation and corrosion. As an alternative to the Draize skin irritation test the new test method according to the EU test Guideline B.40

(Skin Corrosion) has been developed for differentiation between irritation and corrosion. The result of a new study according to this guideline demonstrates that 2-EHA does not have a corrosive potential, and hence, the current classification of 2-EHA as irritant and labelling with "R 38, Irritating to skin" is confirmed. 2-EHA caused mild eye irritation in animal experiments. On the basis of these tests a labelling with R 36 is not warranted.

There exists no standard test method for the assessment of respiratory irritation. Thus, the labelling of 2-EHA with "R 37, Irritating to respiratory tract" according to current EU regulations is not based on results of a specific respiratory irritation test, but on considerations on the general irritation potential of 2-EHA (nasal and ocular irritation noted in a test on acute inhalation toxicity with rats, severe local irritation potential detected on the skin and moderate irritation potential detected on the conjunctivae of rabbits; serious lesions as seen after repeated inhalation of 2-EHA may well be initiated i.a. by primary respiratory irritation). Thus, labelling with R 37 is confirmed on the basis of all of the respective data.

Positive patch-tests are reported for humans. In various test models involving guinea pigs, 2-EHA proved sensitising, with and without adjuvants. 2-EHA has a moderate sensitising potential in experimental animals. Information on respiratory sensitisation is not available. According to the data 2-EHA is classified with "R 43, May cause sensitisation by skin contact".

The relevant toxic effect after 90-day inhalation exposure of rats to 2-EHA was dose-related increased degeneration of the olfactory epithelium at concentrations from 30 ppm and higher (0.225 mg/l). The NOAEC for local effects on the respiratory tract was 10 ppm (0.075 mg/l). Animals exposed to 2-EHA concentrations of 30 ppm or higher showed poor health condition (lethargy, ptosis) during exposure period and reduced body weight gain, but no toxic effect on internal organs was identified (NOAEC for systemic effects). Minimal liver damage was indicated by elevated liver enzyme activities at a concentration of 100 ppm (0.75 mg/l). Valid studies with dermal or oral application routes are not available. Cancer studies and less documented subchronic studies with dermal application revealed that 2-EHA causes skin irritation at concentrations $\geq$ 2.5% (LOAEL).

2-EHA is negative in bacterial mutation tests. Data from mammalian cells give no relevant evidence for clastogenicity; however, a fully reliable study is lacking. 2-EHA seems to have a low potential for induction of gene mutations in mammalian cells. Since this effect is limited to doses with strong cytotoxicity, it is highly unlikely that this potential will be expressed *in vivo*. The data from mammalian cell indicator tests do not add relevant information. An *in vivo* cytogenetic assay was inconclusive (neither positive nor negative); due to severe methodological insuffiencies this study cannot be used for evaluation purposes. Cleavage products of 2-EHA were negative in *in vivo* mutagenicity tests. Taken together, the number of studies available from various tests systems and the negative data on 2-EHA cleavage products are regarded as a sufficient substitute for a fully reliable *in vitro* chromosome aberration test (minimum requirements for genotoxicity testing). From all these data there is no relevant evidence that 2-EHA might be an *in vivo* mutagen.

There are no data available to the carcinogenic effects with respect to oral or inhalation exposure routes. Findings from the dermal mouse carcinogenicity study showed that 2-EHA induces skin tumours at concentrations which were highly irritative. However, other studies on different mouse strains did not confirm this finding. Acrylic acid, the hydrolysis product, did not induce tumours in mice treated dermally and in rats administered orally. Also, there is no concern from cancer data on 2-ethylhexanol. It is concluded that equivocal results from mice painting studies give no significant evidence of carcinogenic properties of 2-EHA.

There are no human data available on the reproductive toxicity of 2-EHA. From animal testing screening information on reproductive toxicity is available from a developmental toxicity study supplemented with data on reproductive organ toxicity investigations from a 3 month repeated dose study. Evaluation of the available screening information so far does not provide evidence for significant reproductive toxicity of 2-EHA. In rats no adverse effects on reproductive organs or on embryo/fetal development had been revealed for inhalation exposures to 2-EHA at concentrations of up to and including 100 ppm (approximately 0.75 mg/l).

4.1.3.2 Workers

4.1.3.2.1 General aspects of occupational risk assessment

Route specific systemic availabilities

Systemic availability via different routes has to be considered, since the assessment of inhalation and dermal exposure is partly based on studies that were not conducted with the relevant route of exposure.

Concerning the oral route a high systemic availability can be concluded from Section 4.1.2.1. Only 1% of C14 labelled 2-EHA was detected in faeces. There is no information available on the dermal and inhalation route and as default assumption an equivalent availability is used for risk assessment.

The following assumptions on systemic availability are taken forward for the calculation of MOS.

Systemic availability after oral intake:	approximately 100% (experimental data)
Systemic availability after dermal contact:	approximately 100% (default assumption)
Systemic availability after inhalation:	approximately 100% (default assumption)

Occupational exposure and internal body burden

Inhalation exposure to vapours and skin exposure are the relevant routes of occupational exposure. Workplace exposure is expected during production and polymerisation of 2-EHA (Scenario 1), formulation of preparations containing up to 21% 2-EHA (Scenario 2), use of formulations containing monomeric 2-EHA in the building trade (Scenario 3) and use of dispersions with residual 2-EHA (< 0.08%) (Scenario 4). In **Table 4.19** the exposure levels of **Table 4.2** are summarised and the route specific and total internal body burden is identified. In case of exposure ranges the higher values are taken forward for the calculation.

Table 4.19 Occupational exposure levels and internal body burden

Area of production and use		Frequency	Inhalation shift average in mg/m³	Dermal shift average in mg/kg/d(2)	Internal body burden(1) in mg/kg/day		
					Inhalation(4)	Dermal	Combined
1	Production and polymerisation of 2-EHA	not daily	2.8(3)	0.15	0.4	0.15	0.55
		daily		-	0.4	-	0.4
2	Formulation of preparations containing up to 21% 2-EHA (without LEV)	not daily	19(2)	0.15	2.7	0.15	2.85
		daily		-	2.7	-	2.7
3	Use of formulations containing monomeric 2-EHA in the building trade	not daily	3(2)	13	0.42	13	13.4
		daily	-	-	-	-	-
4	Use of dispersions with residual 2-EHA (< 0.08%)	not daily	< 1	0.042	0.14	0.042	0.18
		daily					

1) Based on the assumption of 100% systemic availability; breathing volume of 10 m³ per 8 hour; body weight: 70 kg
2) EASE
3) Highest 95th percentile of measurements (reasonable worst case)
4) Shift average x 10 m³/70 kg

Calculation of MOS values

Irritation after inhalation and repeated dose toxicity are assessed on the basis of MOS values. MOS values of irritation after inhalation and repeated inhalation are calculated with the NOAEC of an inhalation study and the occupational exposure concentration. Since a repeated dermal study is not available, systemic effects after dermal exposure are assessed on the basis of calculated internal body burden derived from the inhalation NOAEC and dermal exposure.

The following default values of body weights and physiological parameters are used for the calculation of MOS.

Body weight, rat	250 g
Body weight, worker	70 kg
Respiratory volume of rats	0.8 l/min/kg
Respiratory volume of worker during 8 hours of light activity	10 m³

Evaluation of MOS values

According to TGD (Chapter 4) several aspects have to be considered to decide on the acceptability of MOS values. A minimal MOS is derived by the multiplication of subfactors, that are described below and under the toxicological endpoints. Based on the minimal MOS and the toxicological starting point (e g NOAEC/L) a critical exposure concentration/level is calculated.

Differences in exposure route

An equivalent systemic availability of 100% for all routes of exposure is assumed. No specific factor is applied in the risk assessment.

Differences in exposure duration

Since the assessment of acute and chronic occupational exposure is based on a subchronic study, differences in exposure duration have to be considered. The factors are explained under the respective endpoints.

Interspecies differences

The assessment is mainly based on animal data. Substance specific experimental and human data allowing a quantitative assessment of interspecies differences are not available.

Concerning the local effects in the nose it is known that rodents show a nasal anatomy and respiratory physiology different from man. These differences will influence the toxicokinetics of substances in the upper respiratory tract. A further important point is the hydrolysis of the ester. Release of acrylic acid in the olfactory epithelium is presumed to be an important cause of site-specific effects and the carboxylesterase activity, responsible for the cleavage might be different in rats and humans. However it is not known whether these species differences lead finally to marked sensitivity differences of rats and humans. For that reason a species extrapolation factor of 1 is used.

The assessment of systemic effects relies upon the concept of metabolic rate scaling, because substance specific information on interspecies differences is not available. For inhalation exposure, this principle implies that a specific inhalation exposure level (in mg/m³) is toxicologically equivalent in rats and humans (if the duration of exposure and the status of physiological activity are identical). For interspecies extrapolation of oral or dermal data metabolic rate scaling results in 4-times and 7-times lower effective dose levels in humans (in mg/kg/day) compared to rats and mice.

Adjustment for breathing volume of workers (light activity for 8 hours/day)

For the assessment of local and systemic effects via inhalation the following aspect is considered in the risk assessment. In inhalation studies with repeated administration rats are routinely exposed for 6 hours per day; the respiratory minute volume for the rat is assumed to be 0.8 l/min/kg. Metabolic rate scaling implies that the human NAEL (in mg/p/day) is calculated based on a daily exposure of 6 hours, a human respiratory rate of 0.2 l/min/kg (which is determined by the scaling model) and the experimental NOAEC in mg/m³. Thus, the metabolic rate scaling model determines the human NAEL. A breathing rate of 0.2 l/min/kg for 6 hours is identical to a breathing volume of 5 m³ for a person of 70 kg. That implies a human NAEL (in mg/p/day) that results from the NOAEC in mg/m³ multiplied with 5 m³.

For risk characterisation purposes however, a daily breathing volume of 10 m^3 is assumed for workers (8 hour exposure and light activity). According to Haber's law the toxicological consequence of breathing 10 m³ is different from breathing 5 m³ of the same contaminated air. Thus, for evaluation of direct MOS values, based on the experimental NOAEC (experimental animal, 6 hours per day) and assuming a human breathing volume of 10 m³, a factor of 2 is used for adjustment for breathing volumes.

Further aspects (e. g. intraspecies variability)

Further relevant parameters of MOS evaluation are not covered by scientifically based adjustment factors and are included in a further uncertainty factor. Especially intraspecies variability, but also nature and severity of effects, dose-response relationship, variability in the

experimental data and overall confidence in the database have to be considered. A standard uncertainty factor of about 5 is proposed when risk assessment is based on oral animal data and an available NOAEL. The uncertainty factor may be lower in case of additional relevant data (e.g. human data available, route-to-route extrapolation not necessary) or in case of adverse effects that are not considered severe. The uncertainty factor usually is higher than 5 for e.g. specific reproductive toxicity or lack of NOAEL. The uncertainty factors for the single endpoints are described in the respective chapters.

4.1.3.2.2 Endpoint-specific risk assessment for workers

Acute toxicity

Inhalation

No lethality was observed in rats after 8 hours exposure to a vapour-saturated atmosphere at 20°C (room temperature). Gross pathology revealed nasal and ocular irritation. The calculated saturation concentration for the vapour pressure of 12-17 Pa (20°C) would be approximately 920-1,310 mg/m^3 (120-170 ppm).

This value is compared with the highest estimated inhalation exposure of 77 mg/m^3 (2-hour, EASE, Scenario 2) and 19 mg/m^3 (8-hour, EASE, Scenario 2). As to acute effects concern is not derived.

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.

Dermal

A dermal LD_{50} of approximately 14,000 mg/kg was determined in rabbits. For comparison the oral LD_{50} for rats and mice lies between 4,000 and 6,000 mg/kg. Comparing the dermal LD_{50} of approximately 14,000 mg/kg with the highest acute dermal exposure of about 13 mg/kg (880 mg/person, Scenario 3) concern is not derived.

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

Dermal

2-EHA is strongly irritating to the skin of rabbits in studies on acute irritation, but should not be considered as corrosive (See Section 4.1.2.3). 2-EHA induced local effects at the skin in several subchronic and chronic dermal studies with different mice strains. In a chronic study with male CH3/HeJ-mice a 2.5% solution (3 days/week, lowest concentration) led to transient skin irritations. Concentrations of 21% and 86.5% were highly irritative and induced skin tumours (see Section 4.1.2.6 and 4.1.2.8).

Conclusion (ii) is proposed on the grounds that control measures exist which can minimise exposure and risk of irritation/corrosivity, thereby reducing concern. However, these controls must be implemented and complied with to reduce the risk of damage to skin.

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.

Eyes

Eye irritation is reported to be evident but less significant than local effects on the skin. The mild and reversible eye irritation does not warrant labelling with R 36. Concern as to eye irritation is not derived.

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

2-EHA is considered to be a respiratory irritant based on the results of the acute inhalation testing, skin and eye irritation studies and investigations on repeated inhalation (see Section 4.1.2.3). There are no experimental data to describe a precise threshold for respiratory irritation of single exposures, but some information can be derived from a single 8-hour exposure of rats to a vapour-saturated atmosphere. The calculated saturation concentration for the vapour pressure of 12-17 Pa (20°C) would be approximately 920-1,310 mg/m^3 (120-170 ppm). Gross pathology revealed nasal and ocular irritation, a NOAEC was not determined. Subchronic inhalation exposure of rats demonstrated a NOAEC of 77 mg/m^3 (10 ppm) and minimal degeneration of the olfactory epithelium at 230 mg/m^3 (30 ppm). Due to the limited reliability of the acute inhalation study and the uncertainties about the exposure concentration the subchronic NOAEC of 77 mg/m^3 (10 ppm) is used for the MOS calculation.

For the selection of a minimal MOS the following subfactors are applied:

- duration adjustment (subchronic to acute): 1/3
- Data from acute inhalation with limited reliability indicate that the NOAEC for single exposures and histopathological effects should not be substantially higher than the subchronic NOAEC. In addition other substances like methyl methacrylate and vinyl acetate that affected also the olfactory epithelium, showed no very marked change comparing acute and subacute/subchronic NOAECs. A factor of 1/3, that implies a 3-fold higher acute NAEC compared to the subchronic one, is considered to be appropriate.
- adjustment of breathing volumes (6 to 8 hours; light activity of workers): 2
- interspecies adjustment: 1
- a further uncertainty factor of 3 is considered appropriate to cover intraspecies variability, the nature and severity of effect (minimal nasal effects) and the quality of the database (NOAEC and LOAEC were determined) (see also above under “further aspects”).

A minimal MOS of 2 is derived which results in a critical exposure concentration of 39 mg/m^3 (8 hours). Comparing this concentration with the highest 8-hour concentration of 19 mg/m^3 concern is not derived. Due to the 4-fold reduced exposure time the short term exposure of 77 mg/m^3 for 2 hours (Scenario 2) is also not considered to be 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.

Sensitisation

Dermal

2-EHA is moderately sensitising in guinea pigs. Sensitisation has also been reported in humans, however human data also indicate that 2-EHA has not been an important occupational contact allergen during 1978-1999 (Geukens and Gossens, 2001). This implies that very low concentrations of 2-EHA (< 0.08% in the widespread applicated polymer dispersions) should not be expected to have a considerable sensitising potency in humans.

Since also single contacts might lead to skin sensitisation concern is raised for Scenario 1, 2 and 3. Concern is not expressed as to Scenario 4 because of the very low 2-EHA-concentration in combination with the lacking indications from a comprehensive human survey.

Conclusion (iii) There is a need for limiting the risks: risk reduction measures which are already being applied shall be taken into account. (Scenario 1, 2 and 3)

Inhalation

Data on respiratory sensitisation in man (e.g. case reports) and in experimental animals is not available. Some potential of 2-EHA to cause respiratory sensitisation cannot be excluded with certainty since in the substance demonstrated skin sensitising properties. However at the background of occupational exposure in former years 2-EHA seems at least not to be a strong respiratory sensitiser in humans. For the time being no generally accepted animal model is available which would be able to verify the question of respiratory sensitisation. Concern is not expressed.

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

Inhalation (local effects)

A NOAEC of 77 mg/m^3 (10 ppm) was determined in a subchronic inhalation study on rats (6 h/d). Minimal degeneration was observed in the olfactory epithelium of the nose at 230 mg/m^3 (30 ppm). At 770 mg/m^3 (100 ppm) the degeneration was more marked. The NOAEC of 77 mg/m^3 (10 ppm) is used for the MOS calculation (see **Table 4.20**).

For the selection of a minimal MOS the following subfactors are applied:

- duration adjustment (subchronic to chronic): 2
- The application of a duration adjustment is confirmed by the structurally related compound butyl acrylate. Butyl acrylate was preferred to the other available acrylate studies (methyl or ethyl acrylate) due to the longer and so more comparable side chain. A chronic inhalation study (with interim sacrifices at 12 months of exposure, Reininghaus et al. (1991) showed a change of dose response relationship with time. A default value of 2 is applied (Kalberlah et al., 1999).
- adjustment of breathing volumes (6 to 8 hours; light activity of workers): 2
- interspecies adjustment: 1

- a further uncertainty factor of 3 is considered appropriate to cover intraspecies variability, the nature and severity of effect (minimal nasal effects) and the quality of the database (NOAEC and LOAEC were determined) (see also above under "further aspects").

A minimal MOS of 12 is derived which results in a critical exposure concentration of 6.4 mg/m^3. Concern is raised for Scenario 2.

Conclusion (iii) There is a need for limiting the risks: risk reduction measures which are already being applied shall be taken into account. (for Scenario 2)

Table 4.20 MOS values of repeated dose toxicity and scenarios with daily exposure (local and systemic effects via inhalation)

			Local effects		Systemic effects	
Starting point for MOS calculation			77 mg/m^3		230 mg/m^3	
Minimal MOS			12		12	
Critical exposure concentration			6.4 mg/m^3		19 mg/m^3	
		Exposure (mg/m^3)	MOS	Conclusion	MOS	Conclusion
1	Production and polymerisation	2.8	28	ii	82	ii
2	Formulation of preparations containing up to 21% 2-EHA	19	4	iii	12.1	ii
4	Use of dispersions with residual 2-EHA (< 0.08%)	< 1	> 77	ii	> 230	ii

Inhalation (systemic effects)

Based on the above mentioned inhalation study a systemic NOAEC of 230 mg/m^3 (30 ppm) was determined. At 770 mg/m^3 (100 ppm) elevated activities of transaminase and alkaline phosphatase indicated a minimal liver damage. The NOAEC of 230 mg/m^3 (30 ppm) is used for the MOS calculation (see **Table 4.20**).

For the selection of a minimal MOS the following subfactors are applied:

- duration adjustment (subchronic to chronic): A default value of 2 is used (Kalberlah and Schneider, 1998).
- adjustment of breathing volumes (6 to 8 hours; light activity of workers): 2
- interspecies adjustment: 1
- a further uncertainty factor of 3 is considered appropriate to cover intraspecies variability, the nature and severity of effect (indications for a minimal liver damage) and the quality of the database (NOAEC and LOAEC were determined) (see also above under "further aspects").

A minimal MOS of 12 is derived which results in a critical exposure concentration of 19 mg/m^3. Scenario 2 is a borderline scenario (MOS: 12.1), but it is not considered to be appropriate to raise 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.

Dermal (local effects)

A quantitative risk characterisation can be made for local effects after repeated dermal exposure in Scenario 4 using the chronic study from Wenzel-Hartung (1989) with male CH3/HeJ-mice. Transient skin irritations were observed at the LOAEL of 2.5% solution (25 microliter, 3 days/week, lowest concentration). Assuming that the body surface of a mouse is approximately 40 cm^2 and that 10% of the body surface (4 cm^2) was exposed an effect level of approximately 0.2 mg/cm^2 can be calculated. Comparing this area dose with the assumed daily exposure of 0.004 mg/cm^2 a MOS of approximately 50 is derived which is considered to be sufficiently high to derive no 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.

Dermal (systemic effects)

Valid experimental data for the assessment of systemic toxicity by skin contact is not available. A long term dermal study in male mice with limited histopathological examination (Wenzel-Hartung (1989); Brune and Deutsch-Wenzel (1986)) showed that the highest dose tested (25 µl 86.5% 2-EHA (3 days/week), approximately 1,081 mg/kg/day) is probably without severe systemic effects.

The subchronic inhalation study with rats is however preferred as starting point for MOS calculation, since it is regarded as valid (see Section 4.1.2.6) and included histopathological examinations in both sexes. The NOAEC of 230 mg/m^3 (30 ppm) is used for the MOS calculation (see **Table 4.21**). The NOAEC of 230 mg/m^3 (30 ppm) corresponds to an intake by inhalation of 66 mg/kg/day (respiratory rate of 0.8 l/min/kg for rats), that is used as internal NAEL for MOS calculation (see **Table 4.21**).

For the selection of a minimal MOS the following subfactors are applied:

- duration adjustment (subchronic to chronic): A default value of 2 is used (Kalberlah and Schneider, 1998).
- interspecies adjustment (metabolic rate scaling): 4
- a further uncertainty factor of 3 is considered appropriate to cover intraspecies variability, the nature and severity of effect (indications for a minimal liver damage) and the quality of the database (NOAEC and LOAEC were determined) (see also above under “further aspects”).

A minimal MOS of 24 is derived which results in a critical exposure level of 2.8 mg/kg/day. A chronic and daily exposure was only estimated for Scenario 4. No concern is derived.

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.

Combined inhalation and dermal exposure

The combined exposure values are listed in **Table 4.19** and the respective MOS in **Table 4.20**. The minimal acceptable MOS is the same as for dermal exposure. Scenario 2 is a borderline scenario (MOS: 24.4), but it is not considered to be appropriate to raise 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.

Table 4.21 MOS values of repeated dose toxicity with daily exposure (dermal and combined exposure)

		Dermal			Combined		
Starting point for MOS calculation		66 mg/kg/day			66 mg/kg/day		
Minimal MOS		24			24		
Critical exposure level		2.8 mg/kg/day			2.8 mg/kg/day		
		Exposure (mg/kg/day)	MOS	Conclusion	Combined Exposure (mg/kg/day)	MOS	Conclusion
1	Production and further processing in the large-scale chemical industry	-	-	-	0.4	170	ii
2	Manufacturing of formulations (< 21% 2-EHA) in the large-scale chemical industry	-	-	-	2.7	24.4	ii
4	Use of dispersions containing residual 2-EHA (< 0.08%)	0.042	1,600	ii	0.18	370	ii

Mutagenicity

Based on data on 2-EHA and related compounds 2-EHA is not considered to be an *in vivo* mutagen (see Section 4.1.2.7). Corresponding risks at workplaces are not anticipated to occur.

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.

Carcinogenicity

Dermal

Skin tumours were observed in chronic dermal studies in C3H/HeJ-mice, but not in NMRI-mice at concentrations of 21% and above. The carcinogenic effect is considered to be associated with the highly irritating concentrations tested. Regarding additionally the negative *in vivo* mutagenicity tests and data from related compounds skin tumours are not expected at exposure conditions that do not result in strong chronic irritation (please refer also to the section "Irritation/dermal"). Based on the available data it is concluded, that 2-EHA induces skin tumours by a non-genotoxic mechanism.

The daily dermal exposure is assumed to be negligible (Scenarios 1, 2) or up to 0.04 mg/kg/day (3 mg/person/day, Scenario 4). The non-daily exposure can reach 13 mg/kg/day (Scenario 3). Overall a strong chronic irritation that might lead to skin tumours is not expected in all scenarios.

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

Experimental data for the assessment of carcinogenicity by inhalation is not available. Taking account of the negative *in vivo* mutagenicity and of negative long term inhalation studies of specific acrylates/methacrylates 2-EHA is not suspected to be carcinogenic by inhalation. Corresponding risks at workplaces are not anticipated to occur.

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.

Fertility impairment

A fertility study on 2-EHA is not available, but in the 90-day inhalation study with rats no effect was observed in reproductive organs (testes, seminal vesicles, ovaries, uteri) up to the highest concentration of 770 mg/m^3 (100 ppm). Comparing this NOAEC with the NOAECs of repeated dose toxicity in the same study (local effects: NOAEC of 77 mg/m^3 (10 ppm), systemic effects: 230 mg/m^3 (30 ppm)) fertility impairment is not expected as a specific effect independent of general toxicity. A MOS-calculation is not performed, since no indication of an effect on reproductive organs was observed.

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

In a study on developmental toxicity in rats no adverse effect on embryo/fetal development was observed up to the highest tested concentration of 770 mg/m^3 (100 ppm). At this concentration a slightly reduced food intake and lower maternal weight gain was observed, leading to a NOAEC for maternal toxicity of 580 mg/m^3 (75 ppm). Developmental toxicity is not expected as a specific effect independent of general toxicity. A MOS-calculation is not performed, since no effect on development was observed.

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.

4.1.3.2.3 Summary of occupational risk assessment

In the following **Table 4.22** the results of the toxicological endpoints and scenarios with **conclusion (iii)** are summarised. All other endpoints resulted in **conclusion (ii)**.

Table 4.22 Summary of conclusions for the occupational risk assessment of 2-EHA

Area of production and use		Sensitisation Dermal	Repeated dose toxicity local effects after inhalation
1	Production and polymerisation of 2-EHA	iii	ii
2	Formulation of preparations containing up to 21% 2-EHA	iii	iii
3	Use of formulations containing monomeric 2-EHA in the building trade	iii	ii
4	Use of dispersions with residual 2-EHA (<0.08%)	ii	ii

4.1.3.3 Consumers

Exposure

The measured maximum air concentration during short-term use (painting) was 1 ppm (and 0.0075 mg/l). Taking into account that monomeric 2-EHA was not detectable 25 hours after

painting; there is no reasonable suspicion for repeated exposure by inhalation for consumers after painting. Dermal exposure is considered to be negligible (maximum 11.2 µg/kg bw per event).

Acute toxicity

Following the exposure assessment, consumers are not exposed to 2-EHA in the range of doses which can be derived from acute oral toxicity figures based on animal LD_{50} values (LD_{50} oral (rats, mice): 4,000-6,000 mg/kg). In an experiment with rabbits, the substance has demonstrated very low dermal toxicity (LD_{50} dermal: (rabbits): > 10,000 mg/kg). Following inhalation exposure in rats there were no deaths in a saturated 2-EHA- atmosphere up to 8 hours. The substance is assumed not to justify concern for the consumer in relation to acute oral, inhalation or dermal toxicity.

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

2-EHA caused severe irritation near corrosion after application to the skin of rabbits. Eye irritation was less severe in animal experiments.

The concentration of 2-EHA in the final products for consumer (0.08%) is under the concentration limit which would lead to classification and labelling.

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.

Sensitisation

2-EHA has a moderate sensitising potential in experimental animals. There is also evidence of sensitisation in humans.

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

Following the exposure assessment there is no real chronic exposure to 2-EHA.

Taking into account that monomeric 2-EHA was not detectable 25 hours after painting; there is no reasonable suspicion for repeated exposure by inhalation for consumers after painting. Dermal and oral exposure is considered to be negligible.

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.

Mutagenicity

The bacterial mutation assays was negative, but various *in vitro* studies on mammalian cell cultures produced weakly mutagenic effects. No mutagenic effect was observed in an *in vivo* chromosomal aberration test after treatment with high doses of 2-EHA. Cleavage products of 2-EHA were negative in *in vivo* mutagenicity tests. The overall data-base on structurally related

acrylic compounds supports that there is no *in vivo* mutagenicity. The substance is of no concern in relation to mutagenic effects.

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.

Carcinogenicity

Consumer exposure is considered to be negligible. Based on the exposure assessment, there is no chronic exposure to 2-EHA.

There are no data on carcinogenic effects of 2-EHA with respect to oral or inhalative exposure routes. There are data that 2-EHA produced skin tumours in animals; this was found, however, only in case of established irritative skin lesions. Based on the limited actual knowledge on 2-EHA but taking into account the negative results from long term carcinogenic studies with acrylic acid after oral and dermal administration no concern can be assumed.

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.

Toxicity for reproduction

Following the exposure assessment, consumer exposure is considered to be negligible. Evaluation of the available screening information about 2-EHA does not provide evidence for significant reproductive toxicity of 2-EHA up to 750 mg/m³. As to the relevant metabolites of this compound, for 2-ethylhexanol and for acrylic acid there are no indications for a specific embryo-/fetotoxic or teratogenic potential and for acrylic acid there are no indications for a substance-induced impairment of reproductive functions.

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.

4.1.3.4 Man exposed indirectly via the environment

The main route of indirect exposure in the local scenario is the intake via air, on a regional scale a predominant intake via the consumption of fish is expected.

Repeated dose toxicity

Local scenario

Following the local scenario data (at a point source) a concentration of 8.3 µg/m^3 2-EHA in the air is calculated. The most sensitive effect of 2-EHA in animals was degeneration of the olfactory epithelium in a 90-day inhalation study on rats. The local NOAEC in this study was 0.075 mg/l (and 75 mg/ m³).

Comparison indirect exposure - Local scenario/local effect/NOAEC

Indirect exposure (local)	0.0083 mg/m^3
NOAEC	75 mg/ m^3

The margin of safety expressed by the magnitude between the calculated exposure and the NOAEC is high for the local scenario.

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.

Regional scenario/systemic effect

For the regional scenario (mainly consumption of fish and root crops), the respective figure is 6 ng/kg bw/day. In repeated dose toxicity studies on rats (90-day inhalation) the NOAEC for systemic effects was 30 ppm (and 0.225 mg/l). This NOAEL is converted as follows to the inhaled amount of the substance using the respiratory minute volume 1.3 l/min/kg and exposure duration of 360 min/day:

0.225 mg/l · 0.8 l/minutes/kg · 360 minutes/day = 65 mg/kg bw/day

Comparison indirect exposure - Regional scenario/NOAEL

Indirect exposure (regional)	0.000006 mg/kg bw/day
NOAEL	65 mg/kg bw/day

The margin of safety expressed by the magnitude between the calculated exposure (regional scenario) and the NOAEL is very high for the regional scenario. Thus, 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.

Toxicity for reproduction

Evaluation of the available screening information about 2-EHA does not provide evidence for significant reproductive toxicity of 2-EHA.

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.

4.2 HUMAN HEALTH (PHYSICO-CHEMICAL PROPERTIES)

4.2.1 Exposure assessment

4.2.1.1 Occupational exposure

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

2-ethylhexyl acrylate is not explosive.

4.2.2.2 Flammability

2-ethylhexyl acrylate is not flammable.

4.2.2.3 Oxidising potential

Due to its chemical structure, 2-ethylhexyl acrylate is not expected to possess any oxidising properties.

4.2.3 Risk characterisation

4.2.3.1 Workers

Not applicable

4.2.3.2 Consumers

4.2.3.3 Humans exposed via the environment

5 RESULTS

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.

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:

5.1 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.

2-ethylhexyl acrylate represents, based on the present data configuration, no risk to the environment.

There is therefore at present no need for further testing or gathering of exposure information.

5.2 HUMAN HEALTH

5.2.1.1 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.

5.2.1.2 Workers

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

- The risk assessment reveals concern with regard to local effects after repeated inhalation for the formulation of preparations (Scenario 2).
- Skin sensitisation gives rise to concern for all dermal exposure during production and polymerisation (Scenario 1), the formulation of preparations (Scenario 2) and the use of formulations containing monomeric 2-EHA in the building trade.

5.2.1.3 Humans exposed 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.

5.2.1.4 Risks to human health from physico-chemical properties

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

Conclusion (ii) is reached because there are no risks from physico-chemical properties arising from the use of the substance.

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ABBREVIATIONS

ADI	Acceptable Daily Intake
AF	Assessment Factor
ASTM	American Society for Testing and Materials
ATP	Adaptation to Technical Progress
AUC	Area Under The Curve
B	Bioaccumulation
BBA	Biologische Bundesanstalt für Land- und Forstwirtschaft
BCF	Bioconcentration Factor
BMC	Benchmark Concentration
BMD	Benchmark Dose
BMF	Biomagnification Factor
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)
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.wt	dry weight / dw
dfi	daily food intake
DG	Directorate General
DIN	Deutsche Industrie Norm (German norm)
DNA	DeoxyriboNucleic Acid
DOC	Dissolved Organic Carbon
DT50	Degradation half-life or period required for 50 percent dissipation / degradation
DT90	Period required for 90 percent dissipation / degradation
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]
EbC50	Effect Concentration measured as 50% reduction in biomass growth in algae tests
EC	European Communities
EC10	Effect Concentration measured as 10% effect
EC50	median Effect Concentration
ECB	European Chemicals Bureau
ECETOC	European Centre for Ecotoxicology and Toxicology of Chemicals
ECVAM	European Centre for the Validation of Alternative Methods
EDC	Endocrine Disrupting Chemical
EEC	European Economic Communities
EINECS	European Inventory of Existing Commercial Chemical Substances
ELINCS	European List of New Chemical Substances
EN	European Norm
EPA	Environmental Protection Agency (USA)
ErC50	Effect Concentration measured as 50% reduction in growth rate in algae tests
ESD	Emission Scenario Document
EU	European Union
EUSES	European Union System for the Evaluation of Substances [software tool in support of the Technical Guidance Document on risk assessment]
F(+)	(Highly) flammable (Symbols and indications of danger for dangerous substances and preparations according to Annex 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)
GLP	Good Laboratory Practice
HEDSET	EC/OECD Harmonised Electronic Data Set (for data collection of existing substances)
HELCOM	Helsinki Commission -Baltic Marine Environment Protection Commission
HPLC	High Pressure Liquid Chromatography
HPVC	High Production Volume Chemical (> 1000 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
Koc	organic carbon normalised distribution coefficient
Kow	octanol/water partition coefficient
Kp	solids-water partition coefficient
L(E)C50	median Lethal (Effect) Concentration
LAEL	Lowest Adverse Effect Level
LC50	median Lethal Concentration
LD50	median Lethal Dose
LEV	Local Exhaust Ventilation
LLNA	Local Lymph Node Assay
LOAEL	Lowest Observed Adverse Effect Level
LOEC	Lowest Observed Effect Concentration
LOED	Lowest Observed Effect Dose
LOEL	Lowest Observed Effect Level
MAC	Maximum Allowable Concentration
MATC	Maximum Acceptable Toxic Concentration
MC	Main Category
MITI	Ministry of International Trade and Industry, Japan
MOE	Margin of Exposure
MOS	Margin of Safety
MW	Molecular Weight
N	Dangerous for the environment (Symbols and indications of danger for dangerous substances and preparations according to Annex 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
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
SCHER	Scientific Committee on Health and Environmental Risks
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
vB	very Bioaccumulative
VOC	Volatile Organic Compound
vP	very Persistent
vPvB	very Persistent and very Bioaccumulative
v/v	volume per volume ratio
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

Substance: 2 - (Ethylhexyl) acrylate

melting point:	$MP := 183.15K$
vapour pressure:	$VP := 12 \cdot Pa$
water solubility:	$SOL := 9.6 \cdot mg \cdot l^{-1}$
part. coefficient octanol/water:	$LOGP_{OW} := 3.9$
molecular weight:	$MOLW := 0.184 kg \cdot mol^{-1}$
gas constant:	$R := 8.3143 J \cdot mol^{-1} \cdot K^{-1}$
temperature:	$T := 285 \cdot K$
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

$$HENRY := \frac{VP \cdot MOLW}{SOL} \qquad HENRY = 230 \, Pa \cdot m^3 \cdot mol^{-1}$$

$$\log\left(\frac{HENRY}{Pa \cdot m^3 \cdot mol^{-1}}\right) = 2.362$$

$$K_{air\_water} := \frac{HENRY}{R \cdot T} \qquad K_{air\_water} = 0.0971$$

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

$a := 0.49$ (a,b from TGD, p. 539 "ester")

$b := 1.05$

$K_{OC} := 10^{a \cdot LOGP_{OW} + b} \cdot l \cdot kg^{-1}$ $K_{OC} = 914.113 \cdot l \cdot kg^{-1}$

Suspended matter

$Kp_{susp} := 0.1 \cdot K_{OC}$ $Kp_{susp} = 91.411 \cdot l \cdot kg^{-1}$

$K_{susp\_water} := Fwater_{susp} + Fsolid_{susp} \cdot Kp_{susp} \cdot RHO_{solid}$ $K_{susp\_water} = 23.753$

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

$faktor := 1 + Kp_{susp} \cdot SUSP_{water}$ faktor = 1.0014

Sediment

$Kp_{sed} := 0.1 \cdot K_{OC}$ $Kp_{sed} = 91.411 \cdot l \cdot kg^{-1}$

$K_{sed\_water} := Fwater_{sed} + Fsolid_{sed} \cdot Kp_{sed} \cdot RHO_{solid}$ $K_{sed\_water} = 46.506$

Soil

$Kp_{soil} := 0.02 \cdot K_{OC}$ $Kp_{soil} = 18.282 \cdot l \cdot kg^{-1}$

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

$K_{soil\_water} = 27.643$

Sludge (activated sludge)

$K_{p\_sludge} := 0.37 \cdot K_{OC}$ $K_{p\_sludge} = 338.222 \cdot l \cdot kg^{-1}$

Raw sewage

$K_{p\_sewage} := 0.30 \cdot K_{OC}$ $K_{p\_sewage} = 274.234 \cdot l \cdot kg^{-1}$

biodegradation in different compartments

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

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

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

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

degradation in surface waters

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

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

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

Atmosphere

calculation of CONjunge * SURFaer for the OPS-model

$VPL := \frac{VP}{\exp\left[6.79 \cdot \left(1 - \frac{MP}{285 \cdot K}\right)\right]}$ $VP := wenn(MP > 285 \cdot K, VPL, VP)$ $VP = 12 \cdot Pa$

$Fass_{aer} := \frac{product}{VP + product}$

$Fass_{aer} = 8.333 \cdot 10^{-6}$

degradation in the atmosphere

$kdeg_{air}$ = 0,036 h^{-1}

Distribution in WWTP acc. SimpleTreat 3.0 (debugged version, 7 Feb 97) :

$k=1h^{-1}$

Summary of distribution			
	to air	29,8	
	to water	7,0	
	via primary sludge	7,2	
	via surplus sludge	0,3	
	degraded	55,8	
	total	100,0	%

Smiles	O=C(OCC(CCCC)CC)C=C
Chemical	Propenoic acid, 2-ethylhexyl ester
CAS Number	000103-11-7
Molecular Formula	C11 H20 O2
Molecular Weight	184.28

EPI SUMMARY (v3.10)

Physical Property Inputs	
Water Solubility (mg/L)	9.6
Vapor Pressure (mm Hg)	0.090009
Henry LC (atm-m3/mole)	0.0022705
Log Kow (octanol-water)	3.90
Boiling Point (deg C)	216.00
Melting Point (deg C)	-90.00

Log Octanol-Water Partition Coef (SRC)	
Log Kow (KOWWIN v1.66 estimate)	4.09

Boiling Pt, Melting Pt, Vapor Pressure Estimations (MPBPWIN v1.40)		
Boiling Pt (deg C)	216.92	Adapted Stein and Brown method)
Melting Pt (deg C)	-10.43	(Mean or Weighted MP)
VP(mm Hg,25 deg C)	0.161	(Mean or Weighted MP)
MP (exp database)	-90 deg C	
BP (exp database)	213.5 deg C	
VP (exp database)	1.78E-01 mm Hg at 25 deg C	

Water Solubility Estimate from Log Kow (WSKOW v1.40)	
Water Solubility at 25 deg C (mg/L)	43.24
log Kow used	3.90 (user entered)
melt pt used	-90.00 deg C
Water Sol (Exper. database match)	100 mg/L (25 deg C)
Exper. Ref	CHEM INSPECT TEST INST (1992)

ECOSAR Class Program (ECOSAR v0.99g)
Class(es) found: Acrylates

Henrys Law Constant	(25 deg C)	[HENRYWIN v3.10]
Bond Method	6.72E-004	atm-m3/mole
Group Method	6.00E-004	atm-m3/mole
Exper Database	4.32E-04	atm-m3/mole
Henrys LC [VP/WSol estimate using EPI values]	2.273E-003	atm-m3/mole

Probability of Rapid Biodegradation (BIOWIN v4.00)	
Linear Model	0.9424
Non-Linear Model	0.9982
Expert Survey Biodegradation Results	
Ultimate Survey Model	3.2305 (weeks)
Primary Survey Model	4.0799 (days)
Readily Biodegradable Probability (MITI Model)	
Linear Model	0.7234
Non-Linear Model	0.8610

Atmospheric Oxidation (25 deg C) [AopWin v1.90] Hydroxyl Radicals Reaction	
OVERALL OH Rate Constant	20.1115 E-12 cm3/molecule-sec
Half-Life	0.798 Days (24-hr day; 0.5E6 OH/cm3)
Half-Life	19.146 Hrs
Ozone Reaction	
OVERALL Ozone Rate Constant	0.175000 E-17 cm3/molecule-sec
Half-Life	6.549 Days (at 7E11 mol/cm3)

Soil Adsorption Coefficient (PCKOCWIN v1.66)	
Koc	429
Log Koc	2.632

Aqueous Base/Acid-Catalyzed Hydrolysis (25 deg C) [HYDROWIN v1.67]	
Total Kb for pH > 8 at 25 deg C	1.330E-002 L/mol-sec
Kb Half-Life at pH 8	1.651 years
Kb Half-Life at pH 7	16.512 years

BCF Estimate from Log Kow (BCFWIN v2.14)	
Log BCF	2.303 (BCF = 200.9)
log Kow used	3.90 (user entered)

Volatilisation from Water	
Henry LC	0.00227 atm-m3/mole (entered by user)
Half-Life from Model River	1.735 hours
Half-Life from Model Lake	132.8 hours (5.532 days)

Removal In Wastewater Treatment	
Total removal	57.97 percent
Total biodegradation	0.19 percent
Total sludge adsorption	20.62 percent
Total to Air	37.17 percent

Level III Fugacity Model			
	Mass Amount	Half-Life	Emissions
	(percent)	(hour)	(kg/hour)
Air	4.08	17.1	1,000
Water	26.8	360	1,000
Soil	67.8	360	1,000
Sediment	1.42	1.44e+003	0
Persistence Time			240 hours

Appendix B Calculation of PEClocal for aquatic compartment during production and processing of chemicals at one site

Calculation of PEC_{local} for aquatic compartment during production and processing of chemicals at one site

status: TGD, ESD, IC-3/IC11, generic

$d := 86400 \cdot s$

$a := 365 \cdot d$

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

chemical: (2 - Ethylhexyl) acrylate

Production volume: $T_1 := 70000 \cdot tonne \cdot a^{-1}$

Processing volume: $T_2 := 32000 \cdot tonne \cdot a^{-1}$

Emissionfactor for production (TGD, tab. A1.2): $f_1 := 0.3 \cdot \%$

Emissionfaktor for processing (TGD, tab. A3.10): $f_2 := 0.001 \cdot \%$

Duration of emission for production (TGD, tab. B1.6): $Temission_1 := 300 \cdot d \cdot a^{-1}$

Duration of emission for processing (TGD, tab. B3.9): $Temission_2 := 300 \cdot d \cdot a^{-1}$

Fraction of emission directed to water: (SimpleTreat, k:1h^{-1}; logH:2,36; logK_{ow}:3,9) $Fstp_{water} := 7 \cdot \%$

River flow rate (TGD): $V := 60 \cdot m^3 \cdot s^{-1}$

Factor (1 + K_p * SUSPwater): $FACTOR := 1.0014$

Regional concentration in surface water $PECregional_{water} := 0.0058 \cdot \mu g \cdot l^{-1}$

Emission per day:

$$Elocal_{water} := \frac{T_1 \cdot f_1}{Temission_1} + \frac{T_2 \cdot f_2}{Temission_2} \qquad Elocal_{water} = 701.07 \cdot kg \cdot d^{-1}$$

Concentration in surface water:

$$Clocal_{water} := \frac{Elocal_{water} \cdot Fstp_{water}}{V \cdot FACTOR} \qquad Clocal_{water} = 9.45 \cdot \mu g \cdot l^{-1}$$

$PEClocal_{water}$

$$PEClocal_{water} := Clocal_{water} + PECregional_{water} \qquad PEClocal_{water} = 9.459 \cdot \mu g \cdot l^{-1}$$

Release to wwtp:

$$RELEASE_{wwtp} := T_1 \cdot f_1 + T_2 \cdot f_2 \qquad RELEASE_{wwtp} = 210.32 \cdot tonne \cdot a^{-1}$$

Release to surface water:

$$RELEASE_{sw} := \left(T_1 \cdot f_1 + T_2 \cdot f_2\right) \cdot Fstp_{water} \qquad RELEASE_{sw} = 14.722 \cdot tonne \cdot a^{-1}$$

Annual average local PEC in surface water

$$Clocal_{water\_ann} := Clocal_{water} \cdot \frac{Temission_1}{365 \cdot d \cdot a^{-1}}$$

$$Clocal_{water\_ann} = 7.77 \cdot \mu g \cdot l^{-1}$$

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

$$PEClocal_{water\_ann} = 7.776 \cdot \mu g \cdot l^{-1}$$

Appendix C Calculation of PEClocal for aquatic durino processing of chemicals

Calculation of PEC_{local} for aquatic compartment during processing of chemicals

status: TGD, ESD, IC-11, generic

$d := 86400 \cdot s$

$a := 365 \cdot d$

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

chemical: 2 - (Ethylhexyl) acrylate

Processing volume: $T := 10000 \cdot tonne \cdot a^{-1}$

Emissionfactor for processing (TGD, tab. A 3.10): $f := 0.001 \cdot \%$

Duration of emission for processing (TGD, tab. B 3.9): $Temission := 300 \cdot d \cdot a^{-1}$

Fraction of emission directed to water:
(SimpleTreat, k:1 h^{-1}; logH:2,36; $logK_{ow}$:3,9) $Fstp_{water} := 7 \cdot \%$

River flow rate (TGD): $V := 60 \cdot m^3 \cdot s^{-1}$

Factor (1 + K_p * SUSPwater): $FACTOR := 1.0014$

Regional cocncentration in surface water $PECregional_{water} := 0.0058 \cdot \mu g \cdot l^{-1}$

Emission per day:

$$Elocal_{water} := \frac{T \cdot f}{Temission} \qquad Elocal_{water} = 0.33 \cdot kg \cdot d^{-1}$$

Concentration in surface water:

$$Clocal_{water} := \frac{Elocal_{water} \cdot Fstp_{water}}{V \cdot FACTOR} \qquad Clocal_{water} = 4.49 \cdot 10^{-3} \cdot \mu g \cdot l^{-1}$$

$PEClocal_{water}$

$$PEClocal_{water} := Clocal_{water} + PECregional_{water} \qquad PEClocal_{water} = 0.01 \cdot \mu g \cdot l^{-1}$$

Release to wwtp:

$$RELEASE_{wwtp} := T \cdot f \qquad RELEASE_{wwtp} = 0.1 \cdot tonne \cdot a^{-1}$$

Release to surface water:

$$RELEASE_{sw} := T \cdot f \cdot Fstp_{water} \qquad RELEASE_{sw} = 7 \cdot 10^{-3} \cdot tonne \cdot a^{-1}$$

Annual average local PEC in surface water

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

$$Clocal_{water\_ann} = 3.694 \cdot 10^{-3} \ \mu g \cdot l^{-1}$$

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

$$PEClocal_{water\_ann} = 9.494 \cdot 10^{-3} \ \mu g \cdot l^{-1}$$

Appendix D Default exposure estimation of PEClocalwater (stage of life cycle: formulation of aqueous polymer dispersions, generic)

Default Exposure Estimation of $PEClocal_{water}$ -

status: TGD, table A and B, IC: 11, UC: 43

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

$d := 86400 s$

$a := 365 \cdot d$

chemical : 2 - (Ethylhexyl) acrylate

stage of life cycle: formulation of aqueous polymer dispersions, generic

Total annual tonnage of chemical:	$TONNAGE := 42 \cdot tonne \cdot a^{-1}$
Product tonnage:	$PRODUCT := 210000 tonne \cdot a^{-1}$
Release factor (A-table:2.1):	$f_{emission} := 0.3 \cdot \%$
Fraction of main source (B-table:2.3/2.9):	$Fmainsource := 0.4$
Waste water flow of wwtp:	$EFFLUENT_{stp} := 2000 \cdot m^3 \cdot d^{-1}$
Duration of emission (B-table:2.3):	$Temission := 300 \cdot d \cdot a^{-1}$
Fraction of emission directed to water: (SimpleTreat; k:1h^{-1}; logPow: 3,9; logH:2,36)	$Fstp_{water} := 7 \cdot \%$
Dilution factor (TGD):	$DILUTION := 10$
Factor (1+Kp * SUSPwater):	$FACTOR := 1.0014$
Regional concentration in surface water	$PECregional_{water} := 0.0058 \mu g \cdot l^{-1}$

Emission per day:

$$Elocal_{water} := \frac{TONNAGE \cdot Fmainsource \cdot f_{emission}}{Temission} \qquad Elocal_{water} = 0.17 \, kg \cdot d^{-1}$$

Influent concentration:

$$Clocal_{inf} := \frac{Elocal_{water}}{EFFLUENT_{stp}} \qquad Clocal_{inf} = 84 \, \mu g \cdot l^{-1}$$

Effluent concentration:

$$Clocal_{eff} := Clocal_{inf} \cdot Fstp_{water} \qquad Clocal_{eff} = 5.88 \, \mu g \cdot l^{-1}$$

Concentration in surface water:

$$Clocal_{water} := \frac{Clocal_{eff}}{FACTOR \cdot DILUTION} \qquad Clocal_{water} = 0.59 \, \mu g \cdot l^{-1}$$

PEClocal$_{water}$

$$PEClocal_{water} := Clocal_{water} + PECregional_{water} \qquad PEClocal_{water} = 0.593 \cdot \mu g \cdot l^{-1}$$

Release to wwtp

$$RELEASE_{wwtp} := TONNAGE \cdot f_{emission} \qquad RELEASE_{wwtp} = 0.126 \cdot tonne \cdot a^{-1}$$

Release to surface water

$$RELEASE_{sw} := TONNAGE \cdot f_{emission} \cdot Fstp_{water} \qquad RELEASE_{sw} = 8.82 \cdot 10^{-3} \cdot tonne \cdot a^{-1}$$

Annual average local PEC in surface water:

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

$$Clocal_{water\_ann} = 0.483 \cdot \mu g \cdot l^{-1}$$

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

$$PEClocal_{water\_ann} = 0.488 \cdot \mu g \cdot l^{-1}$$

Appendix E Default exposure estimation of PEClocalwater (stage of life cycle: use (processing) of water based paints and adhesives)

Default Exposure Estimation of $PEClocal_{water}$ -

status: TGD, table A and B, IC: 14, UC: 55/0

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

$d := 86400 \cdot s$

$a := 365 \cdot d$

chemical : 2 - (Ethylhexyl) acrylate

stage of life cycle: use (processing) of water based paints and adhesives

Total annual tonnage of chemical: $TONNAGE := 35.7 \cdot tonne \cdot a^{-1}$

Product tonnage: $PRODUCT := 178500\,tonne \cdot a^{-1}$

Release factor (A-table:3.15/4.5): $f_{emission} := 0.5 \cdot \%$

Fraction of main source (B-table:3.13/4.5): $Fmainsource := 0.05$

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

Duration of emission (B-table:3.13/4.5): $Temission := 300 \cdot d \cdot a^{-1}$

Fraction of emission directed to water: (SimpleTreat; k:1h^{-1}; logPow: 3,9; logH:2,36) $Fstp_{water} := 7 \cdot \%$

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

Factor (1+Kp * SUSPwater): $FACTOR := 1.0014$

Regional concentration in surface water $PECregional_{water} := 0.0058\,\mu g \cdot l^{-1}$

Emission per day:

$$Elocal_{water} := \frac{TONNAGE \cdot Fmainsource \cdot f_{emission}}{Temission} \qquad Elocal_{water} = 0.0298\,kg \cdot d^{-1}$$

Influent concentration:

$$Clocal_{inf} := \frac{Elocal_{water}}{EFFLUENT_{stp}} \qquad Clocal_{inf} = 14.88\,\mu g \cdot l^{-1}$$

Effluent concentration:

$$Clocal_{eff} := Clocal_{inf} \cdot Fstp_{water} \qquad Clocal_{eff} = 1.04\,\mu g \cdot l^{-1}$$

Concentration in surface water:

$$Clocal_{water} := \frac{Clocal_{eff}}{FACTOR \cdot DILUTION} \qquad Clocal_{water} = 0.1\,\mu g \cdot l^{-1}$$

PEClocal$_{water}$

$PEClocal_{water} := Clocal_{water} + PECregional_{water}$ $PEClocal_{water} = 0.11 \cdot \mu g \cdot l^{-1}$

Total release for the regional model

$RELEASE := TONNAGE \cdot f_{emission} \cdot Fstp_{water}$ $RELEASE = 0.012 \cdot tonne \cdot a^{-1}$

Annual average local PEC in surface water:

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

$Clocal_{water\_ann} = 0.085 \cdot \mu g \cdot l^{-1}$

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

$PEClocal_{water\_ann} = 0.091 \cdot \mu g \cdot l^{-1}$

Appendix F Calculation of PEClocal for aquatice compartment durino paper recycling

Calculation of PEC_{local} for aquatic compartment during paper recycling
status: TGD, ESD, IC-12

chemical : 2- (Ethylhexyl) acrylate

$d := 86400 \cdot s$
$a := 365 \cdot d^{-1}$
$\mu g := 10^{-9} \cdot kg$

Total annual consumption of substance for paper:	$Ws := 4200 \cdot kg \cdot a^{-1}$
rate of paper recycling:	$RR := 50 \cdot \%$
Deinking rate (tab. 7,8):	$DR := 90 \cdot \%$
Rate by not adsorption (tab.7):	$NA := 20 \cdot \%$
Number of working days per year:	$Nd := 250 \cdot d \cdot a^{-1}$
Number of recycling sites:	$Ns := 10$
Waste water flow of wwtp:	$EFFLUENT_{stp} := 2000 \cdot m^3 \cdot d^{-1}$
Fraction of emission directed to water: (Simple-Treat, k:1h^{-1}, logH:2,36; logPow:3,9)	$Fstp_{water} := 7 \cdot \%$
Factor (1+Kp*SUSPwater):	$FACTOR := 1.0014$
Dilution factor (TGD):	$DILUTION := 10$
Regional concentration in surface water	$PECregional_{water} := 0.0058 \cdot \mu g \cdot l^{-1}$

Emission per day:

$$Elocal_{water} := \frac{Ws \cdot RR \cdot DR \cdot NA}{Nd \cdot Ns} \qquad Elocal_{water} = 0.151 \cdot kg \cdot d^{-1}$$

Influent concentration:

$$Clocal_{inf} := \frac{Elocal_{water}}{EFFLUENT_{stp}} \qquad Clocal_{inf} = 75.6 \cdot \mu g \cdot l^{-1}$$

Effluent concentration:

$$Clocal_{eff} := Clocal_{inf} \cdot Fstp_{water} \qquad Clocal_{eff} = 5.292 \cdot \mu g \cdot l^{-1}$$

Concentration in surface water:

$$Clocal_{water} := \frac{Clocal_{eff}}{DILUTION \cdot FACTOR} \qquad Clocal_{water} = 0.53 \cdot \mu g \cdot l^{-1}$$

$PEClocal_{water}$

$$PEClocal_{water} := Clocal_{water} + PECregional_{water} \qquad PEClocal_{water} = 0.534 \cdot \mu g \cdot l^{-1}$$

Release to hydrosphere

$$Release_{sw} := Elocal_{water} \cdot Fstp_{water} \qquad Release_{sw} = 0.011 \cdot kg \cdot d^{-1}$$

Annual average local PEC in surface water

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

$$Clocal_{water\_ann} = 0.362 \cdot \mu g \cdot l^{-1}$$

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

$$PEClocal_{water\_ann} = 0.368 \cdot \mu g \cdot l^{-1}$$

Appendix G.1 Atmosphere (OPS-model) – Calculation of Clocalair and PEClocalair, site specific

Atmosphere (OPS-model)

$a := 365 \cdot Tag$ $d := 24 \cdot h$ $mg := 10^{-6} \cdot kg$

Calculation of Clocal $_{air}$ and PEC local $_{air}$, site-specific

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 main source

$Fmainsource := 1$

Duration of emission

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

Fraction of emission

$RELEASE := 0.18 \cdot tonne \cdot a^{-1}$

Local emission during emission episode to air

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

$Elocal_{air} = 0.6 \, kg \cdot d^{-1}$

Fraction of the emission to air from STP

$Fstp_{air} := 0.298$

Local emission rate to water during emission episode

$Elocal_{water} := 122 \cdot kg \cdot d^{-1}$

Local emission to air from STP during emission episode

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

$Estp_{air} = 36.356 \, kg \cdot d^{-1}$

Local concentration in air during emission episode

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

$Clocal_{air} = 0.0101 \, mg \cdot m^{-3}$

Annual average concentration in air, 100m from point source

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

$Clocal_{air\_ann} = 8.307 \cdot 10^{-3} \, mg \cdot m^{-3}$

Regional concentration in air

$PECregional_{air} := 7.88 \cdot 10^{-7} \cdot mg \cdot m^{-3}$

Annual average predicted environmental concentration in air

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

$PEClocal_{air\_ann} = 8.308 \cdot 10^{-3} \, mg \cdot m^{-3}$

Appendix G.2 Atmosphere (OPS – model) – Calculation of $Clocal_{air}$ and $PEClocal_{air}$

Atmosphere (OPS-model)

Calculation of Clocal $_{air}$ and PEC local $_{air}$
2 - (Ethylhexyl) acrylate

$a := 365 \cdot Tag \quad d := 24 \cdot h \quad mg := 10^{-6} \cdot kg$

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 main source

$Fmainsource := 1$

Duration of emission

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

Fraction of emission

$RELEASE := 10 \cdot tonne \cdot a^{-1}$

Local emission during emission episode to air

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

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

Fraction of the emission to air from STP

$Fstp_{air} := 0.298$

Local emission rate to water during emission episode

$Elocal_{water} := 0.33 \cdot kg \cdot d^{-1}$

Local emission to air from STP during emission episode

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

$Estp_{air} = 0.098 \cdot kg \cdot d^{-1}$

Local concentation in air during emission episode

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

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

Annual average concentration in air, 100m from point source

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

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

Regional concentration in air

$PECregional_{air} := 7.88 \cdot 10^{-7} \cdot mg \cdot m^{-3}$

Annual average predicted environmental concentration in air

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

$PEClocal_{air\_ann} = 7.617 \cdot 10^{-3} \cdot mg \cdot m^{-3}$

Calculation of the deposition rate

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

Fraction of the chemical bound to aerosol

$$Fass_{aer} := 8.333 \cdot 10^{-6}$$

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

logH<-2 5E-4 mg * m $^{-2}$ * d^{-1}

-2<logH<2 4E-4 mg * m^{-2} * d^{-1}

logH>2 3E-4 mg * m^{-2} * d^{-1}

$$DEPstd_{gas} := 3 \cdot 10^{-4} \cdot mg \cdot m^{-2} \cdot d^{-1} \cdot kg^{-1} \cdot d$$

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

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

$$DEPtotal_{ann} = 8.246 \cdot 10^{-3} \, mg \cdot m^{-2} \cdot d^{-1}$$

Appendix G.3 Atmosphere (OPS-model)

Atmosphere (OPS-model)

$a := 365 \cdot Tag \quad d := 24 \cdot h \quad mg := 10^{-6} \cdot kg$

Calculation of Clocal $_{air}$ and PEC local $_{air}$
2 - (Ethylhexyl) acrylate
stage of life cycle: formulation of aqueous polymer dispersions
generic, IC:11, UC: 43

Total annual tonnage of chemical: $TONNAGE := 42 \cdot tonne \cdot a^{-1}$

Product tonnage: $PRODUCT := 210000 \cdot tonne \cdot a^{-1}$

Release factor (A-table:2.1, MC3 as default): $f_{emission} := 0.005$

Fraction of main source (B-table:2.3/2.9): $Fmainsource := 0.4$

Duration of emission (B-table:2.3): $Temission := 300 \cdot d \cdot a^{-1}$

concentration in air at source strength of 1kg/d: $Cstd_{air} := 2.78 \cdot 10^{-4} \cdot mg \cdot m^{-3} \cdot kg^{-1} \cdot d$

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

$RELEASE = 0.21 \ tonne \cdot a^{-1}$

Local emission during emission episode to air: $Elocal_{air} := Fmainsource \cdot \frac{RELEASE}{Temission}$

$Elocal_{air} = 0.28 \ kg \cdot d^{-1}$

Fraction of the emission to air from STP: $Fstp_{air} := 0.298$

Local emission rate to water during emission episode: $Elocal_{water} := 1.12 \cdot kg \cdot d^{-1}$

Local emission to air from STP during emission episode: $Estp_{air} := Fstp_{air} \cdot Elocal_{water}$

$Estp_{air} = 0.334 \ kg \cdot d^{-1}$

Local concentation in air during emission episode

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

$Clocal_{air} = 9.279 \cdot 10^{-5} \ mg \cdot m^{-3}$

Annual average concentration in air, 100m from point source

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

$$Clocal_{air\_ann} = 7.626 \cdot 10^{-5} \ mg \cdot m^{-3}$$

Regional concentration in air

$$PECregional_{air} := 7.88 \cdot 10^{-7} \cdot mg \cdot m^{-3}$$

Annual average predicted environmental concentration in air

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

$$PEClocal_{air\_ann} = 7.705 \cdot 10^{-5} \ mg \cdot m^{-3}$$

Calculation of the deposition rate

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

Fraction of the chemical bound to aerosol

$$Fass_{aer} := 8.333 \cdot 10^{-6}$$

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

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

$$DEPstd_{gas} := 3 \cdot 10^{-4} \cdot mg \cdot m^{-2} \cdot d^{-1} \cdot kg^{-1} \cdot d$$

Total deposition flux during emission episode

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

$$DEPtotal = 1.842 \cdot 10^{-4} \ mg \cdot m^{-2} \cdot d^{-1}$$

Annual average total depostion flux

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

$$DEPtotal_{ann} = 1.514 \cdot 10^{-4} \ mg \cdot m^{-2} \cdot d^{-1}$$

Appendix H.1 Exposure of Soil. Input: 2-(Ethylhexyl acrylate): site-specific, production and process

Exposure of Soil

$d := 86400 \cdot s$

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

$a := 365 \cdot d$

$i := 1 .. 3$

Input: 2 - (Ethylhexyl) acrylate): site-specific, prod+proc

annual average total deposition flux: $DEPtotal_{ann} := 0.009115 mg \cdot m^{-2} \cdot d^{-1}$

soil-water partitioning coefficient: $K_{soil\_water} := 27.64$

concentration in dry sewage sludge: $C_{sludge} := 0 \cdot mg \cdot kg^{-1}$

air-water partitioning coefficient: $K_{air\_water} := 0.0971$

rate constant for for removal from top soil: $kbio_{soil} := 0.023 \cdot d^{-1}$

PECregional: $PECregional_{natural\_soil} := 8.12 \cdot 10^{-8} \cdot mg \cdot kg^{-1}$

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:

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

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

	$\frac{Clocal_{soil_i}}{ppm}$
$Clocal_{soil}$ =	$8.513 \cdot 10^{-4}$
$Clocal_{agr.soil}$ =	$8.513 \cdot 10^{-4}$
$Clocal_{grassland}$ =	$1.341 \cdot 10^{-3}$

	$\frac{PEClocal_{soil_i}}{ppm}$
$PEClocal_{soil}$ =	$8.513 \cdot 10^{-4}$
$PEClocal_{agr.soil}$ =	$8.513 \cdot 10^{-4}$
$PEClocal_{grassland}$ =	$1.341 \cdot 10^{-3}$

Indicating persistency of the substance in soil

initial concentration after 10 years:

$\frac{C_{soil\_10_i}}{ppm}$
$8.513 \cdot 10^{-4}$
$8.513 \cdot 10^{-4}$
$1.341 \cdot 10^{-3}$

initial concentration in steady-state situation:

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

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

$\frac{C_{soil\_ss_i}}{ppm}$
$8.513 \cdot 10^{-4}$
$8.513 \cdot 10^{-4}$
$1.341 \cdot 10^{-3}$

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

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

	$Clocal_{soil\_porew_i}$ / $mg \cdot l^{-1}$
$Clocal_{soil\_porew}$ =	$5.236 \cdot 10^{-5}$
$Clocal_{agr.soil\_porew}$ =	$5.236 \cdot 10^{-5}$
$Clocal_{grassland\_porew}$ =	$8.247 \cdot 10^{-5}$

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

	$PEClocal_{soil\_porew_i}$ / $mg \cdot l^{-1}$
$PEClocal_{soil\_porew}$ =	$5.236 \cdot 10^{-5}$
$PEClocal_{agr.soil\_porew}$ =	$5.236 \cdot 10^{-5}$
$PEClocal_{grassland\_porew}$ =	$8.248 \cdot 10^{-5}$

concentration in ground water

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

Appendix H.2 Exposure of soil. Input: 2-(Ethylhexyl acrylate): process – worst case

Exposure of Soil

$d := 86400 \cdot s$

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

$a := 365 \cdot d$

$i := 1 .. 3$

Input: 2 - (Ethylhexyl) acrylate): proc - worst case

annual average total deposition flux: $DEPtotal_{ann} := 0.008246 mg \cdot m^{-2} \cdot d^{-1}$

soil-water partitioning coefficient: $K_{soil\_water} := 27.64$

concentration in dry sewage sludge: $C_{sludge} := 0 \cdot mg \cdot kg^{-1}$

air-water partitioning coefficient: $K_{air\_water} := 0.0971$

rate constant for for removal from top soil: $kbio_{soil} := 0.023 \cdot d^{-1}$

PECregional: $PECregional_{natural\_soil} := 8.12 \cdot 10^{-8} \cdot mg \cdot kg^{-1}$

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:

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

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

	$\frac{Clocal_{soil_i}}{ppm}$
$Clocal_{soil}$ =	$7.701 \cdot 10^{-4}$
$Clocal_{agr.soil}$ =	$7.701 \cdot 10^{-4}$
$Clocal_{grassland}$ =	$1.213 \cdot 10^{-3}$

	$\frac{PEClocal_{soil_i}}{ppm}$
$PEClocal_{soil}$ =	$7.702 \cdot 10^{-4}$
$PEClocal_{agr.soil}$ =	$7.702 \cdot 10^{-4}$
$PEClocal_{grassland}$ =	$1.213 \cdot 10^{-3}$

Indicating persistency of the substance in soil

initial concentration after 10 years:

$\frac{C_{soil\_10_i}}{ppm}$
$7.701 \cdot 10^{-4}$
$7.701 \cdot 10^{-4}$
$1.213 \cdot 10^{-3}$

initial concentration in steady-state situation:

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

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

$\frac{C_{soil\_ss_i}}{ppm}$
$7.701 \cdot 10^{-4}$
$7.701 \cdot 10^{-4}$
$1.213 \cdot 10^{-3}$

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}
$Clocal_{soil\_porew}$ =	$4.737 \cdot 10^{-5}$
$Clocal_{agr.soil\_porew}$ =	$4.737 \cdot 10^{-5}$
$Clocal_{grassland\_porew}$ =	$7.461 \cdot 10^{-5}$

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

	$PEClocal_{soil\_porew_i}$ / mg·l^{-1}
$PEClocal_{soil\_porew}$ =	$4.737 \cdot 10^{-5}$
$PEClocal_{agr.soil\_porew}$ =	$4.737 \cdot 10^{-5}$
$PEClocal_{grassland\_porew}$ =	$7.461 \cdot 10^{-5}$

concentration in ground water

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

Appendix H.3 Exposure of soil. Input: 2-(Ethylhexyl acrylate): formulation aqueous polymer dispersions

Exposure of Soil

$d := 86400 s$

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

$i := 1 .. 3$

Input: 2 - (Ethylhexyl) acrylate): formualtion aqueous polymer dispersions

annual average total deposition flux: $DEPtotal_{ann} := 0.0001514 mg \cdot m^{-2} \cdot d^{-1}$

soil-water partitioning coefficient: $K_{soil\_water} := 27.64$

concentration in dry sewage sludge: $C_{sludge} := 0 \cdot mg \cdot kg^{-1}$

air-water partitioning coefficient: $K_{air\_water} := 0.0971$

rate constant for for removal from top soil: $kbio_{soil} := 0.023 \cdot d^{-1}$

PECregional: $PECregional_{natural\_soil} := 8.12 \cdot 10^{-8} \cdot mg \cdot kg^{-1}$

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:

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

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

	$\frac{Clocal_{soil_i}}{ppm}$
$Clocal_{soil}$ =	$1.414 \cdot 10^{-5}$
$Clocal_{agr.soil}$ =	$1.414 \cdot 10^{-5}$
$Clocal_{grassland}$ =	$2.227 \cdot 10^{-5}$

	$\frac{PEClocal_{soil_i}}{ppm}$
$PEClocal_{soil}$ =	$1.422 \cdot 10^{-5}$
$PEClocal_{agr.soil}$ =	$1.422 \cdot 10^{-5}$
$PEClocal_{grassland}$ =	$2.235 \cdot 10^{-5}$

Indicating persistency of the substance in soil

initial concentration after 10 years:

$\frac{C_{soil\_10_i}}{ppm}$
$1.414 \cdot 10^{-5}$
$1.414 \cdot 10^{-5}$
$2.227 \cdot 10^{-5}$

initial concentration in steady-state situation:

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

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

$\frac{C_{soil\_ss_i}}{ppm}$
$1.414 \cdot 10^{-5}$
$1.414 \cdot 10^{-5}$
$2.227 \cdot 10^{-5}$

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}
$Clocal_{soil\_porew}$ =	$8.696 \cdot 10^{-7}$
$Clocal_{agr.soil\_porew}$ =	$8.696 \cdot 10^{-7}$
$Clocal_{grassland\_porew}$ =	$1.37 \cdot 10^{-6}$

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

	$PEClocal_{soil\_porew_i}$ / mg·l^{-1}
$PEClocal_{soil\_porew}$ =	$8.746 \cdot 10^{-7}$
$PEClocal_{agr.soil\_porew}$ =	$8.746 \cdot 10^{-7}$
$PEClocal_{grassland\_porew}$ =	$1.375 \cdot 10^{-6}$

concentration in ground water

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

Appendix I SimpleBox2.0a – Berechnung regionaler & kontinentaler PEC's

-Anpassung an TGD (1996) / EUSES 1.00: Michael Feibicke (06/98)

Table I.1 INPUT – 2EHA

Parameter names acc. SimpleBox20	Unit	Input	Parameter names according Euses
Physicochemical Properties			
Compound name	[-]	2-EHA	Substance
Mol weight	[g.mol^{-1}]	184	Molecular weight
Melting point	[° C]	-90	Melting point
Vapor pressure (25)	[Pa]	12	Vapour pressure at 25°C
Log kow	[log10]	3.9	Octanol-water partition coefficient
Solubility (25)	[mg.l^{-1}]	9.6	Water solubility
Distribution - Partition coefficients			
- Solids water partitioning (derived from K_{oc})			
Kp(soil)	[l.kg$_d^{-1}$]	18.282	Solids-water partitioning in soil
Kp(sed)	[l.kg$_d^{-1}$]	91.411	Solids-water partitioning in sediment
Kp(susp)	[l.kg$_d^{-1}$]	91.411	Solids-water partitioning in sudpended matter
- Biota-water			
BCF(fish)	[l.kg$_w^{-1}$]	412	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}]	8.64E-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]	[-]	2.98E-01	Fraction of emission directed to air (STPcont)
FR(effstp) [C]	[-]	7.00E-02	Fraction of emission directed to water (STPcont)
FR(sludgestp) [C]	[-]	7.50E-02	Fraction of emission directed to sludge (STPcont)

Table I.1 continued overleaf

Table I.1 continued INPUT – 2EHA

Parameter names acc. SimpleBox20	Unit	Input	Parameter names according Euses
- Regional			
FR(volatstp) [R]	[-]	2.98E-01	Fraction of emission directed to air (STPreg)
FR(effstp) [R]	[-]	7.00E-02	Fraction of emission directed to water (STPreg)
FR(sludgestp) [R]	[-]	7.50E-02	Fraction of emission directed to sludge (STPreg)
Release estimation			
- Continental			
Edirect(air) [C]	[t.y^{-1}]	51.78	Total continental emission to air
STPload [C]	[t.y^{-1}]	13.71	Total continental emission to wastewater
Edirect(water1) [C]	[t.y^{-1}]	5.67	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}]	10.6	Total regional emission to air
STPload [R]	[t.y^{-1}]	38.17	Total regional emission to wastewater
Edirect(water1) [R]	[t.y^{-1}]	0.63	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

Table I.2 OUTPUT – 2-EHA

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	[-]	2-EHA	Substance
Output			
- Continental			
PECsurfacewater (total)	[mg.l^{-1}]	1.38E-07	Continental PEC in surface water (total)
PECsurfacewater (dissolved)	[mg.l^{-1}]	1.38E-07	Continental PEC in surface water (dissolved)
PECair	[mg.m^{-3}]	5.91E-08	Continental PEC in air (total)
PECagr.soil	[mg.kgwwt^{-1}]	2.79E-07	Continental PEC in agricultural soil (total)
PECporewater agr.soil	[mg.l^{-1}]	1.72E-08	Continental PEC in pore water of agricultural soils
PECnat.soil	[mg.kgwwt^{-1}]	6.09E-09	Continental PEC in natural soil (total)

Table I.2 continued overleaf

Table I.2 continued OUTPUT – 2-EHA

Parameter names acc. SimpleBox20	Unit	Output	Parameter names according Euses
Output			
- Continental			
PECind.soil	[mg.kgwwt^{-1}]	6.09E-09	Continental PEC in industrial soil (total)
PECsediment	[mg.kgwwt^{-1}]	3.51E-06	Continental PEC in sediment (total)
- Regional			
PECsurfacewater (total)	[mg.l-1]	5.80E-06	Regional PEC in surface water (total)
PECsurfacewater (dissolved)	[mg.l-1]	5.79E-06	Regional PEC in surface water (dissolved)
PECair	[mg.m-3]	7.88E-07	Regional PEC in air (total)
PECagr.soil	[mg.kgwwt-1]	6.77E-05	Regional PEC in agricultural soil (total)
PECporewater agr.soil	[mg.l-1]	4.16E-06	Regional PEC in pore water of agricultural soils
PECnat.soil	[mg.kgwwt-1]	8.12E-08	Regional PEC in natural soil (total)
PECind.soil	[mg.kgwwt-1]	8.12E-08	Regional PEC in industrial soil (total)
PECsediment	[mg.kgwwt-1]	1.33E-04	Regional PEC in sediment (total)

Appendix J.1 Chemical properties and environmental concentrations (2-ethylhexyl acrylate :site specifice, production and process)

Name: 2 - (Ethylhexyl) acrylate: site-specific, prod + proc

CAS - No.: 103 - 11 - 7

Input

chemical properties

octanol-water partitioning coefficient [-]	$\log K_{OW} := 3.9$ $K_{OW} := 10^{\log K_{OW}}$
Henry - partitioning coefficient [Pa*m^3*mol^{-1}]	$HENRY := 230 \cdot Pa \cdot m^3 \cdot mol^{-1}$
air-water partitioning coefficient [-]	$K_{air\_water} := 0.0971$
fraction of the chemical associated with aerosol particles [-]	$F_{ass\_aer} := 8.333 \cdot 10^{-6}$
half-life for biodegration in surface water [d]	$DT_{50\_bio\_water} := 14.748 \cdot d$

environmental concentrations

annual average local PEC in surface water(dissolved) [mg_{chem} * l_{water}^{-1}]	$PEClocal_{water\_ann} := 0.000116 mg \cdot l^{-1}$
annual average local PEC in air (total) [mg_{chem} * m_{air}^{-3}]	$PEClocal_{air\_ann} := 8.308 \cdot 10^{-3} \cdot mg \cdot m^{-3}$
local PEC in grassland (total), averaged over 180 days [mg_{chem} * kg_{soil}^{-1}]	$PEClocal_{grassland} := 1.341 \cdot 10^{-3} \cdot mg \cdot kg^{-1}$
local PEC in porewater of agriculture soil [mg_{chem} * $l_{porewater}^{-1}$]	$PEClocal_{agr\_soil\_porew} := 5.236 \cdot 10^{-5} \cdot mg \cdot l^{-1}$
local PEC in porewater of grassland [mg_{chem} * $l_{porewater}^{-1}$]	$PEClocal_{grassland\_porew} := 8.248 \cdot 10^{-5} \cdot mg \cdot l^{-1}$
local PEC in groundwater under agriculture soil [mg_{chem} * l_{water}^{-1}]	$PEClocal_{grw} := 5.236 \cdot 10^{-5} \cdot mg \cdot l^{-1}$
regional PEC in surface water (dissolved) [mg_{chem} * l_{water}^{-1}]	$PECregional_{water} := 5.80 \cdot 10^{-6} \cdot mg \cdot l^{-1}$
regional PEC in air (total) [mg_{chem} * m_{air}^{-3}]	$PECregional_{air} := 7.88 \cdot 10^{-7} \cdot mg \cdot m^{-3}$
regional PEC in agriculture soil (total) [mg_{chem}*kg_{soil}^{-1}	$PECregional_{agr\_soil} := 6.77 \cdot 10^{-5} \cdot mg \cdot kg^{-1}$
regional PEC in porewater of agriculture soils [mg_{chem}*l_{water}^{-1}	$PECregional_{agr\_soil\_porew} := 4.16 \cdot 10^{-6} \cdot mg \cdot l^{-1}$

Results of calculation

$DOSE_{tot_{local}} = 1.99116 \cdot 10^{-3} \circ \frac{mg}{kg_{bw} \cdot d}$

$DOSE_{tot_{regional}} = 5.899203 \cdot 10^{-6} \circ \frac{mg}{kg_{bw} \cdot d}$

$RDOSE_{drw_{local}} = 0.083225 \circ \%$

$RDOSE_{drw_{regional}} = 2.0148 \circ \%$

$RDOSE_{air_{local}} = 89.409475 \circ \%$

$RDOSE_{air_{regional}} = 2.862372 \circ \%$

$RDOSE_{stem_{local}} = 5.406237 \circ \%$

$RDOSE_{stem_{regional}} = 0.173339 \circ \%$

$RDOSE_{root_{local}} = 1.058184 \circ \%$

$RDOSE_{root_{regional}} = 28.377083 \circ \%$

$RDOSE_{meat_{local}} = 0.062128 \circ \%$

$RDOSE_{meat_{regional}} = 5.76514 \cdot 10^{-3} \circ \%$

$RDOSE_{milk_{local}} = 0.036617 \circ \%$

$RDOSE_{milk_{regional}} = 3.397866 \cdot 10^{-3} \circ \%$

$RDOSE_{fish_{local}} = 3.944134 \circ \%$

$RDOSE_{fish_{regional}} = 66.563243 \circ \%$

Appendix J.2 Chemical properties and environmental concentrations (2-ethylhexyl acrylate : formulation aqueous polymer dispersions)

Name: 2 - (Ethylhexyl) acrylate:
formulation aqueous polymer dispersions

CAS - No.: 103 - 11 - 7

Input

chemical properties

octanol-water partitioning coefficient [-]	$logK_{OW} := 3.9$ $K_{OW} := 10^{logK_{OW}}$
Henry - partitioning coefficient [Pa*m^3*mol^{-1}]	$HENRY := 230 \cdot Pa \cdot m^3 \cdot mol^{-1}$
air-water partitioning coefficient [-]	$K_{air\_water} := 0.0971$
fraction of the chemical associated with aerosol particles [-]	$F_{ass\_aer} := 8.333 \cdot 10^{-6}$
half-life for biodegration in surface water [d]	$DT_{50\_bio\_water} := 14.748 \cdot d$

environmental concentrations

annual average local PEC in surface water (dissolved) [$mg_{chem} * l_{water}^{-1}$]	$PEClocal_{water\_ann} := 0.000488 \cdot mg \cdot l^{-1}$
annual average local PEC in air (total) [$mg_{chem} * m_{air}^{-3}$]	$PEClocal_{air\_ann} := 7.705 \cdot 10^{-5} \cdot mg \cdot m^{-3}$
local PEC in grassland (total), averaged over 180 days [$mg_{chem} * kg_{soil}^{-1}$]	$PEClocal_{grassland} := 2.235 \cdot 10^{-5} \cdot mg \cdot kg^{-1}$
local PEC in porewater of agriculture soil [$mg_{chem} * l_{porewater}^{-1}$]	$PEClocal_{agr\_soil\_porew} := 8.746 \cdot 10^{-7} \cdot mg \cdot l^{-1}$
local PEC in porewater of grassland [$mg_{chem} * l_{porewater}^{-1}$]	$PEClocal_{grassland\_porew} := 1.375 \cdot 10^{-6} \cdot mg \cdot l^{-1}$
local PEC in groundwater under agriculture soil [$mg_{chem} * l_{water}^{-1}$]	$PEClocal_{grw} := 8.746 \cdot 10^{-7} \cdot mg \cdot l^{-1}$
regional PEC in surface water (dissolved) [$mg_{chem} * l_{water}^{-1}$]	$PECregional_{water} := 5.80 \cdot 10^{-6} \cdot mg \cdot l^{-1}$
regional PEC in air (total) [$mg_{chem} * m_{air}^{-3}$]	$PECregional_{air} := 7.88 \cdot 10^{-7} \cdot mg \cdot m^{-3}$
regional PEC in agriculture soil (total) [$mg_{chem} * kg_{soil}^{-1}$]	$PECregional_{agr\_soil} := 6.77 \cdot 10^{-5} \cdot mg \cdot kg^{-1}$
regional PEC in porewater of agriculture soils [$mg_{chem} * l_{water}^{-1}$]	$PECregional_{agr\_soil\_porew} := 4.16 \cdot 10^{-6} \cdot mg \cdot l^{-1}$

Results of calculation

$DOSE_{tot_{local}} = 3.552534 \cdot 10^{-4} \circ \frac{mg}{kg_{bw} \cdot d}$

$DOSE_{tot_{regional}} = 5.899203 \cdot 10^{-6} \circ \frac{mg}{kg_{bw} \cdot d}$

$RDOSE_{drw_{local}} = 1.962382 \circ \%$

$RDOSE_{drw_{regional}} = 2.0148 \circ \%$

$RDOSE_{air_{local}} = 4.647588 \circ \%$

$RDOSE_{air_{regional}} = 2.862372 \circ \%$

$RDOSE_{stem_{local}} = 0.281022 \circ \%$

$RDOSE_{stem_{regional}} = 0.173339 \circ \%$

$RDOSE_{root_{local}} = 0.099069 \circ \%$

$RDOSE_{root_{regional}} = 28.377083 \circ \%$

$RDOSE_{meat_{local}} = 6.464467 \cdot 10^{-3} \circ \%$

$RDOSE_{meat_{regional}} = 5.76514 \cdot 10^{-3} \circ \%$

$RDOSE_{milk_{local}} = 3.810036 \cdot 10^{-3} \circ \%$

$RDOSE_{milk_{regional}} = 3.397866 \cdot 10^{-3} \circ \%$

$RDOSE_{fish_{local}} = 92.999665 \circ \%$

$RDOSE_{fish_{regional}} = 66.563243 \circ \%$

European Commission

EUR 21641 EN **European Union Risk Assessment Report**
2-ethylhexyl acrylate, Volume 56

Editors: S.J. Munn, R. Allanou, K. Aschberger, F. Berthault, O. Cosgrove, M. Luotamo, S. Pakalin, A. Paya-Perez, G. Pellegrini, B. Schwarz-Schulz, S. Vegro.

Luxembourg: Office for Official Publications of the European Communities

2005 – VIII pp., 140 pp. – 17.0 x 24.0 cm

Environment and quality of life series

The report provides the comprehensive risk assessment of human health part of the substance 2-ethylhexyl acrylate. 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 human health risk assessment for 2-ethylhexyl acrylate concludes that there is concern for workers. There is at present no concern for consumers and humans exposed via the environment. The environmental risk assessment for 2-ethylhexyl acrylate concludes that there is at present no concern for atmosphere, aquatic ecosystem, terrestrial ecosystem and for micro-organisms in the sewage treatment plant.

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.

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European Union Risk Assessment Report

2-ethylhexyl acrylate

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