European Chemicals Bureau

Institute for Health and Consumer Protection

European Chemicals Bureau

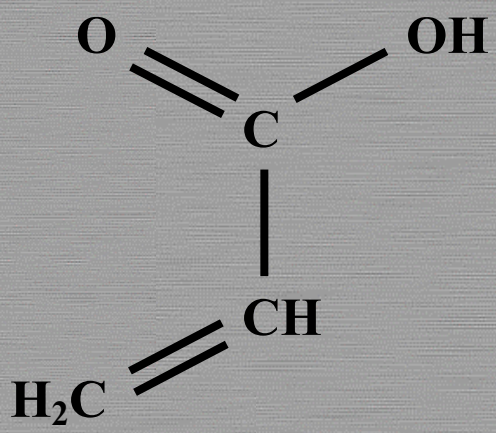
Existing Substances

European Union Risk Assessment Report

CAS No: 79-10-7

EINECS No: 201-177-9

acrylic acid



1st Priority List

Volume: 28

EUROPEAN COMMISSION
JOINT RESEARCH CENTRE

EUR 19836 EN

CAS: 79-10-7
EC: 201-177-9

PL-1
28

European Union Risk Assessment Report

ACRYLIC ACID

CAS No: 79-10-7

EINECS No: 201-177-9

RISK ASSESSMENT

Cataloguing data can be found at the end of this publication

Luxembourg: Office for Official Publications of the European Communities, 2002
ISBN 92-894-1272-0

Printed in Italy

ACRYLIC ACID

CAS No: 79-10-7

EINECS No: 201-177-9

RISK ASSESSMENT

Final Report, 2002

Germany

The risk assessment of acrylic acid (AA) has been prepared by Germany on behalf of the European Union.

Contact point:

Bundesanstalt für Arbeitsschutz und Arbeitsmedizin
Anmeldestelle Chemikaliengesetz (BAuA)
(Federal Institute for Occupational Safety and Health Notification Unit)
Friedrich-Henkel-Weg 1-25
44149 Dortmund
Germany

Fax: +49 (231) 9071-679
e-mail: chemg@baua.bund.de

Date of Last Literature Search:	**1995**
Review of report by MS Technical Experts finalised:	**2001**
Final report:	**2002**

(The last full literature survey was carried out in 1995 - targeted searches (for example on grouting and PBPK model) were carried out subsequently, and information found through scanning certain sources has also been included).

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 Toxicity, Ecotoxicity and the Environment (CSTEE) 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.

Barry Mc Sweeney
Director-General
DG Joint Research Centre

Catherine Day
Director-General
DG Environment

---

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

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

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

0 OVERALL RESULTS OF THE RISK ASSESSMENT

CAS-No.: 79-10-7
EINECS-No.: 201-177-9
IUPAC name: 2-propenoic acid
Synonym: acrylic acid

Environment

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

Acrylic acid (AA) presents, based on the present data, a risk to the environment around point sources. A potential risk to municipal wastewater treatment plants is identified for the downstream use scenarios of super absorber polymers (SAP) production (based on default calculation and highest site-specific PEC_{wwtp}) and wet polymerisation (based on default calculation and known sites L, Q).

Since the $PNEC_{microorganisms}$ is derived from single species tests with ciliated protozoa, there is a need for further data reflecting the integrity of the native ciliate population in sewage sludge as a whole. However, since risk reduction measures are necessary to remove concern for surface water (see below), these measures will also cover the protection of municipal wastewater treatment plants, and additional testing is not required.

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

This conclusion applies to effects on sediment, atmosphere, soil, and secondary poisoning. Conclusion (ii) applies also to the aquatic compartment regarding all production sites, the processing scenario (dry polymerisation), and the relevant use scenarios (leather finishing, textile finishing, formulation of paints and application of water treatment agents).

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

Acrylic acid (AA) presents, based on the present data, a risk to the environment around point sources.

A potential risk to the local aquatic environment is identified from wet polymerisation processes including wet production of SAP (super absorber polymers) by downstream users of monomeric AA (based on default calculations and known sites N, O, Q).

Although an improvement of the data (i.e. effluent measurements and/or site specific data on flow rates) may in principle be possible, it is judged to be unlikely that sufficiently complete representative monitoring data from the downstream users can be obtained with reasonable expenditure of time and money. For certain known SAP production sites and wet polymerisation sites, regular effluent concentrations up to 100 mg/l AA and significantly more have been reported. These data indicate that high effluent concentrations cannot be excluded, even if certain types of process engineering are applied. On the other hand, application of wastewater reutilization / recycling systems is known to result in zero emissions to the hydrosphere at a

number of downstream user sites, processing about 50% of AA used externally for SAP production and about 12% of AA used externally in wet polymerisation processes. For sites applying this kind of technique, no further risk reduction measures are deemed necessary. Measures applied for limiting the risk to the local aquatic environment are presumed to be also protective for municipal wastewater treatment plants.

During the use of a grouting agent containing magnesium diacrylate high concentrations of AA are released via the drainage water. The exposure assessment was based on measured effluent concentrations at a tunnel construction site. A quantitative extrapolation to other construction sites seems difficult, but similar conditions might be anticipated. Measures appropriate to local circumstances should be applied.

Human health

Human health (toxicity)

Workers

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

This conclusion is reached because of:

- concerns for respiratory tract irritation and corrosivity as a consequence of single inhalation exposure arising from production and processing, production of adhesives containing the substance and use of adhesives containing the substance (industrial area and skilled trade),
- concerns for local effects as a consequence of repeated inhalation exposure arising from production and use of adhesives containing the substance,
- concerns for general systemic toxicity as a consequence of repeated inhalation exposure arising from production and use of adhesives containing the substance.

Consumers

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

Humans exposed via the environment

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

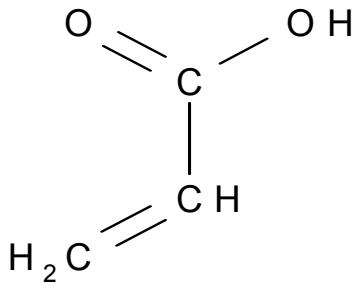
CONTENTS

TABLES

1 GENERAL SUBSTANCE INFORMATION

1.1 IDENTIFICATION OF THE SUBSTANCE

CAS-No.:	79-10-7
EINECS-No.:	201-177-9
IUPAC name:	2-Propenoic acid
Synonyms:	Acrylic acid
Molecular weight:	72.06 g/mol
Molecular formula:	$C_3H_4O_2$
Structural formula:	



1.2 PURITY/IMPURITIES, ADDITIVES

Purity:	99.7% w/w
Impurity:	< 0.05% w/w water
	< 0.05% w/w propionic acid
	< 0.2% w/w acetic acid
	< 0.5% w/w dimers of acrylic acid
Additives:	< 0.02% w/w hydroquinone monomethylether

For commercial acrylic acid (AA) 0.02% hydroquinone monomethylether (MEHQ) is added as stabilizer to avoid a spontaneous polymerisation (Bauer, 1991).

1.3 PHYSICO-CHEMICAL PROPERTIES

Table 1.1 Physico-chemical properties of acrylic acid

Properties	Value	Reference
Physical state	liquid at 20°C [1)]	
Melting point	14°C	Merck Index (1996)
Boiling point	141°C at 1,013 hPa	Merck Index (1996)
Density	1.0621 g/cm^3 at 20°C	Merck Index (1996)
Vapour pressure	3.8 hPa at 20°C - (dynamic method)	BASF AG (1994a)
Surface tension	59.6 mN/m c=1g/l - (ring method)	Hüls AG (1995)
Water solubility	miscible in all ratios	Merck Index (1996)
Dissociation constant	pK_a = 4.25	Weast (1989)
Partition coefficient	log P_{ow} 0.46 at 25°C - (shake flask method)	BASF AG (1988)
Flash point	48-55°C	CHEMSAFE
Auto flammability	395°C - DIN 51794	CHEMSAFE
Flammability	flammable	Test A.12 not conducted because of structural reasons
Explosive properties	not explosive	no test because of structural reasons
Oxidizing properties	no oxidizing properties	no test because of structural reasons

[1)] Under normal conditions acrylic acid is a clear, colourless liquid with a pungent smell

1.4 CLASSIFICATION

Classification and labelling according to the 28th ATP of Directive 67/548/EEC[4]:

Classification:	R10	Flammable
	Xn; R20/21/22	Harmful by inhalation, in contact with skin and if swallowed
	C; R35	Corrosive; Causes severe burns
	N; R50	Dangerous for the environment; very toxic to aquatic organisms
	Note D	
Labelling:	C; N	
	R: 10-20/21/22-35-50	
	S: (1/2-)26-36/37/39-45-61	

Concentration limits

C ≥ 25%:	C; R20/21/22-35
10% ≤ C < 25%:	C; R35
5% ≤ C < 10%:	C; R34
1% ≤ C < 5%:	Xi; R 36/37/38

[4] The classification of the substance is established by Commission Directive 2001/59/EC of 6 August 2001 adapting to technical progress for the 28th 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 225, 21.8.2001, p.1).

2 GENERAL INFORMATION ON EXPOSURE

2.1 PRODUCTION

2.1.1 Production processes

Acrylic acid (AA) is produced commercially by catalytic oxidation of propylene in two steps via acrolein or by a modification of the Reppe process from acetylene. In addition, AA can be prepared by hydrolysis of acrylonitrile (ECETOC, 1995).

2.1.2 Production capacity

As an important intermediate for polymer industry AA is produced in large quantities within the EU. Data from 6 producers and 3 importers are included in the IUCLID-database. The maximum cumulative production capacity from the indicated ranges amounts to 1,610,000 t/a. Taking into account the actual production capacities also provided by all companies a total production capacity of 810,000 t/a is calculated. This corresponds well to SRI's 1996 estimate of 815,000 tonnes annual production capacity (SRI, 1996). The highest actual production capacity at a single site is 330,000 t/a. At most, one large production site is located in a single region. A few isolated import figures have been communicated from different sites for different years, ranging up to 8,800 t per site from outside the EU. According to information from one producer 15,000 t/a are exported outside the EU.

Since only limited information on actual production and import volumes were provided by industry, a total amount of 830,000 t/a AA consumption (810,000 t/a production capacity plus 20,000 t/a total net import) is taken as a basis for the calculations.

The market trend appears quite dynamic during the last decade. Fox et al. (1990) estimated the Western European production of acrylic acid in 1987 at about 342,000 tonnes. For 1994, the worldwide consumption of AA is estimated at 2.4 million tonnes. The worldwide production capacity is expected to expand by another 500,000 t/a within 4 years (Rohe, 1995), that equals ca. 5% growth per year. In Germany a new production site is under construction, the operating company announcing for 1999 an initial production capacity of 80,000 t/a crude AA, and as commercial products 60,000 t/a glacial, i.e. purified AA and 60,000 t/a acrylate esters. An option for quick doubling of the capacity is reported (Europa Chemie 14/98, pp. 4-6: "Ausbau zum Vorzeigestandort").

2.2 USES

Acrylic acid serves as an industrial intermediate product, i.e. it is either processed directly into a polyacrylate or polymerised via the intermediate stage of an acrylate ester. Furthermore, acrylic acid is used as an ingredient and occurs as residual monomer in consumer products like adhesives, paints, binding agents and printing inks.

According to the producers about half of the 830,000 t/a crude AA is processed to purified (glacial) AA, which is further processed both on-site (captive use) and by external downstream users. The other half of crude AA is transformed into various acrylate esters at the production

sites. As for glacial AA, these acrylic esters serve as commercial products, which are further processed both on-site and by external downstream users.

The segmentation of the user industry has been recalculated independently by the producers yielding the new data given in **Table 2.1**. Calculations have been made on the basis of general accessible market information like TECNON:

Table 2.1 Percentage of AA consumption as acid and via esters

Product / use	% in this application
Dispersions	42
Superabsorber polymers	25
Textiles / leather	10
Acrylic fibres	4
Others	19

Among the polyacrylates (homo- and copolymers of AA), superabsorber polymers (SAP) are the most expansive use. In an assessment for 1993, the need for acrylic acid for the manufacture of superabsorbents and detergents in Western Europe was estimated to be 50,000 t/a each (Fox et al., 1990). It is estimated that its part in the global consumption will rise from 15 to 20% (Rohe, 1995). The actual EU figure of 25% represents an amount of about 210,000 t/a used for SAP production.

Further applications of the polyacrylates are co-builders in phosphate-free washing agents, and as flocculating agents for treatment of drinking water and wastewater.

Acrylic acid is also used as a pre-stage in the manufacture of coatings for printed circuit boards, so-called photoresists (Winkelmann et al., 1989; Reichert, 1993; Hoechst, 1994). AA may also be released as a decomposition product, when these photoresists are partially depolymerised by means of UV-light in the electroplating industry.

Recently, release of monomeric AA has been reported from the use of acrylate-based grouting agents at tunnel construction sites.

In the consumer field acrylic acid is used as an ingredient in adhesives. Moreover, acrylic acid occurs as residual monomer, e.g. SAP-containing products like sanitary towels, pantyliners and nappy pants. In addition, residual acrylic acid monomer may be contained in materials used for food packaging and in other commodities covered by specific legislation.

A summary of the content of AA in different products is presented in the Danish Product Register from June 1996 (no production of AA in Denmark), as shown in **Table 2.2**. The most frequent product types are paints, lacquers and varnishes, binding agents and printing inks.

In the Norwegian Product Register from 1994, 76 products containing a total quantity of 19 tonnes AA are registered. The most frequent product types are raw materials and additives (11 t/a), varnishes (4 t/a) and binders for paint, glue and castings (2 t/a).

According to the Swedish Product Register, 13 out of 56 products containing acrylic acid are used by consumers.

Table 2.2 AA-content - ranges in products and amount sold in Denmark (Danish Product Register, June 1996)

Content of AA in the product	Number of products	Quantity of AA sold in Denmark [t/a]
0-1%	700	384
1-10%	75	4
10-80%	27	50
80-100%		
not determined	22	

Table 2.3 gives an overview about the main, industrial and use categories according to the TGD (EC, 1996). Due to uncertainties about the amounts of AA assignable to these categories no quantitative information or percentages can be given.

Table 2.3 Main, industrial and use categories according to the Technical Guidance Document (EC, 1996)

Main category (MC)	Industrial category (IC)	Use category (UC)
isolated intermediate (1b, 1c)	chemical industry (3)	intermediate (33)
matrix-inclusion (2), non dispersive use (3)	polymers industry (11)	intermediate (33)
non dispersive use (3)	paints, lacquers and varnishes industry (14)	intermediate (33)
wide dispersive use (4)	personal / domestic (5), public domain (6)	adsorbents, absorbents (1) adhesives, binding agents (2) cleaning/washing additives (9) flotation agents (23) others (0)

Acrylate esters

Yielded via esterification from about 415,000 t/a AA (50% of the total EU consumption amount), acrylate esters are important monomers for the production of homo- and copolymers, which are used in the paint and adhesives industry, as well as for finishing of paper, textiles and leather; mainly as dispersions but also in solution. 99% of the acrylate esters are n-butyl, ethyl, methyl and 2-ethylhexyl acrylates, butylacrylate predominating quantitatively. Through polymerisation of the esters, transparent UV-resistant acrylic resins are produced, either thermoplastic or duroplastic by inclusion of functional groups like hydroxyl or carboxyl groups. These are employed as binding agents in paints and adhesives as well as coatings, thickeners and dispersion agents. With co-monomers containing vulcanising groups, acrylate rubbers are produced through polymerisation of acrylate esters, especially ethyl- and butyl acrylate (Rohe, 1995).

Fox et al. (1990) present the following consumption breakdown for acrylic esters in Western Europe in 1988, with a projection for 1993:

Table 2.4 Consumption breakdown for acrylic esters

Product	**Consumption [t]**	
	1988	**1993**
Emulsion polymers	206,000 (69%)	236,000 (70%)
Latex Surface Coatings	92,000	110,000
Paper Coatings	20,000	23,000
Adhesives and Sealants	35,000	44,000
Textiles and Non wovens	40,000	40,000
Leather	5,000	5,000
Polishes	4,000	4,000
Miscellaneous	10,000	10,000
Non emulsion Applications	92,000 (31%)	101,000 (30%)
Solvent-Based Surface Coatings	36,000	40,000
Acrylic Fibers	32,000	32,000
Impact Modifiers	6,000	7,000
Copolymers and Miscellaneous	18,000	22,000
Total	298,000	337,000

3 ENVIRONMENT

3.1 ENVIRONMENTAL EXPOSURE

3.1.1 Environmental releases

Releases of AA into the environment are to be expected during production and processing mainly via wastewater and lesser amounts via exhaust gases. Regarding the formulation step, relevant releases may possibly occur during formulation of polymer dispersions.

Further releases are to be expected through residual monomeric AA-contents in the final products. Residual monomeric concentrations determined by several companies in various polymer products ranged from 2.2 to 2,000 ppm. Per tonne AA used, a maximum residue of 4,000 g AA monomer was measured in newly manufactured products. For acrylate esters a residual monomeric AA content of 25 ppm has been reported. Due to the large amounts of AA being processed and related to the specific polymer end-uses the residual amounts of AA have to be taken into account for release estimation.

From the use of grouting agents containing magnesium diacrylate, releases of AA to the hydrosphere occur via drainage water.

Direct releases to agricultural or natural soil are not expected from the current use pattern.

3.1.2 Environmental fate

3.1.2.1 Degradation

Hydrolysis

In sterile water, acrylic acid has proved to be completely stable to hydrolysis at all tested pHs (3, 7, 11) over 28 days of investigation (Shah, 1990).

Photooxidation

When released into the atmosphere, acrylic acid reacts with photochemically produced hydroxyl radicals primarily by addition to the double bond, and with atmospheric ozone, resulting in estimated half-lifes of 39.6 hours and 6.5 days, respectively, assuming a hydroxyl radical concentration of 500,000 molecules/cm^3 and an ozone concentration of $7 \cdot 10^{11}$ molecules/cm^3. Because the UV absorption band extends to 320 nm (Sadtler, 1960), direct sunlight photolysis of acrylic acid is possible, but no experimental data are available.

Biodegradation

Based on the results of three guideline tests acrylic acid can be considered as readily biodegradable:

1. Douglas (1991) found that degradation achieved 81% of the theoretical oxygen demand (ThOD) after 28 days when acrylic acid was tested in a Closed-Bottle-Test (OECD 301 D).

2. In another investigation (Modified MITI Test (I); OECD 301 C) after 14 days the oxygen demand amounted to 68% of ThOD (CITI, 1992).

3. The result of a DOC-Die-Away-Test (OECD 301 A) showed 95% decrease of DOC within 9 days (Hüls, 1995a).

Furthermore, results from other tests are available which show high biodegradation rates. In an investigation conducted by Pahren and Bloodgood (1961) the biodegradation of acrylic acid measured as CO_2 evolution was in one case 70% after 19 days and in another case 71% after 42 days (percentage of theoretical CO_2 evolution, substance concentration 10 mg/l). In a Zahn-Wellens Test 100% of the substance (200 mg/l DOC) was degraded after 5 days (BASF AG, 1993).

A test on biodegradation in soil, performed according to a US EPA guideline is also available (Hawkins et al., 1992). From the test design, the test can be rated as a simulation test. The test conditions and main results are given in **Table 3.1**.

Table 3.1 Test conditions and main results of a test on AA biodegradation in soil

Test conditions	- sandy loam soil - ^{14}C-labelling of the substance - aerobic conditions - content of acrylic acid in soil: 100 mg/kg dw - duration: 28 days - incubation in darkness at 25°C
Main results	- 72.9% mineralisation within 3 days - 81.1% mineralisation after 28 days - DT_{50} for primary degradation < 1 day (estimated) - 10% of radioactivity non-extractable after 28 days

From the soil test it can be concluded that acrylic acid is readily biodegradable in this soil type. The applicability of the comparatively short $DT_{50} < 3$ d to other soil types and environmentally more relevant lower temperatures is not clear. However, the rate constant calculated according to the Technical Guidance Document (TGD) (Ch. 3, Table 6) $kbio_{soil} = 0.023\ d^{-1}$ corresponding to a DT50 of ca. 30 d appears to underestimate biodegradation. As a possibly reasonable approach, the rate constant for ready biodegradability in surface water $kbio_{soil} = 0.047\ d^{-1}$ corresponding to DT50 ≈ 15 d is used, also taking into account the low adsorption rates of AA (see below).

Acrylic acid is also susceptible to degradation by anaerobic microbes. In a screening study using 10% sludge from a secondary digester as an inoculum, acrylic acid was judged to be degradable with > 75% of theoretical methane being produced within 8 weeks of incubation (Shelton and Tiedje, 1984). The result does not reveal environmental conditions as the study was conducted at 35°C.

Results from other biodegradation simulation tests are not available for the different compartments and have to be estimated according to the TGD based on the above-described screening tests and the partitioning behaviour of AA:

Table 3.2 Biodegradation rate constants for different compartments

Compartment / medium	Biodegradation rate
Activated sludge (WWTP)	k_{WWTP} = 1 h^{-1}
Surface water	$kdeg_{water}$ = 0.047 d^{-1}, (Table 5, TGD)
Sediment	$kbio_{sed}$ = 0.0023 d^{-1}
Soil	$kbio_{soil}$ = 0.047 d^{-1}, (See text)

3.1.2.2 Distribution

On the basis of a vapour pressure of 380 Pa and a water solubility of 1 kg/l (AA is miscible with water in all ratios) a Henry's law constant H = 0.027 Pa·m³/mol at 25°C was calculated, which suggests that AA is essentially non volatile. Due to its pKa value of 4.25 the dissociated anionic form of AA is dominating under normal environmental conditions (pH range 5.5-9). Therefore it has to be expected that the real partition of the total AA is determined only by its anionic form with a Henry's law constant being much lower than calculated above.

Adsorption and desorption of AA were examined on five different soils (an aquatic sandy loam sediment, a loamy sand, a clay loam and two loam soils). The Kp values for the adsorption phase ranged from 0.28 to 0.63 l/kg. The Kp values for the three desorption phases were scattered more widely with values ranging from 0.38 to 3.85 l/kg (Archer and Horvath, 1991). All adsorption and desorption results proved to be independent from the organic matter contents. It can be assumed that the adsorption behaviour of the anionic form of AA depends primarily on the inorganic fraction of the different soils. Therefore, and due to missing partition coefficients for different types of solid matter, it was preferred to use a uniform value of 1 l/kg for all partition coefficients (Kp soil, Kp sed, Kp susp, Kp sludge), which is very close to the value of 1.285 as exactly calculated from the given absorption and desorption ranges.

Using the fugacity model of Mackay (EQC model, level 1), the theoretical distribution of AA at equilibrium can be estimated:

Table 3.3 Equilibrium distribution according to fugacity model of Mackay (EQC model 1.0, level 1)

Compartment	%
Air	5.2
Water	94.7
Soil	0.1

Taking into account that the dissociated anionic form of AA is normally dominating (pKa 4.25), more than 99% of total AA has to be expected in the water compartment. In any case, the hydrosphere is the preferred target compartment for distribution.

Elimination in WWTPs

Based on log H = -1.57 and log Pow = 0.46, as well as the biodegradation rate of 1 h^{-1} in WWTP, the elimination through biodegradation and distribution can be estimated with the model SIMPLETREAT 3.0 (Version 1997):

Table 3.4 Elimination in WWTPs

Biodegradation (%)	87.3
Release to air (%)	0
Release to water (%)	12.7
Adsorption to sewage sludge (%)	0
Total elimination from water (%)	87.3

This distribution would not change, if due to the dominance of the anionic form of AA lower and more realistic values for log H and log Pow are assumed.

3.1.2.3 Accumulation

No experimental results on bioaccumulation are available. The measured log Pow of 0.46 does not indicate a potential for bioaccumulation though. Based on this value a **BCF of 0.49 l·kg^{-1}** can be estimated for fish according to the TGD (Chapter 4, Table 6). This calculation implies that the undissociated form of AA is dominating. For the anionic form a much lower BCF has to be expected.

The experimentally determined Kp values for soils in the range of 0.28 to 3.85 l/kg and the ready biodegradability also indicate no potential for geoaccumulation. Acrylic acid is highly mobile in soil and the amount which is not degraded may leach with seepage to the groundwater.

3.1.3 Aquatic compartment

Specific information from the production sites covering on-site processing is taken into account for the exposure assessment. Generic exposure scenarios for external processing and use are based on market information provided recently (1999) by the AA producers.

3.1.3.1 Estimation of $Clocal_{water}$ / Generic approach: production and processing

In the TGD, a generic (i.e. non site-specific) exposure scenario for the release of intermediates into surface water during production and processing is proposed (Emission Scenario Document IC-3).

The highest single production capacity of AA equals 330,000 t/a. Since this capacity is high enough to represent a realistic worst case, this amount is taken for default calculation, considering production and processing of AA at one site. However, as for the actual situation more specific data are available for the largest production site (see below), the generic scenario has not been carried through to risk characterisation. A **$Clocal_{water}$ of 270 µg/l** is estimated (default emission factors: 0.3% for production and 0.7% for processing; duration of emissions:

300 d/a; 87.3% elimination rate in WWTP according to SIMPLETREAT; default river flow rate according to ESD: 60 m^3/s; cf. Appendix A2 for calculation).

3.1.3.2 Estimation of $Clocal_{water}$ / Site-specific approach: production and processing

Using the available specific data for several production sites, more precise PEC estimations can be performed for the hydrosphere. The results are presented in **Table 3.5**.

Table 3.5 Basic data and results of local release estimations into the hydrosphere

Site	Flow of receiving water	Release to WWTP [t/a]	Release to hydrosphere [t/a]	$Clocal_{water}$ [µg/l]	Specific data used for exposure estimation
A	site specific	97	12.3	0.64	estimated annual emission based on measurements of WWTP effluent *; specific WWTP flow
B	site specific	1.28	0.162	0.16	estimated annual emission based on measurements of WWTP effluent; specific WWTP flow
C	site specific	0.08	0.01	0.77	calculated on the basis of effluent monitoring (n=16, two separate 8-d periods, max = 6.6 µg/l, min = 3.6 µg/l – one single measurement 1998: 10 µg/l, C_{local} based on average: 4.8 µg/l); specific WWTP flow, specific dilution
D **	site specific	[4.21]**)	[0.53] **)	[6.6] **)	measured concentrations in WWTP influent (n = 281 24 h composite samples in 1998; max =261 mg/l, 15 samples > 50 mg/l, 49 samples > 15 mg/l, 138 samples < 0.1 mg/l, C_{local} based on 90%ile: 29 mg/l); specific dilution factor
E	-	0	0	0	no release to controlled or surface waters, no wastewater generating process applied
F	-	0	0	0	no production since 1995; wastewater from production was incinerated before
G	-	0	0	0	no AA containing wastewater during regular operation, continuous process; possible releases to hydrosphere during cleaning operations only
H	-	0	0	0	no import since 1994
I	site specific	0.173	0.022	0.043	estimated annual emission based on measurements in WWTP effluent ***; specific WWTP flow
Total		**99**	**12.5**		

* Calculation is based on the detection limit of 100 µg/l, which was not exceeded in any case of 30 measurements. Further measurements applying another analytical method with lower detection limit have to be regarded as not representative (only 3 samples, average ca. 30 µg/l)

** Site D: AA manufacture ceased by October 1999 (confirmed by operating company in March 2000); releases not included in sum for estimation of regional exposure

*** Measurements of WWTP effluent were performed during a production period over one week (six samples, 24h sampling period each, sampling for 3 sec every 3 min); the detection limit was not exceeded and is the basis of calculation

3.1.3.3 Estimation of $Clocal_{water}$ / Generic and site-specific approach: processing by non producers/importers

For the EU, quantities of AA processed at production sites (i.e. captive use) compared to quantities processed at external sites, either of the same company or sold to other companies (non-producer / intermediary), have been communicated by industry.[5]

The production capacity of AA in the EU amounts to 810,000 t/a. A total of 830,000 t/a is taken as EU consumption figure (cf. Section 2.1.2). As mentioned above, half of this amount, i.e. 415,000 t/a, is esterified directly at the production sites. Further, a significant part of the remaining half is processed directly at the production sites, too. All releases due to processing of these quantities are covered by the site-specific emissions of the production sites (see Section 3.1.3.2, **Table 3.5**). Therefore, the estimation of releases due to processing at external downstream user sites can be limited to the remaining tonnage. The following main areas of use have been identified:

- production of super absorber polymers (SAP, wet and dry processes),
- wet polymerisation, namely dispersions, water-treatment agents, detergents, UV-curing agents, others,
- dry polymerisation.

All the main external processing types mentioned above comprise wet and dry polymerisation techniques. According to a comprehensive set of specific confidential information, provided by a significant portion of AA downstream users, an amount of 10,000 t/a processing volume at one generic single site is assumed as a realistic worst case.

Applying the respective default parameters according to the TGD (Table A3.10: emission factor 0.01, Table B3.9: emission episode 300 d, WWTP flow 2,000 m³/d, elimination rate 87.3%, dilution in surface water 1:10), gives:

$$\mathbf{Clocal_{water} = 2.12\ mg/l}$$

(the respective calculation is presented in Appendix A3).

From dry polymerisation no relevant releases with wastewater into the aquatic compartment have to be assumed (emission factor = 0 according to Table A3.10 of the TGD).

Based on additional information provided by external processing companies it was possible to refine the generic scenario and distinguish between production of SAP (wet) and wet polymerisation. The estimates are based on a comprehensive set of specific confidential information provided by downstream users.

<u>Production of super absorber polymers (SAP)</u>

Site-specific volumes of AA explicitly used for SAP production have been provided for almost 90% of AA used for external SAP-production. For almost 50% of the processing volume zero emission to the hydrosphere has been stated, justified by concise descriptions of employed wastewater reutilization / recycling systems. Specific emission data (measured effluent

[5] Important parts of submitted information have been provided on a strictly confidential basis, since sensitive marketing interests are concerned. Therefore, a number of calculation results are presented in a way, which is intended to prevent recalculation of use tonnages, especially of sensitive downstream use branches. The underlying input data and calculations are confidentially filed by the responsible authorities.

concentrations, specific flow rates of wastewater site effluent, receiving WWTP, receiving river) provided for a further portion of AA tonnage used, enabling estimation of $Clocal_{water}$, resulted in values up to:

$Clocal_{water}$ = 0.97 µg/l

Based on the known remaining annual AA tonnage not covered by the above-mentioned specific release data, and applying the respective default parameters according to the TGD, a

$Clocal_{water}$ = 339 µg/l

is calculated.

Wet polymerisation

The volumes of AA applied for wet polymerisation have been provided for thirteen European sites, covering about 30% of AA used for external wet polymerisation:

Number of plants	1	3	7	2
Annual use of AA [t]	< 500	500-1,000	1,000-5,000	5,000-10,000

Three further sites provided site-specific emission data without revealing their processing tonnages. For seven sites (covering ca. 12% of the tonnage), zero release to the hydrosphere has been stated, justified by a concise description of employed wastewater reutilization / recycling systems.

For eight sites processing > 500 t/a, a calculation of the $Clocal_{water}$ is performed based on site-specific tonnages or on measured AA effluent concentrations, incorporating site-specific information on wastewater treatment and dilution as far as available and default release factors if necessary. The underlying site-specific data are confidential. The results are compiled in **Table 3.6**.

Table 3.6 Specific $Clocal_{water}$ calculated for known processing sites applying external wet polymerisation

Site	Clocal $_{water}$ [µg/l]
K	0.004 *
L	0.18 *
M	1.25 *
N	8.70 *
O	12.70 *
P	0.20 *
Q	10,000.00 *
R	0.22 *

* based on measured AA effluent concentrations, sites K, L, M, N with specific dilution factor, sites L, Q without WWTP

The remaining ca. 70% of AA used for external wet polymerisation are not covered by the site-specific calculations presented above. A default calculation, applying the respective parameters according to the TGD (fraction of main source, emission episode, emission factor, standard

WWTP flow, elimination rate of 87.3%, dilution in surface water), results in a $Clocal_{water}$ significantly exceeding 100 µg/l. With regard to the specific Clocal values given in **Table 3.6** above, considering the different degrees of site-specific information incorporated, it can be concluded that the indicated generic Clocal represents a realistic worst case for this scenario "external wet polymerisation".

3.1.3.4 Estimation of $Clocal_{water}$ / Generic approach: use of polymers

According to **Table 2.1**, AA based polymers are mainly used in dispersions, superabsorbents and for treatments of leather and textiles. Additionally, application of water treatment agents has been identified as a relevant use. During these uses, releases of AA are possible due to residual monomeric AA present in the respective products.

Measurements of the residual AA contents in polymer dispersions have been communicated by two AA producers and EPDLA (European Polymer Dispersion and Latex Association, 1998), giving the following results:

Special polymeric dispersion	60 ppm
AA related polymeric dispersions	3-20 ppm; 100-1000 ppm; 1,000-2,500 ppm
Polymer latex	2.2 ppm
Desizing agents for textile finishing industry	110, 350, 370 ppm
Water treatment agents	200, 240, 400, 540, 960, 2,000 ppm

One producer stated residual monomeric AA contents in freshly manufactured products ranging from 260 to 4,000 grams per tonne, related to the initial amount of AA used. The results averaged 1,580 g/t. Compared to the above given range of residual monomeric contents in various products, the latter figure is independent from different portions of AA incorporated into different products. Therefore, this figure equals rather the quantity of monomer remaining unreacted after polymerisation of 1 tonne AA, the exact value primarily depending on applied polymerisation process engineering. This database is preferred for calculation of scenarios, where sufficiently specific residual monomeric AA contents are not known for the used products.

The application areas leather production, textile finishing, formulation of paints and application of water treatment agents are considered relevant for the assessment of local aquatic exposure scenarios. Private use of paints as well as use of superabsorber polymers (mainly in nappy pants, pantyliners and other consumer articles of hygiene) was not considered relevant for local assessment due to wide dispersion of these applications.

<u>Leather finishing</u>

During leather production, various finishing processes are presumably the main application area of AA containing products. Grain leather is generally surface treated by the addition of lacquer and/or resin containing colorants and other additives in either aqueous or organic solvent systems. Leather products typically need several coatings, which contain pigments or metal-complex dyes in dispersion matrices. Emission Scenario Document ESD IC-7 is applied, based on the following assumptions (see Appendix A4 for calculation):

- Percentage of dispersion applied to leather: 5%,
- Maximum residual monomeric AA content of product: 4,000 g/t (actually related to initial AA amount),

gives 0.05 t · 4,000 g/t = 200 g/t residual AA content. According to the ESD, 15 tonnes of leather are processed daily at one site. The calculations presented in Appendix A4 result in:

$Clocal_{water}$ = 0.95 µg/l

Due to insufficient information, two overly conservative assumptions are incorporated in this estimate: (1) the amount of residual AA is based on the assumption, that the applied dispersion contains 100% polyacrylate; (2) the calculation is based on 95% fixation degree, which appears to be an underestimation.

Textile finishing

In textile finishing, various processes are presumed to involve application of treatment agents containing AA based polymers. As an example, desizing agents are listed among the products, for which residual monomeric AA contents were communicated by industry (see above). Emission Scenario Document ESD IC-13 is applied, based on the following assumptions (see Appendix A5 for calculation):

- percentage of dispersion applied to textiles: 5%
- maximum residual monomeric AA content of product: 4000 g/t (actually related to initial AA amount)

gives 0.05 t · 4,000 g/t = 200 g/t residual AA content. According to the ESD, 3 tonnes of textiles are processed daily at one site. The calculations presented in Appendix A5 result in:

$Clocal_{water}$ = 0.19 µg/l

Due to insufficient information, two overly conservative assumptions are incorporated in this estimate: (1) the amount of residual AA is based on the assumption, that the applied dispersion contains 100% polyacrylate; (2) the calculation is based on 95% fixation degree, which appears to be an underestimation.

Paint formulation

The release of monomeric AA is possible during formulation of paints. In the Emission Scenario Document for paints, lacquers and varnishes industry (ESD IC-14, TGD, Chapter 7) no data are available on the release of monomeric substances during formulation of polymeric suspensions. The emissions are therefore estimated with the emission tables (Tables A2.1 and B2.3) presented in Appendix I of Chapter 3 of the TGD.

According to industry, about 15,000 t/a of AA are directly polymerised and used for manufacturing paints by downstream users. This equals a processing volume of 300,000 t/a dispersion solids according to EPDLA statement of 5% AA as typical use level in dispersion solids. Applying 0.4 as fraction of main source according to TGD Table B2.3, a use tonnage of 6,000 t/a AA (120,000 t/a dispersion solids) at one site would be assumed as a worst case.

Referring to additional information from industry, about 400 plants are formulating paints in Europe. Taking into account considerable ranges of plant size and degree of AA use, and considering the data on used AA tonnages as submitted by various downstream users (cf. **Table 3.6**), the processing volume of AA at one single site is assumed to be 2,000 t/a (equalling a processing volume of 40,000 t/a dispersion solids at this site) as a realistic worst case.

Considering a maximum monomeric AA content of 4,000 g/t, related to the initial AA tonnage, an amount of 2,000 t/a · 0.004 = 8 t/a monomeric AA is assumed for this scenario.

Applying release factor 0.003 (Table A2.1), 300 d processing (Table B2.3) and 2,000 m^3/d WWTP flow, the local concentration is estimated to (see Appendix A6 for calculation):

$Clocal_{water}$ = 0.51 µg/l

Application of water treatment agents

Consideration of acrylic polymers in water treatment agents is based on the following information (Schumann 1997; TEGEWA 1999): sodium acrylate is used as co-monomer in anionic polyacrylamides, which serve as coagulation aids in water treatment. Areas of application are raw, drinking and wastewater treatment and mineral processing. Use in the area of drinking water processing is controlled by other fora. About 50% of the total tonnage are used for mineral processing, 60% of this being used in closed circuits, whereby the water is reused after removal of the mineral solids. The amount used in municipal wastewater treatment is of minor quantitative importance compared to cationic products.

A generic estimation of $Clocal_{water}$ is based on a specific application rate ranging around 1 mg/l. Taking into account residual monomeric AA contents communicated for water treatment agents (200 ... 2,000 mg/kg), expected concentrations of residual AA in treated water range to at most 2 µg/l. Assuming default dilution 1:10,

$Clocal_{water}$ = 0.2 µg/l

This value implies the additional worst-case assumption, that all residual monomeric AA is released to water and no portion of AA is biodegraded, neither incorporated in the sludge and thus removed from the water phase.

3.1.3.5 Measured levels

No monitoring data for the aquatic environment are available.

AA measurements had been performed in the drainage water from a tunnel construction site in Norway. Grouting agents are used to reduce water leakage in constructions like tunnels and building parts which are exposed to high inward pressure of groundwater or in sewer systems. During the application of a new grouting agent containing magnesium diacrylate, high concentrations of AA have been found in the drainage water of the Norwegian tunnel.

During application of the product (about 10 t per injection, total amount of 57 t) AA concentrations up to 5.6 mg/l were detected in the drainage water. After injection was terminated, the concentrations decreased rapidly (within one week) to a level between 10 and 100 µg/l.

The drainage water was collected in a treatment plant and the effluent concentrations were analysed. In general, AA concentrations did not exceed 100 µg/l, but when the influent concentrations were above 1,000 µg/l, AA elimination was not significant and during the injection periods (about 2 weeks per injection) effluent concentrations between 2,000 and 5,600 µg/l were measured.

These values represent a site-specific situation and extrapolation to other tunnel constructing sites may not be appropriate due to varying draining conditions.

From the measured effluent concentrations the $Clocal_{water}$ in the receiving surface water can be calculated. The effluent is transported via the river *Alna* into the *Oslofjord*. As the *Alna* is partially covered by casing, it may not represent an ecosystem to be protected. The concentrations in the *Oslofjord* can be estimated using a default dilution factor of 10 for dilution in the *Alna* and an additional factor of 10 for dilution in the *Oslofjord*.

According to the available reports on the tunnel construction site (Aquateam, 1999), the calculation can also be performed on the basis of specific information on dilution. The annual average flow of the *Alna* of 75 m³/min is divided by a factor of 3 to estimate a low flow of 25 m³/min according to the TGD. The average flow of drainage water during the construction period (nearly one year) was 2,550 l/min. Dilution factors for the *Alna* in the *Oslofjord* are available for different distances from the mouth of the *Alna*.

Regarding a realistic worst-case situation the measured effluent concentration of 5.6 mg/l is used for the calculations of the $Clocal_{water}$:

Distance	100 m	200 m	300 m	400 m	500 m	default
Dilution factor	2	6	9	13	18	10
$Clocal_{water}$	280 µg/l	93 µg/l	62 µg/l	43 µg/l	31 µg/l	56 µg/l

3.1.3.6 Sediment

As neither monitoring data on concentrations of AA in sediment nor experimental results with benthic organisms are available and there is no evidence for relevant adsorption of AA onto sediment, there is no need to perform a quantitative risk assessment for this compartment. In addition, as AA dissociates in water and no correlation has been found between adsorption and organic carbon content for this substance the standard estimation method proposed in the TGD would not be applicable.

3.1.4 Atmosphere

3.1.4.1 Estimation of $Clocal_{air}$ / Generic approach: production and processing

No use category document for the release of intermediates into the atmosphere during production and processing is available at the moment. The emissions are therefore estimated with the emission tables presented in Chapter 3, Appendix I of the TGD (1996). A production and processing volume of 330,000 t/a is used for the generic approach corresponding to the highest amount produced at one single site. It is assumed that production and on-site processing can be assigned to main category Ib (isolated intermediates). The vapour pressure at 20°C being in the range of 100 to 1,000 Pa, release fractions of 0.0001 during production and of 0.00001 during on-site processing are proposed. External processing is considered in Section 3.1.4.3.

For the generic scenario, a resulting release of 36.3 t/a is calculated. Applying the OPS-model the local concentration and total annual deposition rate are calculated to (Fmainsource = 1, Temission = 300 d/a, $Fstp_{air} = 0$, $Fass_{aer} = 2.6\ 10^{-7}$; see Appendix A7 for calculation):

$$\mathbf{Clocal_{air} = 34\ \mu g/m^3}$$

$$\mathbf{DEPtotal_{ann} = 40\ \mu g \cdot m^{-2} \cdot d^{-1}}$$

3.1.4.2 Estimation of $Clocal_{air}$ / Site-specific approach: production and processing

In comparison to the generic approach a site-specific calculation using emission data submitted by industry leads to considerably lower values:

Table 3.7 Site-specific emissions to air, $Clocal_{air}$ and $DEPtotal_{ann}$

Site	Emission to air [t/a]	$Clocal_{air}$ [µg/m³]	$DEPtotal_{ann}$ [µg/m²/d]	Specific data used for calculation
A	8	6	9	specific release to air; no emission to air from WWTP
B	0	0	0	neglected due to incineration and flue-gas washing
C	4.8	4	5	specific release to air; no emission to air from WWTP
D	3	3	2	specific release to air; no emission to air from WWTP
E	0.5	0.5	0.4	default release to air (emission factor for processing: 0.0001); no emission to air from WWTP
F	0	0	0	no production or import since 1995
G	1.7	1	2	specific release to air; no emission to air from WWTP
H	0	0	0	no production or import since 1994
I	< 0.025	< 0.02	< 0.03	specific release to air; no emission to air from WWTP
Total	**18**			

3.1.4.3 Estimation of $Clocal_{air}$/ Generic approach: processing by non-producers/importers

Specific information on emissions to atmosphere is known only for a few downstream user sites stating zero emission to atmosphere. For one big processing site specific emission data to air have been communicated. The resulting emission factor is $7.3 \cdot 10^{-7}$. It should be noted, that this specific site applies very efficient air clean-up measures while the corresponding releases to hydrosphere are considerably higher (emission factor $1.2 \cdot 10^{-3}$ based on measured effluent concentrations). Additionally, emission data have been submitted for a number of individual processing sites located on-site at a large AA production site. Resulting emission factors to air range between $1 \cdot 10^{-7}$ and $3.4 \cdot 10^{-4}$. However, there were no data made available to the authors of this report, which could serve as a basis for reliable emission factors being representative for all types of downstream user sites.

All the main processing types applied externally (SAP production, wet polymerisation and dry polymerisation) comprise wet and dry polymerisation techniques. For AA, both wet and dry techniques are allocated to the same default emission factor of 0.01 (TGD, Table A3.10). However, for a generic calculation of downstream user emissions to atmosphere it appears reasonable to lower this factor to 0.001 with regard to the emission factors reported above. This approach covers the worst-case assumption, that the highest specific overall emissions out of all known sites would be directed entirely to air.

According to the comprehensive set of specific confidential information, provided by a significant portion of AA downstream users with regard to releases to the hydrosphere (cf.

Section 3.1.3.3), an amount of 10,000 t/a processing volume at one single site is assumed as realistic worst case.

Applying the respective default parameters according to the TGD (Table B3.9: emission episode 300 d - see Appendix A8 for calculation), gives:

$$\mathbf{Clocal_{air} = 9.3\ \mu g/m^3}$$

$$\mathbf{DEPtotal_{ann} = 11\ \mu g \cdot m^{-2} \cdot d^{-1}}$$

The end-use of polymer products (e.g. formulation and use of leather and textile finishing agents and paints) is considered to be not relevant for environmental exposure to monomeric AA on a local scale.

3.1.5 Terrestrial compartment

The release of AA to soil is expected to occur through atmospheric deposition after local release to the atmosphere at the production and processing site (see Section 3.1.4). The input through sludge application on agricultural soil is considered to be negligible, as AA does not partition to a significant extent to sewage sludge in the WWTP (see Section 3.1.1).

Direct releases to soil are possible due to the use of superabsorbents for soil amelioration. However, this application is assumed to be restricted to special areas like landfills or specific urban soils and is therefore considered as not relevant for natural soil.

Generic approach

With the generic deposition rate calculated in Section 3.1.4.1 ($DEPtotal_{ann} \cdot = 40\ \mu g \cdot m^{-2} \cdot d^{-1}$), equilibrium soil concentrations in direct vicinity to the generic production / processing plant can be calculated according to the TGD (see Appendix A9). The result concerning the ecosystem is:

$$\mathbf{Clocal_{soil} = 2.4\ \mu g/kg}$$

$$\mathbf{Clocal_{soil\text{-}porewater} = 2.4\ \mu g/l}$$

(other non-default input data: $K_{soil\_water} = 1.7$, $K_{air\_water} = 1.1 \cdot 10^{-5}$)

The generic deposition rate calculated in Section 3.1.4.3 for external processing sites is significantly lower ($DEPtotal_{ann} = 11\ \mu g \cdot m^{-2} \cdot d^{-1}$). Therefore, the $Clocal_{soil}$ resulting for production and processing sites covers all downstream user scenarios.

Site-specific approach

The local concentrations in soil calculated with site specific deposition rates for the known production and processing sites A to I (cf. **Table 3.7**) are significantly lower, for most sites below 0.5 µg/kg, and range to a maximum of 1.1 µg/kg. No specific deposition rates are known for the external processing sites of downstream user companies.

3.1.6 Secondary poisoning

As AA does not present indications of a bioaccumulation potential, a quantitative risk assessment for secondary poisoning is not required.

3.1.7 Regional concentrations

For determination of a regional background concentration all releases, from both point and diffuse sources, are considered. 20% of the total exposure quantity from point sources is taken into account for the defined regional EU standard model (densely populated area of 200·200 km with 20 million inhabitants), since AA is produced and processed in large tonnage at local sites. Therefore, the EUSES 1.0 default value for the fraction related to the region of 0.1 has been set to 0.2 for a more realistic regional scenario. This assumption is confirmed by the specific information recently provided by downstream users.

From diffuse sources, the default of 10% is considered for the standard region. The rest (80% from point sources and 90% from diffuse sources) of the total exposure quantity is taken into account for the continental model.

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

Since not all of the previously mentioned releases arising from use of the substance enter the hydrosphere directly, but instead primarily via the wastewater which is possibly purified in municipal wastewater treatment plants, a 70% connection to wastewater treatment plants, in which 87.3% of the substance is eliminated, is assumed for this scenario. The remaining 30% of the water is discharged directly into the hydrosphere.

Point releases

In **Table 3.5** the total annual release of 12.5 t/a AA to hydrosphere is allocated to all production and processing sites. The annual amount released to atmosphere totals 18 t/a (cf. **Table 3.7**). Point releases due to external processing and use are calculated for some generic exposure scenarios and for a number of specific sites as described in Sections 3.1.3.3 and 3.1.4.3. In order to keep the sensitive marketing figures confidential, overall sums are calculated for all releases to hydrosphere and atmosphere, which have been estimated for:

- SAP production based on site-specific information
- SAP production based on default calculation
- wet polymerisation based on site-specific information
- wet polymerisation based on default calculation
- dry polymerisation based on site-specific information
- dry polymerisation based on default calculation

The resulting releases are totalled up to 113 t to hydrosphere and 190 t to atmosphere.

Releases of residual monomeric AA during handling, use and disposal of AA based polymeric products are considered in an overall estimate as diffuse releases (see below). Consequently, the use scenarios considered in Section 3.1.3.4 (leather and textile finishing, formulation of paints, water treatment agents) have been calculated only for local exposure assessment for illustrative purposes.

Diffuse releases

For diffuse releases a conservative estimation is carried out, using the EU consumption amount of 830,000 t/a. A portion of about 38% of this (315,000 t/a) is polymerised to various AA-based polymeric products. Freshly manufactured products contain 260 ... 4,000 g/t monomeric AA, related to the initial amount of AA used (cf. Section 3.1.3.4). The average value of 1,580 g/t is used for calculation. Due to additional reaction steps, acrylate esters contain only about a hundredth of the residual monomeric AA reported above.

During use and disposal of the products the residual monomer can be washed out or evaporate. For the resulting monomer amount of 513 t/a (including 15 t/a residual monomeric AA from acrylate esters) it is assumed that 80% of the monomer (410 t/a) is released during formulation, use and disposal and that 20% remains in products which are incinerated.

As the aquatic compartment is the target compartment of AA, it is assumed that 70% of the releases (287 t/a) occur into the hydrosphere and 30% (123 t/a) into the atmosphere.

With a connection rate of 70% to WWTPs, 86 t/a are calculated to be released directly and 201 t/a into WWTPs of which 26 t/a (12.7%) are released with the effluent to surface water.

As mentioned at the beginning of this Section 3.1.7, for production, processing and formulation 20% of the point-source releases are assumed to occur into a region whereas according to the TGD from the diffuse releases only 10% are considered for the region.

The individual environmental releases are summarised in **Tables 3.8 and 3.9**:

Table 3.8 Summary of environmental releases

Life cycle stage	Distribution to regional / continental model [%/%]	Release into hydrosphere [t/a]			Release into atmosphere [t/a]
		Direct	WWTP	Surface water	
Production and on-site processing of AA	20/80	0	99	12.5	18
External processing by downstream users	20/80	0	891	113	190
Diffuse releases during handling, use and disposal of polymeric products	10/90	86	201	26	123

Table 3.9 Environmental releases for calculation of continental and regional model

	Continental model [t/a]	Regional model [t/a]
Air	0.8 · (18+190) t/a + 0.9 · 123 t/a = 277 t/a	0.2 · (18+190) t/a + 0.1 · 123 t/a = 54 t/a
Soil	--	--
Water direct	0.9 · 86 t/a = 77.4 t/a	0.1 · 86 t/a = 8.6 t/a
into WWTPs	0.8 · (99+891) t/a + 0.9 · 201 t/a = 973 t/a	0.2 · (99+891) t/a + 0.1 · 201 t/a = 218 t/a

In Appendix A10 the input and output figures of a SimpleBox 2.0 calculation adapted to the TGD and EUSES 1.00 are presented. The results of this calculation are consistent with EUSES. The resulting regional concentrations are:

$PEC_{regional_{aquatic}}$	**=**	**0.40**	**µg/l**
$PEC_{regional_{air}}$	**=**	**2**	**ng/m^3**
$PEC_{regional_{agr.-soil}}$	**=**	**0.02**	**µg/kg (wwt)**
$PEC_{regional_{agr.-soil\_porew}}$	**=**	**0.02**	**µg/l**
$PEC_{regional_{natural-soil}}$	**=**	**0.07**	**µg/kg (wwt)**

The calculated regional and continental distributions depend on vapour pressure, log Pow and Henry's law constant which are only valid for the undissociated form of AA. Since no other models are available to calculate the distribution for anionic AA, the values above are taken as an approximation.

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

3.2.1 Aquatic compartment

3.2.1.1 Available effect data

In the following, the most relevant results from acute and long-term toxicity tests with aquatic organisms are presented. Other results are available, but they are not valid as pH effects cannot be excluded (see IUCLID).

Vertebrates

Leuciscus idus (static, open system, nominal concentration) (Juhnke and Lüdemann, 1978)	48-h LC_{50}	315 mg/l
Brachydanio rerio (semi-static, open system, measured concentration) (Hüls, 1995b)	96-h LC_{50}	222 mg/l
Oncorhynchus mykiss (flow-through; measured concentration) (Bowman, 1990)	96-h LC_{50}	27 mg/l

Invertebrates

Daphnia magna (effect: immobilisation; static, open system, nominal concentration) (Bringmann and Kühn, 1982)	24-h EC_{50}	765 mg/l
Daphnia magna (effect: immobilisation; flow-through; measured concentration) (Burgess, 1989)	48-h EC_{50}	95 mg/l
Daphnia magna (effect: immobilisation; static, measured concentration) (Hüls, 1995c)	48-h EC_{50}	47 mg/l
Daphnia magna (effect: maternal toxicity; for reduction of the reproduction rate 21d-NOEC = 12 mg/l; semi-static test, measured concentrations) (Hüls, 1995d)	21-d NOEC	7 mg/l

Plants

Microcystis aeruginosa (blue-green algae)	8-d TGK	0.15 mg/l
Scenedesmus quadricauda (effect: biomass; nominal concentrations) (Bringmann and Kühn, 1977; 1978a)	8-d TGK	18 mg/l

The TGKs or "toxic threshold concentrations" were determined at 3% effect compared to the controls and can therefore be considered as NOECs.

Selenastrum capricornutum (effect: biomass; nominal concentrations; NOEC could not be determined, < 0,13 mg/l) (Forbis, 1989)	96-h EC_{50}	0.17 mg/l
Scenedesmus subspicatus (effect: b = biomass; μ = growth rate measured concentrations) (BASF, 1994b)	72-h E_bC_{50} 72-h E_bC_{10} 72-h NOEC 72-h $E_\mu C_{50}$ 72-h $E_\mu C_{10}$ 72-h NOEC	0.04 mg/l 0.01 mg/l 0.008 mg/l 0.13 mg/l 0.03 mg/l 0.016 mg/l
Scenedesmus subspicatus (effect: b = biomass; μ = growth rate measured concentrations) (Hüls, 1995e)	72-h E_bC_{50} 72-h E_bC_{10} 72-h NOEC 72-h $E_\mu C_{50}$ 72-h $E_\mu C_{10}$ 72-h NOEC	0.06 mg/l 0.01 mg/l < 0.01 mg/l 0.205 mg/l 0.031 mg/l 0.025 mg/l

Protozoa

Entosiphon sulcatum	72-h TGK	20 mg/l
Uronema parduczi	20-h TGK	11 mg/l
Chilomonas paramecium (effect: biomass; nominal concentrations), (Bringmann and Kühn, 1978a; 1980)	48-h TGK	0.9 mg/l

The TGKs or "toxic threshold concentrations" were determined at 5% effect compared to the controls and can therefore be considered as NOECs.

Bacteria

Pseudomonas putida (effect: biomass), (Bringmann and Kühn, 1977)	16-h TGK	41 mg/l

The TGK or "toxic threshold concentration" was determined at 3% effect compared to the controls and can therefore be considered as a NOEC.

activated sludge	30-min EC_{20} 30-min NOEC	900 mg/l 100 mg/l
(effect: respiration inhibition; nominal concentration) BASF, 1993)		

3.2.1.2 Determination of $PNEC_{aquatic}$

The above-cited test results with acrylic acid are only those which show a clear substance related toxicity and no pH effects. Acute and long-term tests reveal that algae are the most sensitive organisms. As their EC_{50} and NOEC values are more than two orders of magnitude lower than those for species of other trophic levels it is obvious that acrylic acid shows a specific toxicity to algae although for marine algae a high natural AA content is reported. (The result of one algae test showing a NOEC of 18 mg/l might be caused by recovery over a longer test period of eight days.)

For growth rate reduction, the lowest EC_{10} value derived in two tests (BASF, 1994b; Hüls, 1995e) was 0.03 mg/l for *Scenedesmus subspicatus*. The respective values based on biomass reduction are ≤ 0.01 mg/l. Striking correspondence is noted regarding most resulting EC values of these two independently conducted tests.

A recent study investigated the influence of growth pattern on effective concentrations (EC) of cell number, biomass integral and growth rate in the alga growth inhibition test (AGIT), systematically evaluating 38 existing AGITs and investigating several test scenarios by simulated AGITs (Ratte, 1998). In summary, it was concluded that preference of $EC_{biomass}$ or EC_{growth_rate} has to be based on a case-by-case decision, depending on various experimental conditions. For the majority of test scenarios, growth rate was found to provide the most reliable estimate of "true" toxicity, featuring the advantages of

- less susceptibility to experimental disturbances,
- independence of test duration,
- better statistics, and
- immediate ecological relevance.

Neither of the *Scenedesmus* test reports gives specific cause to prefer estimates based on biomass. Since a NOEC value depends on individual test design (intervals of test concentrations, number of replicates, variability of treatments and control), it is preferred to use available EC_{10} values for PNEC derivation, provided that a smooth concentration-response curve is obtained as in the present tests. Keeping in mind the above-mentioned arguments for using effect estimates based on growth rate, the $PNEC_{aqua}$ is derived from the $E_{\mu}C_{10}$ values. This conclusion is confirmed in a critical re-evaluation of the relevant AGITs by a distinguished expert (Nyholm, 1999).

Although long-term NOECs/EC_{10}-values are available from only two trophic levels, an assessment factor of 10 can be chosen because of the comparatively high toxicity of AA to algae. The acute EC_{50} values for fish are in the same range as those for daphnids and with high probability a NOEC for fish will not be lower than that of algae.

Therefore: **$PNEC_{aqua}$ = 30 µg/l / 10 = 3 µg/l.**

3.2.1.3 Determination of $PNEC_{microorganisms}$

There are three test results available which can be used for the derivation of this PNEC. According to the different endpoints and sensitivities of the test systems different assessment factors (AF) have to be applied:

Pseudomonas putida	NOEC (16 h) = 41 mg/l	AF = 1	⇒ PNEC = 41 mg/l
Uronema parduczi	NOEC (20 h) = 11 mg/l	AF = 1	⇒ PNEC = 11 mg/l
Chilomonas paramecium	NOEC (48 h) = 0.9 mg/l	AF = 1	⇒ PNEC = 0.9 mg/l
activated sludge	NOEC (30min) = 100 mg/l	AF = 10	⇒ PNEC = 10 mg/l

As a worst-case assumption a $PNEC_{microorganisms}$ of **0.9 mg/l** has to be used in the risk characterisation for municipal wastewater treatment plants.

For industrial on-site plants, a $PNEC_{microorganisms}$ of **10 mg/l** is proposed.

3.2.2 Atmosphere

Data on biotic or abiotic effects in the atmosphere are not available. Because of the short half-life, effects of AA are not to be expected.

3.2.3 Terrestrial compartment

To assess the effects of acrylic acid on terrestrial organisms, only a test on the respiration inhibition of natural soil microflora is available (Hossack et al., 1992). During 28 days of exposure, 100 mg/kg dry weight had no effects, while 1,000 mg/kg completely blocked the respiration rate. An EC_{50} was not determined. A light sandy loam soil with an organic carbon content of 0.3% was used.

Determination of $PNEC_{soil}$

Based on the above cited soil respiration test a derivation of a PNEC is possible. With an assessment factor of 1,000 a $PNEC_{soil}$ of 0.1 mg/kg would result. A conversion to standard soil is not necessary as the organic content of the soil is not critical for the adsorption behaviour of AA. On the other hand, according to the TGD, Chapter 3.6.2.1, the aquatic PNEC can be used for the risk characterisation of the soil ecosystem.

With a $PNEC_{water}$ = 3 µg/l and a K_{soil_water} = 1.7 m^3/m^3, the **$PNEC_{soil}$ amounts to 3 µg/kg.** As this is the lower value it will be taken for comparison with the PEC_{soil}.

3.2.4 Secondary poisoning

As AA does not present indications of a bioaccumulation potential, an effect assessment for secondary poisoning is not required.

3.3 RISK CHARACTERISATION

3.3.1 Aquatic compartment

3.3.1.1 Wastewater treatment plants

There is a need for further information and/or testing.

Because of the significant differences in responsibilities, functional control measures and data quality the possible risk to microorganisms is evaluated separately for municipal and industrial wastewater treatment plants (WWTP).

All WWTP at production and processing sites are considered as industrial plants (except site D, based on specific information). The highest effluent concentrations for industrial plants are reported for the external processing site O, which is equipped with an on-site WWTP:

$$\mathbf{PEC_{microorganisms} = 1\ mg/l}\ \text{(industrial plant site O, measured conc.)}$$

With a $\mathbf{PNEC_{microorganisms}}$ of **10 mg/l** for industrial plants, PEC/PNEC << 1 for all respective industrial sites.

The following effluent concentrations were calculated for the standard treatment plants of 2,000 m^3/d which might be considered as municipal plants, and for specific municipal plants, if respective data have been available:

Table 3.10 Calculation of effluents concentrations for wastewater treatment plants

$PEC_{microorganisms}$ [µg/l]	Scenario
246 [1)]	production site D, specific
3,390	external SAP production, wet process, default
990	external SAP production, wet process, highest specific
>> **1,000**	external wet polymerisation, default
0.04	external wet polymerisation, site K
19,050 [2)]	external wet polymerisation, site L
283	external wet polymerisation, site M
76	external wet polymerisation, site N
12,700 [2)]	external wet polymerisation, site Q
21,200	external wet polymerisation, generic site
9.5	leather finishing
1.9	textile finishing
5.1	formulation of paints
2	application of wastewater treatment agents

1) based on measured discharge concentration (90%ile), further dilution on entry into WWTP, elimination according to SIMPLETREAT

2) based on measured wastewater concentration, elimination in WWTP considered according to SIMPLETREAT - the respective specific sites communicated the statement “no biological WWTP”; nevertheless, these values are considered as realistic worst cases for a treatment plant

With a **$PNEC_{microorganisms}$** of **900 µg/l** for municipal plants, PEC/PNEC >> 1 for the downstream use scenarios of SAP production (default calculation and highest site-specific PEC figures) and wet polymerisation (default calculation and known sites L, Q).

An improvement of exposure data may in principle be possible e.g. by performing effluent measurements. However, $PEC_{microorganisms}$ for the known processing sites L and Q are based on measured AA effluent concentrations (ranges 40 … 100 mg/l and 50 … 150 mg/l). Likewise, the $PEC_{microorganisms}$ for a large known SAP producing site is based on measured effluent concentrations (n=9, range 29 … 850 mg/l; ranges in earlier years 0.2 … 360 mg/l, 41 … 1,430 mg/l, 40 …505 mg/l); this site shows quite low Clocal values only because of high dilution in the receiving wwtp and river (overall dilution factor > 100,000). These site-specific data indicate, that high effluent concentrations cannot be excluded. At the same time, gathering of sufficiently complete information for all downstream user sites applying wet polymerisation techniques seems not achievable with reasonable expenditure of time and money.

Since the $PNEC_{microorganisms}$ is derived from single species tests with ciliated protozoa, there is a need for further data reflecting the integrity of the native ciliate population in sewage sludge as a whole. However, regarding the conclusion for surface water (see below), it is accepted to postpone this testing need, since risk reduction measures necessary to remove concern for surface water will also cover the protection of wastewater treatment plants.

3.3.1.2 Surface water

In **Table 3.11** the comparison between PEC_{local} and $PNEC_{aqua}$ for all relevant exposure scenarios are presented (PNEC = 3 µg/l). As described in Section 3.1.7, the $PEC_{regional}$ is calculated to be 0.4 µg/l.

Regional model

The release assessment for the regional model was carried out on the basis of several calculated default emissions. Although received specific emission data have lowered the $PEC_{regional}$, it appears as impossible to gain specific and actual emission data for the whole area of downstream use with reasonable expenditure of time and money.

Local assessments

There is a need for further testing / gathering of exposure information or for limiting the risk.

As for several exposure scenarios PEC/PNEC >> 1, a risk for the aquatic compartment has to be deduced for the present data. **Table 3.11** gives an overview on all scenarios considered.

Table 3.11 Clocal, PEClocal and PEC/PNEC ratios for hydrosphere

Scenario	C_{local} [µg/l]	PEC_{local} [µg/l]	PEC/PNEC
Production / processing at site			
A	0.64	1.1	0.4
B	0.16	0.6	0.2
C	0.77	1.2	0.4
[D	*6.6*	*7.0*	*2.3]**
E	0	0.4	0.1
F	0	0.4	0.1
G	0	0.4	0.1
H	0	0.4	0.1
I	0.04	0.5	0.2
External processing			
SAP production (wet)			
default	339	339	**113**
highest specific	0.97	1.4	0.5
Polymerisation (wet)	>> 100	>> 100	**>> 33**
unknown sites default	2,120	2,120	**707**
generic site			
Known external processing sites			
K	0.004	0.4	0.1
L	0.18	0.6	0.2
M	1.25	1.7	0.6
N	8.7	9.1	**3.0**
O	12.7	13.1	**4.4**
P	0.2	0.6	0.2
Q	10,000	10,000	**3,333**
R	0.22	0.62	0.2
Use in leather finishing (default)	0.95	1.5	0.5
Use in textile finishing (default)	0.19	0.6	0.2
Use in paint formulation (default)	0.51	0.9	0.3
Use of grouting agent (default)	56	56	**19**
100 m from the mouth of river	280	280	**93**

* Site D: manufacture of AA ceased by October 1999 (confirmed by operating company in March 2000)

Producers / importers

At site D, the respective C_{local} exceeds the PNEC by a factor of more than two. The calculation is based on measurements of AA concentrations in WWTP influent. Specific updated dilution factors have been provided. For a refined estimate of site-specific releases to hydrosphere, detailed results of discharge monitoring and specific flow rates have been submitted. 20% out of the 281 24-hour composite samples showed AA concentrations resulting in PEC/PNEC ratios exceeding one. Nonetheless, further risk reduction measures are not necessary for this site because AA manufacture was ceased by October 1999 (confirmed by operating company in March 2000).

External processing, use of polymers

For several known downstream users where AA is applied for wet polymerisation processes, for the default scenarios of wet polymerisation and wet SAP production covering tonnages without

specific release data, and for the use of a grouting agent, PEC/PNEC ratios above one are calculated and a risk for the aquatic compartment has to be deduced on the basis of the present data. Risk reduction measures are recommended.

As an assessment factor of 10 is used for PNEC derivation, it is not likely to remove the concern by further testing.

For the wet polymerisation scenarios an improvement of exposure data may in principle be possible e.g. by performing effluent measurements. However, it is questionable whether sufficiently complete representative monitoring data from all downstream users can be obtained with reasonable expenditure of time and money. Moreover, regarding the dynamic year-to-year variations of used AA amounts, being typical for this market, the goal of sufficiently comprehensive collection of up-to-date monitoring data appears not appropriate. As additional background for the present risk characterisation, the following points should be noted:

- Site-specific information accounts for ca. 90% of AA externally used in SAP production and for ca. 30% of AA externally used in wet polymerisation processes. Only a limited part of this information is sufficient for derivation of emission factors, the calculated figures ranging between $2 \cdot 10^{-6}$ and $1.2 \cdot 10^{-3}$ (TGD default $1 \cdot 10^{-2}$).

- For both SAP production sites and wet polymerisation sites, regular effluent concentrations up to 100 mg/l AA and significantly more have been reported. At two of these sites, comparatively low C_{local} figures result only because of high dilution factors (magnitude 10^6). However, these data indicate that high effluent concentrations cannot be excluded, even if certain types of process engineering are applied.

- On the other hand, application of wastewater reutilization / recycling systems is known to result in zero emissions to the hydrosphere at a number of downstream user sites which are processing about 50% of AA used externally for SAP production and about 12% of AA used externally in wet polymerisation processes.

During the use of a grouting agent containing magnesium diacrylate high concentrations of AA are released via drainage water. The exposure assessment is based on measured concentrations of drainage water at a tunnel construction site. A quantitative extrapolation to other construction sites seems difficult, but similar conditions might be anticipated. Measures appropriate to local circumstances should be applied.

The use of water based emulsion polymers, containing residual AA, covers a wide range of applications, different products and used technologies making it nearly impossible to calculate a resulting PEC for each application. Therefore it has to be noted that most of the polymer applications could not be assessed specifically in Section 3.1.3.4 due to the lack of representative information for emulsion acrylates.

3.3.1.3 Sediment

Neither monitoring data on concentrations of AA in sediment nor experimental results with benthic organisms are available. As there is no evidence for relevant adsorption of AA onto sediment, there is no need for performing a quantitative risk assessment for this compartment. From the current manufacturing and use of AA no risk for the sediment compartment is expected.

3.3.2 Atmosphere

Due to the physical properties of AA and the Mackay distribution (see Section 3.1.1) the atmosphere is not regarded as a target of distribution. Emissions of AA into the atmosphere occur only on a local scale (concentrations in a distance of 100 m to the point source are between <0.02 and 9.3 µg/m^3). Furthermore AA reveals a short half-life for atmospheric photooxidation of 39.6 h (oxidation by hydroxyl radicals) or 6.5 d (oxidation by ozone). With these data the regional PEC for the atmosphere was calculated to 2 ng/m^3. Because of these features a risk to the atmosphere by abiotic or biotic effects is not to be expected and the performance of a plant fumigation test is not considered as high priority.

3.3.3 Terrestrial compartment

A generic exposure scenario representing a worst-case situation in the vicinity of a production and processing site was used for the calculation of the concentration in the soil porewater due to atmospheric deposition. This scenario covers also the refined generic estimation for exposure of soil in vicinity of a downstream user site applying wet or dry polymerisation processes, including SAP production.

The $Clocal_{soil\text{-}porewater}$ was 2.4 µg/l in the vicinity of such a site and on a regional scale a concentration of 0.02 µg/l was estimated.

An indicative risk assessment can be performed on the basis of the aquatic PNEC resulting in a $PNEC_{soil}$ of 3 µg/l (soil pore water, cf. Section 3.2.3). Considering this PNEC as a quite conservative figure, the resulting PEC/PNEC ratio of 0.8 can be regarded as maximum for the generic worst-case scenario production and processing. For the site-specific scenarios for production and processing as well as for the generic scenario for external processing PEC/PNEC ratios between < 0.16 and 0.36 are resulting.

For the aquatic compartment, algae were the most sensitive species tested. Extrapolation of the toxicity to terrestrial plants is difficult due to physiological and exposure route differences. Thus, performance of a terrestrial plant test would be desirable. However, as the PNEC derived from aquatic tests is more than 30 times lower than the PNEC derived from the available soil microorganism test and in addition the exposure of the terrestrial compartment is only a local problem, additional testing of higher plants is not considered as high priority.

Therefore, no risk for the soil compartment is expected from the current manufacturing and use of AA.

3.3.4 Secondary poisoning

As AA does not present indications of a bioaccumulation potential, a risk characterisation for secondary poisoning is not required.

4 HUMAN HEALTH

4.1 HUMAN HEALTH (TOXICITY)

4.1.1 Exposure assessment

4.1.1.1 General discussion

In the Swedish product register, 13 out of 56 products containing acrylic acid are used by consumers.

In the Federal Republic of Germany, products containing acrylic acid are also used by consumers. Thus, the consumer may be exposed to acrylic acid. This may occur via the inhalation, dermal or oral route.

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

Acrylic acid is primarily used as a chemical intermediate which is further processed to acrylic esters, homopolymers and copolymers. For some of the processed products e.g. water treatment materials and sizing preparations manufactured from acrylic acid the content of the residual monomer is known (less than 900 ppm). Further information on the monomer content in other products such as paints is not available. By comparison with the concentration of monomeric methacrylic acid in paints (up to 700 ppm) it may be concluded, that the content of acrylic acid lies in the same range. Adhesives may contain up to 10% acrylic acid.

Acrylic acid may arise as a decomposition product during the production of printed circuit boards and during the removal of paints using gas flames.

The substance may also be released during the use of grouting agents.

4.1.1.2 Occupational exposure

The following occupational exposure limits are established for acrylic acid (ILO, 1994):

UK, CH, S, US (NIOSH/OSHA)	30 mg/m³ (10 ml/m³)
B	29 mg/m³ (10 ml/m³)
AUS, US(ACGIH), NL*, DK**, F	5.9 mg/m³ (2 ml/m³)

and the following short-term exposure limits are established for acrylic acid (ILO, 1994):

UK	60 mg/m³ (20 ml/m³)
S	45 mg/m³ (15 ml/m³)
F	30 mg/m³ (10 ml/m^3)

* De Nationale MAC lijst (Ministerie van Sociale Zaken en Werkgelegenheid, 1996)

** Grænseværdier for stoffer og materialer (Arbejdstilsynet, 1996)

For occupational exposure, exposures during the
- production and further processing of acrylic acid,
- manufacture of adhesives,
- use of adhesives containing acrylic acid,
- decomposition of photoresist materials,
- gas flame removal of paints

is considered.

Acrylic acid may be released from grouting agents used to reduce water leakage in constructions like tunnels and building parts which are exposed to high inward pressure of groundwater or in sewer systems. During application AA concentrations up to 5.6 mg/l were detected in the drainage water. For the highly automated rehabilitation method in use (injection procedure) exposure may occur during tasks like cleaning of the equipment. The exposure level depends on the dissociation of the acrylate compound within the grouting agent (< 10% w/w) during the grouting works. Since the producer confirmed that the used acrylate dissolves only slightly to acrylic acid at the given conditions, within the framework of this exposure assessment, it is assumed that this scenario is of minor relevance.

4.1.1.2.1 Occupational exposure during production and further processing in the large-scale chemical industry

Production and further processing as a chemical intermediate

Acrylic acid is primarily used as a chemical intermediate which is further processed to acrylic esters, homopolymers and copolymers. Production and further processing are performed in closed systems which may be breached during sampling, transfer, filling, cleaning, maintenance, and repair works. According to information provided by a manufacturer acrylic acid is distributed in railway tank wagons or trucks and is transferred into storage tanks and reactors via closed pipes (gas displacement device).

Products manufactured from acrylic acid (e.g. sizing preparations, water treatment materials) contain less than 900 ppm acrylic acid monomer. For adhesives it is known that the monomer is present at concentrations of 1-10%. Further information of monomer content in other products e.g. paints is not available. By comparison with the concentration of monomeric methacrylic acid in paints (up to 700 ppm) it may be derived, that the content of acrylic acid lies in the same range.

Pure acrylic acid and preparations containing ≥ 5% AA are labelled as corrosive.

Workplace measurements

Results of workplace measurements provided by the manufacturers are presented in the following table, and are classified according to 8-hour TWA and short-term measurements.

Table 4.1 Acrylic acid exposures at workplaces during production and further processing

Job category / activities	Year of measure-ment	Number of samples	Range of measurement data [mg/m³]	Geometric mean [mg/m³]	90th-percentile [mg/m³]	Duration and frequency
8-h TWA						
Manufacture (not specified in detail)	1987 – 1997	83	0.06 - 7.8 [1)]	0.51	----	----
Manufacture, distillation	1993	3	0.33 - 0.67 [2)]	----	----	----
Manufacture, polymerisation, charging, piping into vessels	1994 – 1996	4 16	0.13 - 2.59 0.01 - 0.46	----	----	----
Manufacture / further processing	1990 – 1996	245	< 15	----	----	----
Esterification (not specified in detail)	1986 – 1994	37	0.03 - 6.0	----	----	----
Polymerisation (not specified in detail)	1986 – 1994	18	0.03 - 6.0	----	---	----
Filling	1986 – 1994	10	0.03 - 6.0	----	----	----
Pilot plant / laboratory (not specified in detail)	1986 – 1994	36	0.03 - 6.0	----	----	----
Short-term values						
Manufacture	1987 – 1997	36	0.06 - 29.4 [1)]	0.7	----	----
Filling drums	1987 – 1997	12	0.0.78 - 34.8 [1)]	5.5	----	----
Loading/unloading tank trucks / railway tank wagons	1987 – 1997	84	0.03 - 187.2 [1)]	1.38	----	----
Charging acrylic acid	1987 – 1997	121	0.3 - 44.4 [1)]	1.65	----	----
Laboratory, development work, quality control	1987 – 1997	51	0.06 - 11.43[1)]	0.63	----	----

1) analytical method using HPLC or GC not described in detail
2) derivatisation to ethyl ester, detection with GC-FID

The applied analytical method comprises HPLC with UV detection after adsorption of the substance to a special scavenger (XAD-8) and desorbing with a methanol/water mixture (OSHA No. 28). The detection limit of the method is 0.042 mg/m^3 (0.002 ml/m^3). Two manufacturers used different analytical methods (see **Table 4.1**).

The measurement results are regarded as valid, although the results are based on workplaces and activities which, in part, are described in general terms only. In addition, further detailed information on the duration and frequency of exposure as well as on the collective of the exposed workers is missing.

The shift average values (8-h TWA) are measured between 0.03 and < 15 mg/m^3 (0.01-5 ml/m^3) with a 90th percentile at 3 mg/m³ (1 ml/m^3) for different tasks, short-term values between 0.03-187.2 mg/m³ (0.01-62.4 ml/m^3) without calculation of the 90th percentile. Taken into account the geometric mean values between 0.63-5.5 mg/m³ (0.2-1.8 ml/m^3), the maximum of 187.2 mg/m³ (62.4 ml/m^3) is assumed as an outlier.

Because on the basis of the available data it is not possible to calculate the 90th percentile (reasonable worst case), the next available result of 44.4 mg/m³ (14.8 ml/m^3) is taken as representing a 90th percentile.

Manufacture of adhesives

Acrylic acid is used as an additive for the production of one- and two-package (anaerobic and radiation-hardened) polymerisation adhesives. In the manufacture of these special-purpose adhesives for high-quality bonding of metal, acrylic acid monomer is added (concentration: 1-10%) to improve adhesion. Adhesives are manufactured either quasi continuously or batchwise in both closed and partially open systems (lidded mixer). For the production of solvent-based adhesives it is known, that only high volume preparations are produced quasi continuously. Therefore batchwise production is to be assumed within the chemical industry, whereby partially open systems operate in conjunction with local ventilation equipment.

Within the chemical industry exposure is possible during sampling and analysis, filling and drumming, as well as during cleaning, maintenance and repair work. Because neither workplace measurements nor detailed information on the duration and frequency of exposure are available, exposure assessment is performed applying the EASE model (see Section 4.1.1.2.4) assuming daily exposure for two hours (manufacturing of formulations).

Dermal exposure during manufacture and further processing in the large-scale chemical industry

On account of the corrosive effect of pure acrylic acid as well as of preparations (labelled as corrosive at a content of ≥ 5%) and taking into consideration the highly accepted use of suitable protective equipment within the large-scale chemical industry, it can be assumed that, as a rule, daily repeated skin contact is avoided to a large extent by using suitable personal protective equipment (gloves and eye protection). During activities like drumming, filling, cleaning and maintenance potential exposure is assumed only by single contacts. The corresponding exposure level is assessed by the EASE model.

Daily repeated dermal exposure is assessed to be low.

4.1.1.2.2 Occupational exposure in the further processing industries, outside the chemical industry

Manufacture of adhesives

Further processing of acrylic acid to special-purpose adhesives may not be limited to the large-scale chemical industry but occurs in the industrial area, too, as well as in small and medium-sized chemical companies. Batchwise production is also assumed (see Section 4.1.1.2.1).

In these areas it cannot be excluded, that the substance is handled in open systems during certain tasks, e.g. metering and filling activities, and that suitable technical measures (LEV, local exhaust ventilation) are not used (Voullaire and Kliemt, 1995).

Inhalation exposure is possible during sampling and analysis, filling and drumming, as well as during cleaning, maintenance, and repair work. Because neither workplace measurements nor detailed information on the duration and frequency of exposure is available, exposure assessment is performed applying the EASE model (see Section 4.1.1.2.4) assuming daily exposure for two hours (manufacturing of formulations).

The use of personal protective equipment (gloves and eye protection) is assumed during certain tasks (e.g. filling, drumming) considering the corrosive effect of the pure acrylic acid and the produced preparations (content of AA ≥ 5%). Furthermore at a site at which corrosive products are handled it is assumed, that the workers will protect themselves even if they handle preparations with irritant effects. The daily dermal exposure for these scenarios is assessed to be low. During activities like filling, transfer, cleaning, maintenance and repair work, potential exposure is assumed only by single contacts.

Use of adhesives in the further processing industry

In the field of engineering, device and tool construction industries, one- and two- package adhesives with up to 10% acrylic acid are used to bond metals either anaerobically or radiation-hardened during assembly. Automatic or semi-automatic bonding machines are employed within continuous production processes (production lines). It is assumed that low amounts of the adhesives are applied to bond small areas. If radiation-hardened adhesives are used the bonded workpiece is hardened by UV light within closed systems. The components, which are still warm, are in some cases stored in open systems.

Inhalation and dermal exposure is possible during charging and bonding work (semi-automatic machines), during cleaning, maintenance and repair work, and during work in the vicinity of openly-stored components which are still warm. It is to be assumed that not every production plant is equipped with suitable technical ventilation equipment and it cannot be excluded that PPE (gloves) is not worn (Voullaire and Kliemt, 1995), if non-corrosive adhesives are handled.

On account of the corrosive effect of adhesives (content of AA ≥ 5%), it can be assumed, as a rule, that daily repeated immediate skin contact is avoided to a large extent by using suitable personal protective equipment (gloves and eye protection). In this case daily dermal exposure is assessed to be low. During activities like filling, cleaning and maintenance potential exposure is assumed only by single contacts. The corresponding exposure level is assessed applying the EASE model (see Section 4.1.1.2.4)

In the case of handling adhesives not labelled as corrosive, frequent immediate skin contact has to be taken into consideration. Generally workers avoid immediate skin contact with adhesives that can be removed only with difficulties (Kliemt, 1995). The corresponding adhesives could be removed more easily because they harden only slowly, and thus have the opportunity to penetrate the skin. These adhesives are removed later with the aid of skin cleaning agents which are also employed after contact with paints. The corresponding exposure level is assessed applying the EASE model (see Section 4.1.1.2.4).

Neither workplace measurements nor information on the duration and frequency of exposure or on the collective of the exposed group are available.

Decomposition of photoresist materials during the production of integrated circuits

Acrylic acid may be released as a decomposition product during the production of integrated circuits or in the galvanic industry, when photoresist materials (e.g. methyl methacrylate) are partially depolymerized by means of UV light. The subsequent development with dissolving the depolymerized material is performed in closed systems (Reichert, 1993), so that it is to be assumed that inhalation exposure to acrylic acid can be neglected. No information could be obtained whether the UV light treatment occurs in closed systems or if the workplaces are equipped with local exhaust ventilation. However, it is assumed that during UV light treatment

only a part of the formed acrylic acid is evaporated, and, unless other information is provided, that inhalation exposure is low.

Because acrylic acid is released during thermal processes, normally no immediate skin contact occurs. The dermal exposure level caused by touching of acrylic acid contaminated surfaces (indirect exposure) is regarded as being low.

4.1.1.2.3 Occupational exposure in the skilled trade sector

Use of adhesives

Adhesives which contain acrylic acid are used for metal repair e.g. workpieces. During repair works, which may involve rather small areas, workers may be subjected to inhalation and dermal exposure. It may be assumed that exhaust ventilation systems are absent, and that suitable personal protective equipment is not worn if adhesives not labelled as corrosive are handled. For further description of dermal exposure during the handling of corrosive adhesives and adhesives not labelled as corrosive see Section 4.1.1.2.2, "use of adhesives in the further processing industry".

Neither workplace measurements nor information on the duration and frequency of exposure or on the collective of the exposed group are available.

It is to be assumed, that the adhesives are not handled daily and that the duration is much shorter than the shift length.

Gas flame removal of paints

Acrylic acid may be released as a thermal decomposition product when paints are removed from steel or concrete surfaces by means of gas flames.

Occupational exposure measurements (n=6) during the removal of old paints from windows with gas flame or with a hot-air blower under realistic workplace conditions show no release of acrylic acid above the detection limits of 0.01-0.06 ppm (Työterveyslaitos, written communication from 08.12.1998).

In laboratory experiments acrylic acid was detected as a decomposition product during gas flame removal of 8 out of 10 steel protective paints, used in the Finnish shipyards (Henricks-Eckerman et al., 1990). The emissions of acrylic acid were determined between 0.2-1 mg/m^3 (n = 8).

The air concentration of free acrylic acid during normal working processes involving welding, flame cutting or straightening of painted steel sheets is estimated to be approximately three orders of magnitude lower (Työterveyslaitos, written communication from 08.12.1998).

Because acrylic acid is released during thermal processes, normally no immediate skin contact occurs. According to a rough estimation of dermal exposure caused by touching acrylic acid contaminated surfaces (indirect exposure), the exposure level is regarded as being low.

4.1.1.2.4 Estimation of the exposure according to the EASE model

The estimation of the level of inhalation and dermal exposure performed on accordance with the EASE model (TGD) produces the following results:

Inhalation exposure

a) Exposure to acrylic acid vapour during the manufacture and further processing of acrylic acid in the large-scale chemical industry:

Input parameters:	T = 20°C closed system significant breaching LEV present
Estimated exposure:	1.5-9 mg/m³ (0.5-3 ml/m³)

b) Use of adhesives containing acrylic acid, with local exhaust ventilation:

Input parameters:	T = 20°C non dispersive use direct handling LEV present
Estimated exposure:	1.5-9 mg/m³ (0.5-3 ml/m³)

c) Exposure to acrylic acid vapour when adhesives on the basis of acrylic acid are produced or adhesives containing acrylic acid are used, without suitable local exhaust ventilation:

Input parameters:	T = 20°C non dispersive use direct handling dilution ventilation
Estimated exposure:	30-150 mg/m³ (10-50 ml/m³)

The partial vapour pressure of acrylic acid cannot be considered, because the composition of the adhesives is not known. Therefore it is assumed for the above estimation that the partial vapour pressure is in the same EASE category as the vapour pressure of pure acrylic acid.

The EASE model cannot be applied to estimate the exposure to acrylic acid as a decomposition product (e.g. gas flame removal of paints).

Dermal exposure

a) Potential dermal exposure during production and further processing of acrylic acid in the chemical industry and in the industrial sector as well as during the use of adhesives (labelled as corrosive at a content of ≥ 5%):

Input parameters:	T = 20°C non dispersive use direct handling incidental
Estimated exposure:	0-0.1 mg/cm²/day

Use of adhesives: Considering a content of 10% AA in adhesives, dermal exposure amounts to 0-0.01 mg/cm²/day

b) Dermal exposure via immediate contact with adhesives (not labelled as corrosive) which are used:

Input parameters:	T = 20°C non dispersive use direct handling intermittent
Estimated exposure:	0.1-1 mg/cm²/day

Use of adhesives: Considering a content of 5% acrylic acid in adhesives, dermal exposure amounts to: 0.005-0.05 mg/cm²/day

4.1.1.2.5 Integrated Assessment

General

Acrylic acid is primarily used as a chemical intermediate which is further processed to acrylic esters, homopolymers and copolymers. For some of the processed products e.g. water treatment materials and sizing preparations manufactured from acrylic acid the content of the residual monomer is known (less than 900 ppm). Further information on the monomer content in other products e.g. paints is not available. By comparison with the concentration of monomeric methacrylic acid in paints (up to 700 ppm) it may be concluded, that the content of acrylic acid lies in the same range. Anaerobic and radiation-hardened polymerisation adhesives may contain up to 10% acrylic acid.

Production and further processing in the large-scale chemical industry

Production and further processing as a chemical intermediate

The submitted measurement results are regarded as valid although the results are based on workplaces and activities which, in part, are described in general terms only. In addition further detailed information on the duration and frequency of exposure as well as on the collective of the exposed group is missing. The undifferentiated presentation of the measurement results makes it impossible to state exposure levels typical for particular workplaces.

The measurements in the range of 0.03-7.8 mg/m^3 (0.01-3.9 ml/m³) agree well with the inhalation exposure level of 1.5-9 mg/m³ (0.5-3 ml/m³) which is estimated in application of the EASE model. For the assessment of the risks of inhalation exposure to acrylic acid during manufacture and further processing in the chemical industry, the 90th percentile of one measurement collective of 3 mg/m^3 (1 ml/m^3) is to be assumed, whereby the short-term exposure may be even higher during certain activities: concentrations as high as 187.2 mg/m³ (62.4 ml/m³) have been measured when tankers or railway tank wagons are loaded and unloaded. For the assessment of the risk 44.4 mg/m³ (14.8 ml/m^3) should be used as a reasonable worst case (see Section 4.1.1.2.1).

Manufacture of special-purpose adhesives in the large-scale chemical industry

Within the chemical industry inhalation exposure is possible during sampling and analysis, filling and drumming, as well as during cleaning, maintenance, and repair work. Because neither workplace measurements nor detailed information on the duration and frequency of exposure are available, exposure assessment is performed applying the EASE model (see Section 4.1.1.2.4) assuming daily exposure for two hours (manufacturing of formulations). For the assessment of the risks of inhalation exposure to acrylic acid during manufacture of adhesives exposures of 0.375-2.25 mg/m^3 (0.125-0.75 ml/m^3, with LEV, 2 h) estimated according to the EASE model are to be assumed.

Dermal exposure

On account of the corrosivity of pure acrylic acid and adhesives (labelled as corrosive at a content of ≥ 5% AA) immediate skin contact is only assumed by single contacts, because, in general, suitable personal protection equipment (gloves and eye protection) are worn to avoid the contact to a large extent. The estimation of a potential exposure by single contacts according to the EASE model is about 0-0.1 mg/cm^2/day; on account of an exposed skin area of about 420 cm^2 an exposure level of 0-42 mg/person/day would result. Taken into consideration that the use of gloves has a high acceptance within the chemical industry, daily dermal exposure is assessed to be low, even if non-corrosive adhesives are handled.

Occupational exposure in the further processing industry, outside the large-scale chemical industry

Manufacture of special-purpose adhesives

Further processing of acrylic acid to one- and two-package polymerisation adhesives may not be limited to the large-scale chemical industry but occurs in the industrial area, too, as well as in small and medium-sized chemical companies. In these areas it cannot be excluded, that acrylic acid is handled also in open systems during certain tasks, e.g. metering and filling activities, and that no suitable technical measures (LEV, local exhaust ventilation) are used, when preparations not labelled as corrosive are handled.

Since no workplace measurements are available, the EASE model is applied to estimate inhalation exposure. In the case of workplaces provided with suitable local exhaust ventilation systems, the inhalation exposure is calculated to 1.5-9 mg/m^3 (0.5-3 ml/m^3). If it is assumed that no local exhaust ventilation system is present, the level of inhalation exposure amounts to 30-150 mg/m^3 (10-50 ml/m^3). Taking into consideration a daily duration of exposure of 2 hours, exposures of 0.375-2.25 mg/m^3 (0.125-0.75 ml/m^3, with LEV) or 7.5-37.5 mg/m^3 (2.5-12.5 ml/m^3, without LEV) result.

The use of personal protective equipment (gloves and eye protection) is assumed during certain tasks (e.g. filling, drumming) considering the corrosive effect of the pure acrylic acid and the produced preparations (content of AA ≥ 5%). Furthermore for a site at which corrosive products are handled it is assumed, that the workers will protect themselves even if they handle preparations with irritant effects. The daily dermal exposure for these scenarios is assessed to be low. During activities like filling, transfer, cleaning, maintenance, and repair work, potential exposure is assumed only by single contacts. Considering the exposure level of 0-0.1 mg/cm^2/day assessed using the EASE model and an exposed area of 420 cm^2, the exposure amounts to 0-42 mg/person/day.

Use of special-purpose adhesives in the further processing industries

It is to be assumed that in the industrial sector, adhesives (containing 1-10% acrylic acid) are sometimes handled in open systems during certain activities such as dosage, filling and bonding. Further, if radiation-hardened adhesives are used, acrylic acid can partially evaporate after the (UV) hardening process if the warm workpiece is stored openly. Estimation in application of the EASE model (measurements are not available) produces a potential exposure of 1.5 – 9 mg/m³ (0.5-3 ml/m³) for workplaces with local exhaust ventilation and 30-150 mg/m³ (10–50 ml/m³) for workplaces without local exhaust ventilation. For workplaces without local exhaust ventilation, the lower value of the estimated concentration range (30 mg/m^3) appears more realistic in view of the method of use (bonding small areas).

In the case of handling adhesives (labelled as corrosive at ≥ 5%) potential dermal exposure is assumed only by single contacts. The exposure level estimated using the EASE model amounts to 0.-0.01 mg/cm²/day. Generally, small areas of the body are affected. Assuming that an area of 210 cm^2 (fingers) is exposed a level of 0-2.1 mg/person/day is obtained. Taking into account the corrosive effect of adhesives, daily repeated skin contact is avoided to a large extent by using suitable personal protective equipment, so that daily dermal exposure is assessed to be low.

When preparations not labelled as corrosive (containing < 5% acrylic acid) are handled, it cannot be excluded that gloves are not worn (see Section 4.1.1.2.2). In this case daily dermal exposure is assessed applying the EASE model to 0.005-0.05 mg/cm^2/day. Considering an exposed area of 210 cm^2, dermal exposure amounts to 1-10.5 mg/person/day.

Decomposition of photoresist materials during the production of integrated circuits

Acrylic acid may be released as a decomposition product during the production of integrated circuits or in the galvanic industry, when photoresist materials (e.g. methyl methacrylat) are partially depolymerized by means of UV light. No information could be obtained whether the UV light treatment occurs in closed systems or if the workplaces are equipped with local exhaust ventilation. However, it is assumed that during the UV light treatment only a part of the formed acrylic acid is evaporated, and, unless other information is provided, that inhalation exposure is low.

Because acrylic acid is released during thermal processes, normally no immediate skin contact occurs. The dermal exposure level caused by touching of acrylic acid contaminated surfaces (indirect exposure) is regarded as being low on account of the inhalation exposure levels.

Occupational exposure in the skilled trade

Use of special-purpose adhesives in the skilled trade

Adhesives which contain acrylic acid are used for repair e.g. metal workpieces. It is assumed that rather small areas are bonded and that exhaust ventilation systems are absent, and that suitable personal protective equipment is not worn if non-corrosive adhesives containing < 5% acrylic acid are handled. For the use of adhesives in the skilled trade sector, it has to be taken into account that the overall duration of open handling of adhesives is probably much shorter than the shift duration. Therefore the inhalation exposure level is assumed to be lower than in the comparable industrial sector (without LEV, 30 mg/m^3).

The dermal exposure levels may be in the same order of magnitude or even lower than assessed for the use of corrosive and non-corrosive adhesives in the industrial sector (corrosive adhesives:

low, potential exposure: 0-2.1 mg/person/day; non-corrosive: 1-10.5 mg/person/day). It is to be assumed that these repair activities will not be done daily. Since neither workplace measurements nor information on the duration and frequency of exposure are available, no further statements can be made.

Gas flame removal of acrylic paint

Acrylic acid may be released as a thermal decomposition product when paints are removed from steel or concrete surfaces by means of gas flames (Henricks-Eckerman et al., 1990). The workplace measurements show that the inhalation exposure level under realistic workplace conditions are below the detection limits of 0.01-0.06 ppm (Työterveyslaitos, 1998). Because of the low not detectable exposure level it is assumed that this exposure scenario is only of minor relevance for the assessment of the occupational exposure.

4.1.1.2.6 Summary of exposure data relevant for workplace risk assessment

The following table shows the exposure data of acrylic acid which are relevant for occupational risk assessment.

Table 4.2 Summary of exposure data of acrylic acid which are relevant for occupational risk assessment

Area of production and use	Form of exposure Activity		Duration and frequency	Inhalation exposure		Dermal exposure			
				Exposure level shift average [mg/m³]	Method	Exposure level [mg/cm²/day]	Exposed area [cm²]	Shift average [mg/p/day]	Method
Chemical industry									
Production, further processing	vapour / liquid	filling, transfer, cleaning, maintenance, repair work	shift length, daily	3.0	90th percentile	low	---	low	exp. judg.
			short term, daily	44.4	assumed reasonable worst case				
			single contacts	---	---	0 - 0.1	420 (palms of hands)	0 - 42	EASE
Manufacture of adhesives (1 - 10% acrylic acid)	vapour / liquid		assumed 2 h/ daily	0.375 - 2.25	EASE with LEV	low	---	low	exp. judg.
		cleaning, maintenance, repair work, drumming	single contacts	---	---	0 - 0.1	420 (palms of hands)	0 - 42	EASE
Industrial area									
Manufacture of adhesives (1 - 10% acrylic acid)	vapour / liquid	filling, transfer, cleaning, maintenance, repair work	assumed, 2 h daily	0.375 - 2.25 7.5 -37.5	EASE with LEV without LEV	low	---	low	exp. judg.
			single contacts	---	---	0 - 0.1	420 (palms of hands)	0 - 42	EASE

Table 4.2 continued overleaf

Table 4.2 continued Summary of exposure data of acrylic acid which are relevant for occupational risk assessment

Area of production and use	Form of exposure Activity		Duration and frequency	Inhalation exposure		Dermal exposure			
				Exposure level shift average [mg/m³]	Method	Exposure level [mg/cm²/day]	Exposed area [cm²]	Shift average [mg/p/day]	Method
Use of adhesives: - ≥ 5% acrylic acid (labelled as corrosive)	vapour / liquid	handling, gluing, charging	shift length, daily	1.5 - 9 30 [1)]	EASE with LEV without LEV	low	---	low	exp. judg.
			single contacts	---	---	0 - 0.01	210 (fingers)	0 - 2.1	EASE
- < 5% acrylic acid (not labelled as corrosive)	vapour / liquid	handling, gluing, charging	intermittent / assumed shift length, daily	1.5 - 9 30 [1)]	EASE with LEV without LEV	0.005 - 0.05	210 (fingers)	1 - 11	EASE
Decomposition during production of integrated circuits	vapour		shift length, daily	low [2)]	exp. judg.	low [3)]	---	low [3)]	exp. judg.
Skilled trade									
Use of adhesives: - ≥ 5% acrylic acid (labelled as corrosive)	vapour / liquid	handling, gluing	shorter than shift length, not daily	< 30	exp. judg.	low	---	low	exp. judg.
			single contacts	---	---	0 - 0.01	210 (fingers)	0 - 2.1	EASE
- < 5% acrylic acid (not labelled as corrosive)	vapour / liquid	handling, gluing	shorter than shift length, not daily / intermittent	< 30	exp. judg.	0.005 - 0.05	210 (fingers)	1 - 11	EASE

1) Lower level of the estimated range is assumed to be realistic (expert judgement)
2) Acrylic acid is released as a decomposition product, inhalation exposure is assumed to be low
3) Dermal exposure by touching contaminated surfaces is assumed to be low on account of the inhalation exposure levels

4.1.1.3 Consumer exposure

According to the Swedish Product Register, products containing acrylic acid are used as adhesive or glue and in adhesive substances on the basis of solvents. They are offered in car and motorcycle repair shops for public use. Furthermore, acrylic acid in sealing compounds is also used by consumers.

According to data reported by industry to the BgVV for poison information centres, acrylic acid is used in the Federal Republic of Germany as a component of adhesives (content of acrylic acid up to 8.5%) and as a surface sealing material (content of acrylic acid 6%).

Inhalation exposure

Exposure to UV-hardening adhesives

For the assessment of the inhalation exposure of the consumer, a computer simulation with the aid of the US EPA model SCIES was used showing consumer exposure under different conditions (Appendix B1).

Under conditions of proper use of 1 g UV-hardenable adhesive (content of acrylic acid 6%) 4 times per year for 1 hour each, the consumer using the adhesive is exposed to an average of 0.384 mg/m^3 with a peak value of 0.542 mg/m^3 during the period of use; after use, a peak concentration of 0.448 mg/m^3 is calculated.

It is exclusively the monomer that accounts for the content of acrylic acid in the adhesive.

Dermal exposure

Exposure to sanitary towels, pantyliners and nappy pants

There are no data on the contents of polyacrylates and the weights of the above-mentioned products. Concerning these products, babies may be considered as a worst-case scenario with regard to the duration of the contact, the contact surface and the body weight. As to the assessment of the dermal exposure of babies to acrylic acid from the residual content of monomer acrylic acid in homopolymerisates of acrylic acid which are used as "superabsorbents" in nappy pants, the following data have been submitted by the Industrieverband für Körperpflege- und Waschmittel (industrial association for body care products and detergents/EDANA):

Amounts of acrylic acid in the residual dampness of nappy:

- Daytime: 0.36 µg
- Naptime: 0.43 µg
- Nighttime: 1.08 µg

Under the assumption, that 4 nappies will be used during daytime, a total of acrylic acid of 2.95 µg can be calculated. 20% of acrylic acid is available for absorption (=0.59 µg). The body weight of newborn children is taken as 3.16 ± 0.35 kg, and that of a 1-year-old child 9.74 ± 1.07 kg (mean ± SD). Taken this distribution into consideration, the dermal exposure to acrylic acid will be as follows [*]:

Table 4.3 Dermal exposure of children to acrylic acid

	Dermal Exposure (µg/kg bw)		
	Median	**95% Percentile**	**Maximum**
Newborns	0.18	0.22	0.29
1-year-old children	0.06	0.073	0.102

* The @-RISK-program (Palisade-Corp., New-York) was used for calculations.

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, acrylic acid has not been detailed. Exposure data due to limitations given by the directive are therefore not available.

In comparison with other plastic material (e.g. MMA) the amounts of acrylic acid should be low and therefore may be neglected.

As compared to this, the t-TDI value (temporary tolerable daily intake) is determined as 0.1 mg/kg bw by the Wissenschaftlicher Lebensmittelausschuß (Scientific Committee on Food) of the EU.

Remark

For the assessment of consumer exposure, preferably standard assumptions should be used applicable to typical cases of use; in situations where this is not possible, assumptions will rather be arbitrary. The amounts used per application are based on the data given by the manufacturer. As a rule, an adult of 60 kg bw will be considered as a standard consumer.

On account of the variability of the exposure conditions, individual exposure cannot be defined exactly. Therefore, it would be more appropriate to indicate fields of possible exposure. These have been divided by log ranges (lower range: 1-10, middle range 10-100, upper range 100-1,000).

4.1.1.4 Humans exposed 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. As a worst-case scenario, the maximum intake due to exposure in the vicinity of a point source (generic model) is calculated. This is compared to an average intake due to exposure via the regional background concentration. In Appendix A11, the detailed calculations are presented.

The following input parameters were used:

	Local scenario	Regional scenario
Concentration in grassland soil:	4.69 µg/kg $_{ww}$	0.02 µg/kg $_{ww}$
Concentration in agricultural soil	24.2 µg/kg $_{ww}$	0.02 µg/kg $_{ww}$
Concentration in surface water:	222 µg/l *	0.40 µg/l
Concentration in the atmosphere:	28 µg/m^3	0.002 µg/m^3
Concentration in groundwater:	2.42 µg/l	0.02 µg/l

* The annual average local aquatic concentration of 222 µg/l calculated for a generic production and processing site is chosen as a realistic worst case, because the emission to atmosphere and soil were estimated on the basis of the same scenario. The higher result, i.e. 1.74 mg/l for external wet polymerisation was not used to avoid unreasonable combination of worst-case emissions. Although the concentration in surface water mentioned above is 8 times higher than the chosen one, the overall scenario is still regarded as a worst case.

The resulting total daily dose is:

$$\mathbf{DOSE_{tot} = 50\ \mu g \cdot kg_{bw}^{-1} \cdot d^{-1}\ (local\ scenario)}$$

$$\mathbf{DOSE_{tot} = 15.1\ ng \cdot kg_{bw}^{-1} \cdot d^{-1}\ (regional\ scenario)}$$

The calculated total doses comprise the following routes:

	% of total dose	
Route	**Local**	**Regional**
Drinking water	11.8	74.8
Fish	0.3	2.1
Stem	76.6	19.6
Root	< 0.1	0.7
Meat	< 0.1	<0.1
Milk	< 0.1	<0.1
Air	11.2	2.8

The main route of indirect exposure is the intake via plant stems for the local and via drinking water for the regional scenario.

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

4.1.2.1 Toxicokinetics, metabolism and distribution

Toxicokinetics

Following gavage of an aqueous solution of [^{11}C]-acrylic acid (26 µg/kg bw) to rats, acrylic acid was rapidly (within 1 h) absorbed and excreted, mainly as $^{11}CO_2$. Stomach intubated rats retained 37.0% of the administered radioactivity at 65 min, whereas approximately 60% of the radioactivity had been expired as $^{11}CO_2$. Relative retention of the radioactivity after 65 min was $\geq$ 1% in the liver (2.6%), adipose tissue (1.9%), small intestine (1.5%), kidneys (1.2%), and spleen (1.0%) and was less than <1% in lungs, skin, blood, heart, brain and muscle. Approximately 6% of the administered radioactivity was excreted in the urine (Kutzman et al., 1982).

Rats received single oral doses of [2,3-^{14}C]-acrylic acid (4, 40 or 400 mg/kg bw in a 0.5% aqueous methylcellulose solution). Within 8 hours, 35-60% of the dose was eliminated from the animal, mostly as expired CO_2. After 72 hours, 44-65% of the radioactivity had been eliminated via expired air, while 2.9-4.3% remained in urine, 2.4-3.6% in faeces and 18.9-24.6% in tissues examined (adipose tissue 9-15%, liver 1.7-2.2% and blood 0.8-1.1%) (De Bethizy et al., 1987).

Following single oral administration of 400 mg [2,3-^{14}C]-acrylic acid/kg bw to rats within 3 days 78% of the radiolabel was exhaled as $^{14}CO_2$, while 6.3% was excreted in urine, 1.1% in faeces and 12.8% remained in tissues (4.8% in muscle, 3% in liver, 1.3% in fat, 2% in skin). This excretion pattern was consistent with that of [1-^{14}C]-propionate administered in the same manner (Winter et al., 1992).

Following gavage of an aqueous solution of 400 mg [1-^{14}C]-acrylic acid to rats, acrylic acid was well absorbed and excreted primarily (approximately 80%) as $^{14}CO_2$ within 24 h of administration. Exhalation of volatile compounds was negligible (<0.5% of the dose). Excretion in urine accounted for 5%, excretion in faeces was 9% within 3 days. Tissue concentrations of acrylic acid derived radioactivity were generally low after 3 days, in liver 0.4% of the dose, in muscle 0.4, in skin 0.2, in other tissues below 0.1% (Winter and Sipes, 1993).

Mice and rats, respectively, were treated orally (40 or 150 mg/kg bw) or dermally (10 or 40 mg/kg bw in acetone) with [1-^{14}C]-acrylic acid. Mice rapidly absorbed and metabolised orally administered acrylic acid, with about 80% of the dose exhaled as $^{14}CO_2$ within 24 h. Excretion in urine and faeces accounted for approximately 3% and 1% of the dose, respectively. Elimination of the ^{14}C radiolabel from plasma, liver and kidney was rapid but slower from fat. The disposition of orally administered acrylic acid in rats was similar to the results obtained from mice. After cutaneous application to mice, about 12% of the dose was absorbed, while the remainder was apparently evaporated. Approximately 80% of the absorbed fraction of the dose was metabolised to $^{14}CO_2$ within 24 h. Excretion in urine and faeces each accounted for less than 0.5% of the dose. Elimination of radioactivity from plasma, liver, and kidney was rapid; however, levels in fat were higher at 72 h (0.5% of the higher dose) than at 8 h (0.1% of the higher dose). After cutaneous administration to rats, 19-26% of the dose was absorbed. Disposition of the absorbed fraction of the dose was similar to results found in mice (Black et al., 1995).

Rats were treated dermally with [^{14}C]-labeled acrylic acid (5 mg/kg bw) in either phosphate buffer (pH 6 or 7.4) or acetone for 24 h. The dosing solution (0.1 ml) was spread evenly on the

clipped skin within covered Plexiglas cells (7.3 cm² surface area). The rate of appearance of $^{14}CO_2$ in exhaled breath was used as a measure of the rate of absorption. The absorption rate was dependent on the vehicle and decreased in the order acetone > buffer pH 6 > buffer pH 7.4. Cumulative absorption after 24 h was 22% from acetone, approximately 19% from buffer pH 6, and 9% from buffer pH 7.4 (D'Souza and Francis, 1988).

Percutaneous absorption of acrylic acid was studied following topical administration of 100 µl of a 4% (v/v) solution of [1-^{14}C]-acrylic acid in acetone using a skin-mounted, charcoal-containing trap covered with fixed aluminum discs to ensure complete recovery of the radiolabel. Excretion of acrylic acid-derived radioactivity was determined in urine, faeces and expired air over a period of 3 days. After 3 days 73% of the radioactivity had volatilized from the skin, 6% was detected in the skin. 16% of the applied dose, representing 75% of the absorbed dose, was exhaled as $^{14}CO_2$ within 12 h. Only 0.9% of the applied radioactivity was found in urine, 0.2% in faeces, and 0.4% was in the major tissues after 72 h (Winter and Sipes, 1993).

Rats were nose-exposed to gaseous [^{11}C]-acrylic acid for 1 minute. At 1.5 minutes 18.3% of the delivered dose was retained in the rats. Relatively large amounts of radioactivity were found in the upper respiratory tract. After 65 minutes the radioactivity in the snout was reduced to 8.1% and approximately 60% of the radiolabel had been expired as $^{11}CO_2$. The elimination of $^{11}CO_2$ was biphasic with $t_{1/2}$ of the αphase of 30.6 min. The amount of radioactivity retained in liver, fat and stomach increased markedly between 1.5 and 65 minutes post exposure. The authors postulate that a portion of acrylic acid was ingested after inhalation. Urinary and fecal excretion was estimated to be 15% within 65 minutes (Kutzman et al., 1982).

A hybrid computational fluid dynamics (CFD) and physiologically-based pharmacokinetic (PBPK) dosimetry inhalation model was constructed to estimate the regional tissue dose of acrylic acid in the rat and human nasal cavity, respectively (Frederick et al., 1998). The rodent model uses two olfactory compartments to incorporate both the olfactory epithelium in the projection extending along the dorsal meatus and the ethmoid olfactory region. This model was based on a compartmental rat nasal model of Bush et al., 1998. The human model uses one olfactory compartment since the human nasal cavity lacks a counterpart for the rodent ethmoid olfactory region (Subramaniam et al., 1998). The liquid phase of the model of Bush et al. was modified to include the effect of buffering capacity on the ionization of the acid in the mucus, diffusion of both the ionized form of the acid and the non-ionized species, liquid:air partition coefficients, tissue:blood partition coefficients (Black and Finch, 1995), and metabolism of acrylic acid (Black and Finch, 1995).

CFD simulations provided estimates of the volume of air flowing through various regions of the rat and human nasal cavities, respectively, at inhalation flow rates respective of resting to light activity physiological conditions (rat: 100-500 ml/min, human: 11,400 or 18,900 ml/min, laminar flows). The simulated regional gas phase mass transport coefficients for the rat nasal cavity are 1-2 orders of magnitude higher than those of the human nasal cavity.

A hybrid CFD-PBPK inhalation model was constructed with the aim to evaluate the relationship between inhaled acrylic acid vapour concentration and the tissue concentration in various regions of the nasal cavity of rats and humans, respectively. An explicit effort was made to derive the parameters for rat and human used in the model either from experimental data or from physicochemical principles without "fitting" model parameters (gas phase diffusivity: 0.1 cm^2/sec; air minute volumes: 250 ml/min (rat), 7,500 ml/min (human); blood flow to nasal cavity (human) estimated). The results of the quantitative sensitivity analysis of the model parameters are not available completely. Deposition of vapours in the rat nasal cavity is relatively insensitive to significant variation in the gas phase mass transport coefficients, but the

human CFD-PBPK model was sensitive to variation in air phase and liquid phase parameters (liquid diffusivity, mucus:air partition coefficient). The diffusivity of acrylic acid (ionized and non-ionized) in mucus and epithelium was defined as 0.01 cm^2/h as an adjustable parameter. The mucus:air partition coefficient was defined as 1,780 (saline, pH 2.0; the liquid:air partition coefficient value for saline, pH 7.4, is 3,210).

Unidirectional simulations were conducted with the model at a flow rate of 500 ml/min (rat) to estimate the steady-state tissue concentration in the anterior olfactory epithelium lining the dorsal meatus of the rat nasal cavity over a wide range of acrylic acid vapour concentrations (0 to 25 ppm for one hour). A dose-response of acrylic acid exposures was simulated for an adult resting male rat and an adult resting male human using the appropriate inspiratory flow rate (based on the minute volumes of each species), nasal anatomy, and nasal air flow patterns from CFD simulations. The cyclic flow simulation was conducted for a reference resting rat and human exposed to 2 ppm acrylic acid for 3 min (minute volume 250 ml/min (rat), 7,500 ml /min (human)).

The CFD-PBPK model simulations predict that olfactory epithelium of the human nasal cavity is exposed to 2-3 fold lower tissue concentrations of acrylic acid than the olfactory epithelium of the rodent nasal cavity under either unidirectional flow exposure conditions or cyclic flow conditions. Frederick et al. (1998) are of the opinion that the model predicts olfactory tissue concentrations for acrylic acid that correlate with acute histopathological lesions observed *in vivo* (rats, exposed with 75 ppm acrylic acid for 3 or 6 h in a chamber) and with those observed *in vitro* (rats nasal septa incubated for two hours at 37°C with concentrations from 0.0 to 6.0 mM acrylic acid).

Metabolism

After oral administration of 4, 40, or 400 mg/kg bw [2,3-^{14}C]-acrylic acid in a 0.5% aqueous methylcellulose solution to rats within 72 h 44-65% of the radioactivity had been eliminated via expired air and 2.9-4.3% remained in the urine. The HPLC profile of metabolites observed in the urine of rats indicated two major metabolites. One of the major metabolites co-eluted was 3-hydroxypropionic acid. Radioactivity could not be detected at the retention times corresponding to that of 2,3-epoxypropionic acid or N-acetyl-S-(2-carboxy-2-hydroxyethyl)cysteine. One hour following an oral dose of acrylic acid (4, 40, 400 or 1,000 mg/kg) in rats a significant depletion of NPSH in the glandular stomach was reported at doses above 4 mg/kg. In the forestomach NPSH depletion occurred at a dose of 1,000 mg/kg. No significant effect of acrylic acid on NPSH in the blood or liver was observed (DeBethizy et al., 1987).

Winter et al. (1992) compared the metabolites of acrylic acid and propionic acid using ^{13}C-NMR analysis of the urine of rats after gavage of single doses (400 mg/kg bw). 3-Hydroxypropionic acid, N-acetyl-S-(2-carboxyethyl)cysteine and N-acetyl-S-(2-carboxyethyl)cysteine-S-oxide were identified as metabolites of acrylic acid. No unchanged acrylic acid was detected. In contrast, the spectra of urine from a propionic acid-treated rat revealed only a few minor ^{13}C-enriched signals that were assigned to methylmalonic acid. These metabolites (CO_2 and methylmalonic acid) are consistent with the known major vitamin B_{12}-dependent pathway of propionate metabolism in mammals. An alternative pathway involves β-oxidation. Acrylyl-CoA forms 3-hydroxypropionic acid that can then be oxidized to malonic semialdehyde. Further catabolism yields acetyl-CoA and CO_2. It is conceivable that excretion and detection of the mercapturates are a consequence of the high dose used in this experiment.

Following single doses (40 or 150 mg/kg) of [1-^{14}C]-acrylic acid to rats urinary metabolites and tissues were analyzed by HPLC. A major polar metabolite which could not be identified accounted for approximately 2 to 3% of the dose. A metabolite that coeluted with

3-hydroxypropionic acid was also detected. Small amounts of several other metabolites were detected. Plasma and liver from orally dosed rats were also analyzed for acrylic acid and metabolites by HPLC. One hour after dosing, a metabolite in plasma that co-eluted with 3-hydroxypropionic acid accounted for about 0.5% of the dose after 40 mg/kg bw. This metabolite was also detected in plasma after application of the higher dose. Neither acrylic acid nor metabolites were detected in plasma or liver at times later than 1 h. They were not detected in kidney at any time after administration (Black et al., 1995).

In other experiments, livers from mice dosed by gavage following a similar dosing regime were analyzed for acrylic acid and metabolites by HPLC. Several metabolites of higher polarity than those of acrylic acid including 3-hydroxypropionic acid were detected 1 h after administration, but not at times later than 1 h. Acrylic acid was not detected in livers from mice at any time after cutaneous administration of 40 mg/kg bw. After cutaneous dosing in rats, a peak that coeluted with acrylic acid was detected in urine along with the major metabolite found after oral dosing. A trace amount of another metabolite was detected in urine from the 40 mg/kg bw cutaneous dose group but not after dosing 10 mg/kg bw (Black et al., 1995).

In vitro studies

Hepatic microsomes were prepared using conventional methods from rats and incubations were started by the addition of 10 µl of [2,3-^{14}C]-acrylic acid. No epoxidized metabolites could be detected and the parent compound was recovered from the incubation mixture unchanged (DeBethizy et al., 1987).

In vitro percutaneous absorption studies using excised human cadaver skin have indicated that acrylic acid absorption can vary significantly as a function of the pH value and nature of the vehicle. *In vitro* flux estimated after a 1 mg dose varied 600-fold within the treatments studied and decreased in the order acetone >> buffer pH 6.0 > buffer pH 7.4 (D'Souza and Francis, 1988).

Miller et al. (1981c) have studied the metabolism of acrylic acid in rat tissue homogenates. Acrylic acid did not react with reduced glutathione either in presence or absence of the soluble enzyme fraction. Non-protein sulphydryl concentrations were not appreciably lower in blood after addition of acrylic acid *in vitro*.

The rate of $^{14}CO_2$ formation from [^{14}C]-acrylic acid was measured *in vitro* with preparations from rat liver hepatocytes. Rapid oxidation of acrylic acid to CO_2 was observed. Mitochondria isolated from the liver homogenates were incubated with acrylic acid under the same conditions and yielded higher rates of acrylic acid-oxidation than homogenates. HPLC analysis of the mitochondrial incubation mixtures indicated 3-hydroxypropionic acid as a major metabolite (Finch & Frederick, 1992).

Black et al. (1993) determined the rate of the *in vitro* oxidation of acrylic acid in 13 tissues of mice. The maximal rate of acrylic acid oxidation in kidney, liver and skin was 2,890, 616 and 48 nmol/h/g, respectively. In remaining organs acrylic acid was oxidized at rates less than 40% of the rate in liver. 3-Hydroxypropionic acid was the only metabolite detected by HPLC analysis.

Acrylic acid oxidation rates and blood tissue partition coefficients were studied in slices of rat tissue using [1-^{14}C]-acrylic acid. Acrylic acid oxidation in rat kidney and liver slices was described by saturable kinetics with maximal rates of about 4 and 2 µmol/h/g, respectively. Acrylic acid oxidation rates in 11 additional tissues were 40% or less than that in liver (Black & Finch, 1995).

Conclusion

Acrylic acid is rapidly absorbed in rats and mice after oral or inhalation administration. A hybrid computational fluid dynamics and physiologically-based pharmacokinetics inhalation dosimetry model was constructed for interspecies (rat-human) extrapolation of acrylic acid tissue dose in the olfactory region of the nasal cavity. The model simulations indicate that under similar exposure conditions human olfactory epithelium is exposed with acrylic acid to 2-3 fold lower than rat olfactory epithelium. After dermal administration some acrylic acid is evaporated, the remainder undergoes rapid absorption in these animals. Dermal absorption is strongly dependent on the vehicle and the pH value of the solution.

Acrylic acid is rapidly metabolised by oxidative pathways to CO_2. The main metabolic pathway of acrylic acid seems to be a secondary, non-vitamin-B_{12} dependent pathway of propionic acid metabolism consisting in reactions similar to fatty acid β-oxidation. In urine poorly characterized substances of a higher polarity than those of acrylic acid are detected. Unmetabolised acrylic acid could not be detected in urine, however small amounts of 3-hydroxypropionic acid were found. Epoxide intermediates were not detected. *In vitro* (stomach tissue) and *in vivo* acrylic acid reacts with GSH and NPSH to a very low extent. High dosages of acrylic acid leading to tissue damage cause the formation of small amounts of mercapturic acid derivates.

4.1.2.2 Acute toxicity

Studies in animals

In tests with animals, acrylic acid causes acute harmful effects by the oral and dermal routes of exposure. A review on acute toxicity studies with acrylic acid is given in a publication by Tyler et al., 1993.

The oral LD_{50} values reported for rats cover a range from as low as 140 mg/kg up to 1,400 mg/kg (Dow Chemical 1979a, unpublished report) depending on the concentration of the test substance, similar acute oral toxicity is demonstrated for mice and rabbits (Carpenter et al., 1974). The only clinical signs observed during acute oral toxicity testing with this corrosive substance were a short reflex period of motor excitation followed by lethargy.

More detailed information on lesions caused after oral application of the acid are given within the report on a test with male rats treated with a 10% aqueous solution of acrylic acid (purity 99%, pH of the aqueous solution 2.5) where an oral LD_{50} value of 1,350 mg/kg bw was detected. This aqueous solution caused mortalities within 2 days after treatment (no further data on methodology). In rats of the 700 mg/kg (non-lethal dose), 900 and 1,100 mg/kg dose groups that were killed 48 hours after treatment, histopathology revealed necrosis in the gastric epithels and irritation infiltrates in the gastric mucosa in approximately 50% of animals assessed. In addition, these animals demonstrated acute degeneration of liver parenchyme and in some cases liver necrosis. Animals necropsied 14 days after treatment did not show relevant pathologic changes (Majka et al., 1974).

Acute dermal toxicity is dominated by the severe local corrosivity caused by dermal contact; dermal LD_{50} values of 300 mg/kg (Carpenter et al., 1974) and 640 mg/kg (BASF AG 1979, unpublished report) are demonstrated for rabbits. In the BASF study doses of 400 and 640 mg/kg of undiluted acrylic acid were occlusively applied for 24 hours to the skin of 5 male and 5 female rabbits per dose. After application of 400 mg/kg 1/5 males and 1/5 females died on day 7 or later; after application of 640 mg/kg 2/5 males and 3/5 females died within 24 hours. In addition

to severe local necroses, apathy, laboured respiration and poor general state were observed; necropsy demonstrated dilatated heart and lung oedema.

Data on acute inhalation toxicity of acrylic acid normally demonstrate severe irritation in the respiratory tract but no mortalities. Majka et al. (1974), however, state an inhalation LC_{50} of 3,600 mg/m^3/4 h (3.6 mg/l/4 h) for male rats in a poorly reported study. The animals were exposed to acrylic acid vapours (purity of the acid 99%) in an inhalation chamber of 0.045 m^3 volume (dynamic system with air flow of 100-120 l/hour, no more data on methodology). A LC_{50} of 3,600 mg/m^3/4 h was detected with mortalities occurring within 48 hours after treatment. Histopathology in rats killed 48 hours after treatment revealed in the 2,970 mg/m^3 (non-lethal concentration) and 3,600 mg/m^3 (concentration of the LC_{50} value) groups hyperemia of the inner organs. In the respiratory system severe irritation of the bronchial mucosa, exudate into the bronchial lumen, macrophages in the vesicle lumen and focal intraparenchymal irritation in the lungs was observed. Necropsy at the end of the 14-day observation period demonstrated signs of respiratory irritation.

Carpenter et al. (1974) reported data from vapour inhalation tests with rats using glacial acrylic acid (no data on purity) as test substance within a list of test results for many different chemicals. The following data on inhalation toxicity of acrylic acid are given: Maximum inhalation time of 1 hour caused no deaths in rats inhaling "concentrated vapours" of acrylic acid (no information on this concentration); none of six rats died after a 4-hour inhalation of 2,000 ppm (5.9 mg/l/4h) of the substance (no data on testing methodologies).

In the majority of the other test reports acute inhalation toxicity is stated to be low - supposedly because acrylic acid interacts with humidity of the air prior to reaching the respiratory tract (BAMM, 1988, unpublished report) and causes respiratory irritation instead of acute inhalation toxicity (BASF AG, 1980, unpublished report). The LC_{50} for rats inhaling acrylic acid vapours is reported to be >5.12 mg/l/4 h (BASF AG, 1980, unpublished report). The mentioned acute inhalation toxicity studies were conducted with rats using whole body exposure to "saturated" acrylic acid vapours. No mortalities occurred in these studies and the clinical signs of "respiratory irritation" were perinasal wetness and encrustation and abdominal breathing. No pathological changes were observed at necropsy. In addition, the laboratory staff of the 1988 study judged that the vapours interacted with the relative humidity of the water soluble test material prior to reaching the respiratory tract. Thus, the observed clinical signs of respiratory irritation demonstrate that substance vapours normally will not reach the lungs.

Studies in humans

No data available.

Conclusion on acute toxicity

Data on human experience with acute exposure to acrylic acid are not available. Pure acrylic acid is a very reactive chemical substance and accordingly exhibits severe corrosive properties in contact with biological material. Acute toxicity detected in animal tests consequently is dominated by chemical interactions with water and/or biological material. Thus, acrylic acid causes acute harmful effects by the oral and dermal routes of exposure. The oral LD_{50} values for rats cover a range from as low as 140 mg/kg up to 1,400 mg/kg depending on the concentration of the test substance. An oral LD_{50} of 1,350 mg/kg was detected for male rats in a study with a 10% aqueous solution of acrylic acid - pH of this solution was 2.5 and thus, corrosive effects demonstrated in this study are not caused by the pH of the test substance (Majka et al., 1974); a dermal LD_{50} of approximately 640 mg/kg was detected for rabbits in a study with undiluted

acrylic acid (BASF AG, 1979). Acute inhalation toxicity however is normally stated to be low because acrylic acid interacts with the humidity of the air prior to reaching the depth of the respiratory tract. Despite this an inhalation LC_{50} of 3.6 mg/l/4 hours is detected for male rats in a study by Majka et al. The findings of acute toxicity the respiratory tract seem to depend on the mode of exposure. For classification, see Section 1.4.

4.1.2.3 Irritation/Corrosivity

Studies in animals

Acrylic acid causes severe burns to skin and eyes and severe irritation in the respiratory tract. Reported dose-response assessment for acute toxic effects caused by acrylic acid are based on demonstration of dose dependency of the severe corrosive properties of this chemical:

In a test according to EEC and OECD guidelines, a single topical application of 0.5 ml of 99.8% pure glacial acrylic acid to the intact skin of 5 Albino rabbits for 3 minutes under a semi-occlusive dressing caused in all animals brownish discolouration of the skin within 1 hour after substance application. In 2 animals the finding was assessed after the 1-hour reading by macroscopic pathology indicating superficial necrosis, slight oedema and discolouration of the application area. The brownish discolouration of the remaining 3 animals was assessed after 14 days by histopathology. This examination revealed deep focal necrosis (full thickness necrosis), loss of epidermal adnexa in necrosis area, perifocal moderate epithelial hyperplasia and diffuse inflammatory reaction (corium to subcutis) in the application area (BASF AG, 1998, unpublished report).

After 24 hours of occluded application of 400 mg/kg to the skin of rabbits 2 out of 10 animals died 7 days after exposure or later; after application of 640 mg/kg 5 out of 10 rabbits died within 24 hours (BASF AG, 1979, unpublished report). After 1 minute of exposure to undiluted acrylic acid or of a 50% aqueous substance solution rabbit skin exhibited necrosis; a 10% aqueous solution caused skin irritation after 5 minutes of exposure (BASF AG, 1958, unpublished report).

The serious damage to eyes caused by acrylic acid is not due to the acidic properties of this chemical. In an experiment with acrylic acid neutralized with potassium hydroxide (forming a neutral 60% aqueous solution of potassium acrylate) severe ocular damage was demonstrated for rabbit eyes not flushed with water immediately after instillation of the neutral solution: 0.1 ml of 60% (neutral) aqueous solution of potassium acrylate was instilled into each of the eyes of 9 albino rabbits. The eyes of 6 animals were flushed with lukewarm water. Three animals flushed 2 seconds after instillation and 3 animals flushed 4 seconds after instillation developed corneal opacities that cleared within 7 days. The 3 animals with unwashed eyes demonstrated irreversible severe ocular damage with corneal opacity occurring after 1 hour and persisting for the duration of the study (18 days). In addition, 1 animal was administered 0.1 ml and flushed with water 20 seconds later, and 1 animal was treated with 0.1 ml solution and flushed 4 seconds after instillation for a period of 1 minute; both animals developed irreversible corneal opacity (Hoechst Celanese Corp., 1992, unpublished report).

In a series of poorly reported Draize tests with rabbits the following relationship between eye lesions and concentrations of aqueous solutions of acrylic acid (purity 99%) was detected: The undiluted acid caused severe irritation that reversed within 20 days resulting in irreversible tissue changes such as scarring of the eyelids and corneal opacity. Similar but less pronounced lesions resulted after instillation of a 10% aqueous solution. A 3% solution caused irritation that

reversed within 6 days, lesions caused by a 1% aqueous solution disappeared 2 days after instillation of the test substance. No more data are reported (Majka et al., 1974).

All studies conducted in order to assess acute inhalation toxicity of acrylic acid demonstrate severe irritation in the respiratory tract. Majka et al. exposed rabbits to acrylic acid vapours (purity of the acid 99%) in an inhalation chamber of 0.045 m^3 volume (dynamic system with air flow of 100-120 l/hour, no more data on methodology). A LC_{50} of 3,600 mg/m^3/4 h was detected with mortalities occurring within 48 hours after treatment. Histopathology in rats killed 48 hours after treatment revealed in the 2,970 mg/m^3 (non-lethal concentration) and 3,600 mg/m^3 (concentration of the LC_{50} value) groups hyperemia of the inner organs. In the respiratory system severe irritation of the bronchial mucosa, exudate into the bronchial lumen, macrophages in the vesicle lumen and focal intraparenchymal irritation in the lungs was observed. Necropsy at the end of the 14-day observation period demonstrated signs of respiratory irritation (Majka et al., 1974). For further information see Section 4.1.2.2 on Acute toxicity of acrylic acid.

Studies in humans

Human data are submitted by industry mentioning that 3 accidents with acrylic acid occurred within the time period of 1967-1992: Two workers needed hospitalisation because of skin corrosion and 1 worker because of irritation of the respiratory tract (BASF AG, 1992, unpublished information).

Conclusion on irritation and corrosivity

Data on accidents at the workplace demonstrate that acrylic acid causes skin corrosion and irritation of the respiratory tract in humans. In tests with rabbits pure acrylic acid caused severe burns to skin and eyes; a 50% aqueous substance solution caused necrosis to rabbit skin after 1 minute of exposure, even a 10% aqueous solution caused skin irritation within 5 minutes. Severe ocular damage caused by acrylic acid cannot be avoided by neutralizing the acid. For classification, see Section 1.

4.1.2.4 Corrosivity

See Section 4.1.2.3

4.1.2.5 Sensitisation

Studies in animals

Eight acrylates and methacrylates including acrylic acid, hydroxyethyl acrylate, hydroxypropyl acrylate, aminoethyl methacrylate hydrochloride, Na-2-sulphoethylmethacrylate, 2-sulphoethyl methacrylate, hydroxyethyl methacrylate, hydroxypropyl methacrylate and an additional 64 substances were tested in guinea pigs with the use of a modified Split Adjuvant Test. The purity of the test substances is not mentioned. The highest concentration which did not cause primary irritation was used but no data are given on the test concentrations. Ten animals per test received a 0.1 ml aliquot of the test material to the backs four times in 10 days. At the time of the third application, 0.2 ml Freund´s adjuvant was injected at one point adjacent to the insult site. After a 2-week rest period, the guinea pigs were challenged with the test material on one flank and a solvent (if used) on the other flank. The challenge site was evaluated for erythema and oedema at

24 and 48 hours. Acrylic acid and six other acrylates and methacrylates were negative (0/10) and only aminoethylmethacrylate hydrochloride was positive in 1/10 animals (Rao et al., 1981).

In a modified Freund´s Complete Adjuvant test 8 guinea pigs/group received 3 intradermal injections during the induction phase on days 0, 5 and 9 (the test substance was mixed with FCA in a volume of 0.1 ml). Non-irritant test concentrations were used for challenge at day 21. The test concentrations for intradermal injections were 1.2% and for challenge 7.2% in Aramek, a mixture of 2 parts methyl ethyl ketone and 1 part of peanut oil. Distilled acrylic acid was negative but commercial acrylic acid is a strong skin sensitizer. The skin reactions were due to the presence of varying quantities (up to 7%) of α,β-diacryloxypropionic acid (DAPA). Positive skin reaction was still present after a third challenge on day 49 (Waegemakers and van der Walle, 1984).

Recent investigations on the occurrence of DAPA in industrial acrylic acid have shown that DAPA is not present (at a detection limit of 20 ppm) in current commercial samples of acrylic acid (Elf Atochem, 1998).

The stabilizer added to the trade product (hydroquinone monomethyether) is a known skin sensitizer (EHC 191), however the concentration added (0.02% w/w) is too low to propose labeling.

There is no information available on the potential of acrylic acid to produce respiratory sensitisation in animals.

Studies in humans

Human data are available showing the severe local corrosive properties of acrylic acid. Exposed persons can exhibit contact dermatitis. One worker showed a positive reaction in a Patch test with acrylic acid but not with acrylic resin compounds (Fowler, 1990). One woman was patch tested with individual components of Fixomull® tape adhesive. A positive response to acrylic acid, a component of the tape, was demonstrated (Daecke et al., 1993). Negative results in six workers patch tested with 0.1% acrylic acid in petrolatum are also reported (Conde-Salazar et al., 1988). However, data on the purity of the acrylic acid products were not given by the different authors. Since 1989 more than 450 workers in production plants using acrylic acid as a base material have been regularly medically examined. No cases of sensitisation to acrylic acid have been observed (information by BASF AG, 1998).

Respiratory sensitisation has not been observed.

Conclusion on sensitisation

Pure acrylic acid does not show skin sensitising properties in animal sensitisation tests. But skin sensitisation was observed in humans. This might be caused by an impurity of acrylic acid, DAPA. However, data on the impurity in the acrylic acid samples tested in humans were not available. Recent investigations on the occurrence of DAPA in industrial acrylic acid have shown that DAPA is not present (at a detection limit of 20 ppm) in current commercial samples of acrylic acid. Medical examinations performed with more than 450 workers in production plants using acrylic acid as a base material demonstrated that since 1989 commercial acrylic acid and present occupational health protection precautions are sufficient to avoid sensitisation hazard at the workplace.

Respiratory sensitisation has not been observed in humans.

4.1.2.6 Repeated dose toxicity

Studies in animals

Several studies investigating the effects of acrylic acid after repeated oral and inhalation exposures of rats and mice were regarded as valid according to the requirements of the directive 793/93/EEC.

Oral administration

In an oral 90-day study (BASF AG, 1987; Hellwig et al., 1993) acrylic acid (approx. 99%) was administered by gavage in two dosages (150 and 375 mg/kg bw/d) to Wistar rats.

The clinical examinations revealed vocalization beginning after the first week of treatment and tympanies of the gastrointestinal tract, which was frequently connected with cyanosis and dyspnoea, in most of the animals from week 3 onwards. 6 males and 9 females of the high-dose group and 5 males and 5 females of the low-dose group died prematurely (day 14-81 of the treatment) showing apathy, hypothermia and piloerection before death. Dose-related severe toxic effects were recorded in both dose groups consisting of reduced body weight gain, thickening of the plica marginata and hyperaemia or erosions/ulcerations of the gastric mucosa. Degeneration/necrosis of renal tubules were observed in the five males in each of both dose groups and four and seven females of the low and high dose groups, respectively, that died during the study. The testing parameters did not include hematology, clinical chemistry, urinalysis; histopathological examination was done on selected organs of the gastrointestinal tract, liver, kidney, urinary bladder, adrenals, tongue, buccal and nasal mucosa. The LOAEL of this study was 150 mg/kg, no NOAEL was derived.

Wistar rats which received acrylic acid in the drinking water at doses of 120, 800, 2,000 or 5,000 ppm (approx. 6, 40, 100 or 200 mg/kg bw/d in males, and 10, 66, 150 or 375 mg/kg bw/d in females) for 3 months (10 rats/group/sex) and 12 months (20 rats/group/sex) showed reduced water consumption at 2,000 ppm and 5,000 ppm dosages (BASF AG, 1987; Hellwig et al., 1993). No treatment-related premature deaths occurred. Lower food consumption was seen in high dose males and reduced body weight gain was observed in males from 2,000 ppm and 5,000 ppm groups. Although there were transient decreases of red cell counts and hematocrit values and increased MCH and MCHC values for high dose males at week 12 only, no treatment-related effect on hematology, clinical chemistry, and urine parameters were found. No substance-related toxic effect could be microscopically demonstrated in a comprehensive list of organs examined in the two high doses. Obviously due to the bad palatability treated animals had lower drinking water uptake, which was considered to result in lower food consumption and body weight gain. Mortality and toxic effects in the kidney and the stomach found in the gavage study were not confirmed in this study at comparable doses indicating that these effects were attributable to high local and blood peak values after bolus administration by gavage. Based on the reduced body weight gain in males and lower water consumption in both sexes, the NOAEL of this study was considered to be 800 ppm (40 mg/kg). In females the NOAEL was considered to be 5,000 ppm (331 mg/kg) because reduced water consumption was not interpreted as a clear adverse health effect.

Fischer 344 rats (15 animals/sex/group) in another 90-day drinking water study (Bushy Run Research Center, Inter-Company Acrylate Study Group, 1980) were administered doses of 83, 250, or 750 mg/kg bw/d of acrylic acid. No deaths occurred during the treatment period. However, clear dose-related effects were observed.

At the high-dose level there was reduced food and water consumption, reduced body weight gain, lower organ weights of liver, kidneys, spleen, heart, brain, and elevated testes weight and some altered clinical chemistry parameters (increased levels of serum urea nitrogen, glucose, alkaline phosphatase and aspartate transaminase). Furthermore, there was a statistically significant decrease in total serum cholesterol noted for the high level females. In both sexes increases of urinary protein and specific gravity and a decrease in urinary pH values were noted. No significant prevalence of microscopic lesions was found in any of the animals.

At 250 mg/kg bw, a decrease in water consumption was noted for both sexes. Body weight gain was lower in females. Kidney weights were increased in both sexes and relative testes weights were increased in males. Effects on serum urea nitrogen, cholesterol and alkaline phosphatase in female rats and urinary specific gravity and protein in both sexes were similar, but less pronounced than those observed at the high dose level.

At 83 mg/kg bw the only effects noted were a reduction of water consumption by male rats and a slight increase in red blood cells in female rats. Both findings were not considered to be of toxicological relevance, therefore 83 mg/kg was the NOAEL of this study.

Inhalation route

A 90-day inhalation study on Fischer 344 rats and B6C3F1 mice (Miller et al., 1981a, 1981b; Dow Chemical Company, 1979b) using 15 animals/sex/group exposed to doses of 5, 25 or 75 ppm (approx. 0.015, 0.074 or 0.221 mg/l) of acrylic acid vapour on 6 hours/day on 5 days/week revealed lower mean body weight gains in female mice from 25 ppm and 75 ppm dose groups. Histopathological examinations were performed on tissues of 10 animals/sex/group.

Male mice of the 25 ppm and 75 ppm groups and in female mice of the 75 ppm group had a slight decrease of the mean hemoglobin concentration without further corroborative alterations of the hematological parameters. There were no relevant treatment-related effects on organ weights, hematological parameters, clinical chemistry parameters or urinary parameters.

Acrylic acid vapour induced slight focal degeneration of the nasal olfactory epithelium in 7/10 male and 10/10 female rats at the 75 ppm dose. No lesions of the nasal mucosa were observed in the mid and low dose groups.

Similar dose-related lesions on the nasal mucosa were demonstrated in all groups of treated mice which were examined microscopically. Slight to moderate focal degeneration of the olfactory epithelium was observed in 10/10 males and 11/11 females of the high dose group, 11/11 males and 9/10 females of the mid group and 1/10 males and 4/12 females of the low dose group. Nasal lesions in high dose animals showing degenerated olfactory epithelium were corroborated by focal replacement of the olfactory epithelium by a lower columnar-type epithelium which resembled respiratory-type epithelium. There also appeared to be very slight focal hyperplasia of the submucosa glands and infiltrations of inflammatory cells in the mucosa and submucosa in all animals affected. Areas normally lined by respiratory epithelium appeared to be totally unaffected. In the mid-dose group, 1/11 male and 2/10 females had also focal infiltration of inflammatory cells. Similar lesions were not observed in any mice of the control groups.

In this study, histopathology of four cross-sections were examined in 10 animals/sex/group at different levels of the nasal turbinates, being the target organ identified.

For local effects, this study revealed a NOAEC of 25 ppm for rats. No local NOAEC was derived in mice, the LOAEC (local) is 5 ppm. There was no systemic toxicity in rats and male

mice and systemic NOAEC was therefore 75 ppm. Because of lower body weight gain, the NOAEC for female mice was 5 ppm.

Other studies which were not in full compliance to the minimal requirements of OECD/EEC guidelines for 28-day studies on repeated dose toxicity are described hereafter to give further information on the substance.

In a range-finding study acrylic acid vapour was inhaled by Fischer 344 rats and B6C3F1 mice (5 animals/sex/dose) during 2 weeks (6 h/d, 5 d/week) at doses of 25, 75, and 225 ppm (approx. 0.074, 0.221 or 0.662 mg/l) (Miller et al., 1979). The high dosage resulted in clinical signs as nose scratching and reduced body weight gains in rats and mice of both sexes as well as in reduced fat depots in female rats. Dose-related inflammatory and degenerative lesions of the nasal mucosa were recorded in all rats and mice of the high dose groups, in all mice of the mid dose group and in 2/5 male mice and 4/5 female mice of the low dose group. All male and female rats in the 225 ppm group had some areas with focal squamous metaplasia of the nasal tissue.

Inhalation by 15 female B6C3F1 mice of 5 or 25 ppm acrylic acid for a period of 15 days (4.4, 6, or 22 h/d) resulted in disorganization and atrophy of the olfactory epithelium, basal cell hypertrophy with squamous differentiation, epithelial necrosis with desquamation, and Bowmans' gland degeneration (Lomax et al., 1994, Rohm and Haas, 1994). After a six-week recovery period of 5 females/group, animals which were exposed 22 h/d to 25 ppm acrylic acid vapour exhibited regions of respiratory metaplasia.

Following repeated inhalation of acrylic acid vapour 6 h/d in four male and four female rats/group toxic effects were briefly reported (Gage, 1970). Four male and four female rats exposed for 4 days to 1,500 ppm acrylic acid vapour showed nasal discharge, lethargy, weight loss and congested kidneys. Inhalation of 300 ppm acrylic acid vapour for 20 days resulted in nasal irritation, lethargy and reduced body weight gain. Vapour concentration of 80 ppm induced no sign of toxicity.

Respiratory function (respiratory minute volumes (-23% in rats, -27-34% in mice) and respiratory rates (-17% in rats, -32-37% in mice) were slightly depressed after inhalation of 75 ppm acrylic acid vapour by male rats and male mice for 5 days (6 h/d) (Barrow, 1986). Assuming an even distribution of acrylic acid, the dose expressed as acrylic acid concentration (µl/litre)/minute volume (l/min)/nasal cavity surface area (cm²) was nearly twice in mice (3.5-3.8 µl/min/cm²) than in rats (1.8-2.1µl/min/cm²). Histopathology of sections from four levels of the nasal cavity was characterised by severe lesions in both species. Mice had more severe lesions, as seen by the presence of more cellular exudate in the lumen of the nose and a much greater loss of sensory cells. Under the assumption of an evenly distributed chemical the damage of the tissue should be distributed regularly throughout the nasal cavity. However, the principal location of the lesions was on the dorsal meatus of level 3, where epithelial cell counts indicated a 50 percent decrease in mice versus 15 percent in rats. Cell proliferation studies showed a statistically significant increase in cell turnover of the olfactory epithelium in the dorsal meatus of both species. Turnover rate was 4 percent in treated rats (versus 0.9 percent in controls) and 1.7 percent in treated mice (versus 0.1 percent in controls).

Dermal route

Skin irritation become prevalent with higher incidence and severity in mice treated with 4% acrylic acid compared to 1% acrylic acid or vehicle control (acetone) after 13 weeks of dermal application (3 d/wk) (Basic Acrylate Monomer Manufacturers, 1991; Tegeris et al., 1988). No irritant effect was evident after long-term application of 1% acrylic acid in acetone in mice (Inter-Company Acrylate Study Group, 1982, see Section 4.1.2.8).

Studies in humans

No data available.

Effect levels used for the risk characterization

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

Oral administration: 40 mg/kg bw/d (90 d, male rats)
(BASF AG, 1987; Hellwig et al., 1993)

83 mg/kg bw/d (90 d, female rats)
(Bushy Run Research Center, Inter-Company Acrylate Study Group, 1980)

Inhalation exposure: LOAEC for local effects
5 ppm (resp. 0.015 mg/l), 90 d, mice
(Miller et al., 1981a, 1981b; Dow Chemical Company, 1979b)

Inhalation exposure: NOAEC for systemic effects

5 ppm (0.015 mg/l) (90 d, female mice)
75 ppm (0.221 mg/l) (90 d, male and female rats, male mice)
(Miller et al., 1981a, 1981b; Dow Chemical Company, 1979b)

Conclusion on repeated dose toxicity

Overall, the toxic profile of acrylic acid is dominated by its local irritation effects irrespective of the way of application. Prolonged inhalation of concentrations from 5 ppm or higher in mice and 75 ppm in rats induced degeneration of the olfactory mucosa. It causes severe mucosal damage to the stomach after repeated gavage administration of $\geq$150 mg/kg bw/d, but not after application via drinking water at similar or higher doses. Long-term exposure of the skin to acrylic acid at a concentration of >1% resulted in irritation whereas no effect on the skin was evident at 1% (see also dermal carcinogenicity studies in Section 4.1.2.8). Following oral, dermal or inhalation administrations no other systemic toxic effects were detected except premature deaths and tubular degeneration/necrosis in the kidneys which were evident after gavage administration of dosages >150 mg/kg bw/d in a rat 3-month study. Effects were attributed to the high peak concentrations and did not occur in drinking water studies at similar or higher doses. Some studies with repeated application revealed minimal changes of single red blood cell parameters, however no clear hematotoxic effect was found. Changes in clinical chemistry parameters, observed in drinking water studies, were assumed to be associated with reduced consumption of water and/or food.

4.1.2.7 Mutagenicity

Bacterial systems

A bacterial mutation test with Salmonella typhimurium tester strains TA98, TA100, TA1535 and TA1537 was negative in concentrations up to 5,000 µg/plate with and without S-9 mix. Doses from 1,000 µg/plate upwards induced toxic effects (Cameron et al., 1991). Negative results were also found in further bacterial mutation studies (Zeiger et al., 1987; BASF, 1977).

In vitro systems with mammalian cells

A mammalian cell gene mutation test with CHO cells (HPRT locus) was negative at doses up to 1.9 µl/ml (2,000 µg/ml) without S-9 mix and up to 2.4 µl/ml (2,500 µg/ml) with S-9 mix. Survival at the highest concentrations was 35% and 24%, respectively (McCarthy et al., 1992).

In the mouse lymphoma assay increases of mutation frequencies were found in two independent investigations. Cameron et al. (1991) reported on a positive mouse lymphoma assay with and without metabolic activation: without S-9 mix 3-fold to 6-fold dose-related increases of mutant frequencies were induced in the dose range 2.65 to 5.44 mmol/l (191 to 392 µg/ml); with S-9 mix 3-fold to 8-fold increases were found in the dose range 16.2 to 26.5 mmol/l (1,167 to 1,910 µg/ml). The relative total growth at the highest concentrations was 15% and 20%, respectively. Another mouse lymphoma assay was run only without metabolic activation: in the dose range 300 to 600 µg/ml 4-fold to 8-fold increases of mutant colonies were induced in a dose-related and reproducible manner; the survival was about 20% at the highest concentration (Moore et al., 1988). In both studies the majority of the mutants gave small colonies.

Positive effects were described also for *in vitro* chromosomal aberration assays. McCarthy et al. (1992) reported a positive chromosomal aberration assay in CHO cells with and without metabolic activation. Without S-9 mix, doses of 3.8 and 5.0 µl/ml (3,942 and 5,230 µg/ml) induced 11% and 30% aberrant cells (2% in the negative control); with S-9 mix, in the dose range 1.6 to 2.8 µl/ml (1,689 to 2,977 µg/ml) aberration frequencies of 9% to 28% were found in a dose-related manner (1% in the negative control). The positive response was not bound to drastic cytotoxic effects (42% and 35% relative cloning efficiency at the highest concentrations) or to decreases in pH (pH was adjusted to pH 7.0). A chromosomal aberration assay with L5178Y mouse lymphoma cells was only run without S-9 mix. In the dose range 300-500 µg/ml 7%-21% aberrant cells were induced in a dose-related manner (4% in the control culture); the effect was not related to drastic cytotoxicity (Moore et al., 1988). Furthermore, Ishidate (1988) reported that acrylic acid induced chromosomal aberrations in CHL cells without S-9 mix at the highest dose tested of 750 µg/ml; cytotoxicity data are not given.

In primary rat hepatocytes acrylic acid did not induce unscheduled DNA synthesis (UDS) in non-toxic doses up to 0.4 µl/ml (420 µg/ml). Higher doses could not be analysed due to high cytotoxicity (McCarthy et al., 1992).

Wiegand et al. (1989) investigated induction of micronuclei, UDS and cell transformation in tertiary cultures of Syrian hamster embryo (SHE) cells without addition of an external metabolising system. Results, although negative for all three endpoints, are of low reliability due to the screening character of the test, poor description and methodological insufficiencies (e.g., in the micronucleus test only doses in the non-toxic range were analyzed; there is confusion whether diethylstilbestrol or B(a)P was used as positive control, concentrations were not given, nor the percentage of micronucleated cells).

Drosophila test

A test with Drosophila melanogaster for induction of sex-linked recessive lethal mutations was negative after administration via feeding or injection (McCarthy et al., 1992).

In vivo systems with mammals

Two *in vivo* bone marrow chromosomal aberration assays with rats gave negative results (McCarthy et al., 1992). Chromosome aberrations were analyzed (5 animals per sex, 50 metaphases per animal) at 6, 12, and 24 h after oral doses of 100, 333 or 1,000 mg/kg or after exposure to 2,000 or 5,000 ppm acrylic acid in drinking water for 5 days. In the acute as well as in the repeated dosing regimens the highest doses led to reduced body weight gains. Mitotic activity of bone marrow cells was not influenced by the treatments.

A dominant lethal assay led to negative results. Male mice were given single oral doses (gavage) up to 324 mg/kg or five daily oral doses up to 162 mg/kg. Immediately after dose administration male mice were mated. Females were checked for vaginal plugs each morning. Each mated female was replaced with a virgin female. The mating process was continued for 46 days. An analysis of the uterine contents of female mice was made 12-15 days after observation of the vaginal plug (McCarthy et al., 1992).

Mutagenicity data for structurally-related acrylic compounds

Negative results from *in vivo* bone marrow tests (micronucleus or chromosomal aberration assays) were reported for several structurally-related acrylic compounds:

- methyl methacrylate (Hachiya et al., 1982);
- ethyl acrylate (Ashby et al., 1989; Morita et al., 1997);
- methyl acrylate (Fh-ITA, 1994);
- butyl acrylate (Engelhardt and Klimisch, 1983).

Positive micronucleus assays were also reported, however, they seem to be of low reliability (Przybojewska et al., 1984; methodological insufficiencies) (Kligerman et al., 1991; weak effect in mouse splenocytes for doses in the toxic range paralleled by a negative in bone marrow chromosomal aberration test).

Conclusion on mutagenicity

Acrylic acid did not induce gene mutations in Salmonella or CHO cells (HPRT locus) but was clearly positive in the mouse lymphoma assay and in the *in vitro* chromosomal aberration test. Since in the mouse lymphoma assay small colonies were induced preferentially, the mutagenic potential of acrylic acid seems to be limited to clastogenicity. *In vivo*, acrylic acid did not induce mutagenic effects in either rat bone marrow cells or mouse germ cells after oral administration. Based on the present results and taking into account data on structurally-related acrylic compounds, it is unlikely that acrylic acid is mutagenic *in vivo*.

4.1.2.8 Carcinogenicity

Studies in animals

In a valid carcinogenicity study (BASF AG, 1989; Hellwig et al., 1993) Wistar rats were exposed to doses of 120, 400 or 1,200 ppm (mean substance uptake 9, 31, or 88 mg/kg bw/d) acrylic acid (99%, stabilized with 200 ppm hydroquinone monomethylether) in drinking water for 26 months (males) or 28 months (females). Except a slightly reduced water consumption in 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 4.4**).

Studies which did not fulfil the requirements of guideline testing protocols for regulatory purposes give additional information on acrylic acid (see also **Table 4.5**):

In a dermal carcinogenicity study no tumours of the skin or subcutis were induced in treated mice or in the vehicle controls (Intercompany Acrylate Study Group, 1982). 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 lifetime. Histological 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 showed 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 unusual gross lesion. No treatment-related signs of skin irritation, toxicity, clinical signs or skin tumors 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, 1992a), 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.

After subcutaneous administration of 1.4 mg acrylic acid in 0.5 ml trioctanion in 2/30 female Hsd-(ICR)Br mice two sarcomas were observed at the application site after 49.5 weeks (once weekly) (Segal et al., 1987). Malignancies were not observed in 20 mice receiving solvent (trioctanion) alone and in 100 untreated mice. These results were not considered to be relevant because of the application route (Grasso, 1987).

Studies in humans

No information on potential human carcinogenicity is available.

Conclusion on carcinogenicity

There is no evidence that acrylic acid administered orally to rats or applied dermally to mice is carcinogenic. There are no cancer data available with respect to human exposure.

Table 4.4 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, 1200 ppm AA in drinking water	Ø	no	yes	BASF (1989)

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

Table 4.5 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 40 males	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	100 µl 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

Fertility

Possible effects on reproductive performance were investigated by oral administration (via drinking water) in two different studies with rats.

In a one-generation study with F334/N rats (DePass et al., 1983), the animals (10 males and 20 females per dose group) received acrylic acid at dose levels corresponding to 0, 83, 250 or 750 mg/kg bw/d for 13 weeks. Each male was then mated with 2 females and exposure continued for both sexes throughout gestation and lactation. Dose-related reductions in food and water consumption and consequently in body weight gain were observed in the F_0 animals, most pronounced and statistically significant at the 750 mg/kg bw/d dose level. At the high-dose level the fertility index of males and females, the gestation index, the number of pups born alive and the percentage of pups weaned were clearly reduced. In the high-dose group, pups of both sexes showed decreased body weight gain, also in males a reduction in absolute and relative liver weights and in females a reduction in both absolute and relative spleen weights was observed. Nevertheless, these findings were not considered as an indication of any substantial deleterious effect of acrylic acid on reproductive performance, because there were no statistically significant differences among the treated and the control groups. However, the fertility index and litter size of the control group of this study were atypically low.

In a two-generation study (OECD 416) acrylic acid was administered orally (in drinking water) to Wistar rats at doses of 0, 500, 2,500, 5,000 mg/l (53, 240, 460 mg/kg bw/d). The following results were observed (BASF, 1994c; Hellwig et al., 1997): in the male F_0 parental generation there were no signs of general toxicity. In the female parental generation reduction of food and drinking water consumption were observed at 5000 mg/l during the period of pregnancy. Dose dependent reduction of food and drinking water consumption during the lactation period were observed at 2,500 mg/l.

In the F_1 generation in both sexes dose-dependent reduction of food and drinking water consumption at 2,500 mg/l were observed. Body weight and body weight gain in both sexes were reduced. But in both sexes of the F_0 and F_1 generation there were no abnormal clinical signs. No adverse effects on fertility and pre-implantation development could be detected; no effects on reproductive organs have been observed. The mating index of males in both generations and in all dose groups was 100%.

The fertility rate in the F_0 generation was between 92-96%; in the F_1 generation in all dose groups the fertility rate was 100%. The rate of pregnancy in both generations was not reduced. In both generations there were no differences in numbers of pups born alive.

NOAEL for reproductive function was 460 mg/kg bw/d.

NOAEL for general toxicity was 240 mg/kg bw/d for the F_0 generation, but 53 mg/kg bw/d for the F_1 generation.

Developmental Toxicity

Oral

Developmental studies with the oral route of administration are not available. However, in the above-mentioned two reproductive toxicity studies in rats acrylic acid (in drinking water) produced some signs of postnatal developmental toxicity in offspring, predominantly decreased

body weight gain, following the exposure of the parental generation at a dose level leading to reduced food and water intake and to reduced body weight gain in the F_1 animals. No gross abnormalities were observed in the offspring in either study (DePass et al., 1983; BASF, 1994 c; Hellwig et al., 1997).

NOAEL (offspring): 53 mg/kg bw/d.

Inhalation

Groups of 30 pregnant Sprague-Dawley rats were exposed (6 h/d, whole-body) to atmospheres containing acrylic acid at 0, 40, 120, and 360 ppm (= 0, 120, 350 and 1,060 mg/m^3) during days 6 to 15 of gestation. After exposure the dams were observed up to day 20 of gestation (Klimisch and Hellwig, 1991). The animals' body weight and food consumption were determined on gestation day 0 and subsequently on every third day up to gestation day 20. After sacrifice dams were subjected to a gross pathological examination. After external examination of each foetus their body weights and lengths were measured and they were further processed for skeletal and visceral examination. In the dams, irritation of the respiratory tract and the eyes was observed in the highest dose group. A dose-related reduction in food and water intake resulting in a decrease in body weight gain was observed in the 120 and 360 ppm groups. Also in the 40 ppm group a slight but statistically significant effect was seen on body weight gain of the dams. A NOAEL for maternal toxicity could therefore not be derived from this study. But no effects on reproductive performances were observed. There were no signs of group-related trends or significant differences between groups in terms of pre-implantation losses, live foetuses, or resorptions. There were also no signs of group-related differences in the incidences of abnormalities, variations, or retardations in the foetuses in terms of general appearance, foetal body weights and the conditions of the internal organs or the skeleton.

Groups of 16 pregnant New Zealand rabbits were exposed (6 h/d, whole-body) to atmospheres containing acrylic acid at 0, 25, 75, and 225 ppm during days 6-18 of gestation (Bushy Run Research Center, 1993; Neeper-Bradley et al., 1997). All doses were observed daily for morbidity and mortality. During the exposure period, animals were observed for clinical signs preceding and subsequent to daily exposures and from outside during actual exposures. Maternal body weights were measured on gestation day 0, 3, 6, 12, 24, and 29. Food consumption was measured daily throughout the study beginning on gestation day 3. After sacrifice on gestation day 29, maternal liver and kidney weights were determined. All foetuses were weighed and examined for external malformations and variations, for thoracic and abdominal visceral abnormalities including internal sex organs, for craniofacial abnormalities and for skeletal malformations and variations. Dose-related clinical signs (as perinasal/perioral wetness and nasal congestion, as well as reduced body weight gain and food consumption) were observed in the 75 and 225 ppm groups. The overall pregnancy rate was equivalent for all groups (94-100%). No dose-related effects were observed in the reproduction function of the dams. There were no effects on the number of ovarian corpora lutea, the number of total viable or non-viable (early and late resorptions and dead foetuses) implantations/litter. Percentage live foetuses and sex ratio were equivalent across groups. Foetal body weights were unaffected by test substance exposure. There were no exposure-related increases in the incidences of external, visceral or skeletal malformations or variations.

NOAEL for maternal toxicity was 25 ppm (Bushy Run Research Center, 1993; Neeper-Bradley et al., 1997).

Inhalation exposure of pregnant rats and rabbits to atmospheres containing acrylic acid at concentrations up to 360 ppm (rats) and 225 ppm (rabbits) produced no evidence of developmental toxicity in either species.

NOAEL (rats): 360 ppm = 1,060 mg/m^3

NOAEL (rabbits): 225 ppm = 663 mg/m^3

(BASF, 1983; Klimisch and Hellwig, 1991; Bushy Run Research Center, 1993; Neeper-Bradley et al., 1997).

Human data: not available.

Conclusion on toxicity for reproduction

In oral reproductive toxicity studies (rats) no effects on reproductive function (fertility) were observed. Some signs of postnatal developmental toxicity (retarded body weight gain of the pups) were seen following exposure of the parental generation, however at dose levels that led to reduced food intake and weight gain in the dams. No gross abnormalities were observed in the offspring. A NOAEL/fertility of 460 mg/kg bw/d was derived from a guideline 2-generation study in rats (BASF, 1994c; Hellwig et al., 1997). No prenatal developmental toxicity was observed (rats and rabbits, inhalation), even at concentration levels that produced some signs of maternal toxicity. No specific teratogenic potential could be revealed for dose levels up to and including 360 ppm (rats), resp. 225 ppm (rabbits). A NOAEL/developmental toxicity of 225 ppm (according to 663 mg/m^3) was derived from the developmental toxicity study in rabbits (Bushy Run Research Center, 1993; Neeper-Bradley et al., 1997). According to the present database for toxicity for reproduction there are no reasons to classify acrylic acid as a reproductive toxicant.

4.1.3 Risk characterisation

4.1.3.1 General aspects

Acrylic acid is absorbed via the lungs in animals and humans, absorption via the oral and dermal routes of exposure is demonstrated. In animals with solely nasal respiration, it is resorbed at the nasal mucosa. A hybrid computational fluid dynamics and physiologically-based pharmacokinetics inhalation dosimetry model was constructed for interspecies (rat-human) extrapolation of acrylic acid tissue dose in the olfactory region of the nasal cavity. The model simulations indicate that under similar exposure conditions human olfactory epithelium is exposed with acrylic acid to 2-3 fold lower than rat olfactory epithelium. After dermal administration some acrylic acid is evaporated; the remainder undergoes rapid absorption. The extent of absorption depends on pH and solvent with direct dependency on substance concentration. Mouse skin shows better permeability than human skin. Whole body distribution was observed. In mice acrylic acid is rapidly and completely metabolised mainly in liver and kidney by the normal catabolic pathways by beta-oxidation of fatty acids and in the citrate cycle**.** Elimination preferably occurs as carbon dioxide (exhalation). Small amounts of 3-hydroxypropionic acid but no unmetabolised acrylic acid could be detected in urine.

Acrylic acid causes acute harmful effects following oral and dermal exposure in rats (oral LD_{50}: 140 up to 1,400 mg/kg bw; dermal LD_{50}: 300-600 mg/kg bw). Acute inhalation toxicity is normally stated to be low because acrylic acid interacts with humidity of the air prior to reaching the respiratory tract and causes respiratory irritation. Inhalation LC_{50} values ranging from 3.6 to >5.1 mg/l/4 h have been determined. Human data are not available.

Acrylic acid causes severe burns to skin and eyes in animals and severe irritation in the respiratory tract. The severe corrosive properties of the substance are demonstrated in a dose-dependent manner. In humans acrylic acid causes skin corrosion and irritation of the respiratory tract.

Persons exposed to acrylic acid can exhibit contact dermatitis. This was attributed to oligomeric impurities in the raw material; the pure acid does not show skin sensitizing properties.

Following repeated oral and inhalation exposure of acrylic acid in rats and mice, dose-related severe toxic effects were recorded. Gavage treatment with acrylic acid for 90 days revealed dose-dependent mortality, irritation and ulceration of the stomach, and renal tubular necrosis in rats (LOAEL 150 mg/kg bw/d).

No specific toxic effects were noted in further studies where acrylic acid was given with the drinking water. Reduced palatability (decreased water consumption) and non-specific signs of toxicity (decreased food consumption, body weight gain) at dosages >2,000 ppm (100 mg/kg bw/d in male rats, 150 mg/kg bw/d in females) were observed in subchronic and chronic studies A NOAEL of 40 mg/kg bw/d was derived for male rats, and of 83 mg/kg bw/d for female rats.

Toxic effects of relevance were seen in inhalation studies on rats and mice. In a 90-day inhalation study, acrylic acid vapour induced degenerative lesions on the olfactory mucosa in mice at 5 ppm (0.015 mg/l) and in rats at 75 ppm (0.0221 mg/l). Mice seemed to be more sensitive than rats, thus a LOAEC of 5 ppm (0.015 mg/l) was derived for local effects.

Long-term dermal exposure at concentrations >1% resulted in skin irritation.

There is no information on the health effects in humans of repeated exposure to acrylic acid.

Acrylic acid did not induce gene mutations in Salmonella or CHO cells (HPRT locus) but was clearly positive in the mouse lymphoma assay and in the *in vitro* chromosomal aberration test. Since in the mouse lymphoma assay small colonies were induced preferentially, the mutagenic potential of acrylic acid seems to be limited to clastogenicity. *In vivo*, acrylic acid did not induce mutagenic effects in either rat bone marrow cells or mouse germ cells after oral administration. Based on the present results and taken into account data on structurally-related acrylic compounds, it is unlikely that acrylic acid is mutagenic *in vivo*.

There is no concern for carcinogenicity from long-term studies on animals.

In oral reproductive toxicity studies (rats) no effects on reproductive function (fertility) were observed but some signs of postnatal developmental toxicity (retarded body weight gain of the pups) were seen following exposure of the parental generation. No gross abnormalities were observed in the offsprings. No prenatal developmental toxicity was observed (rats and rabbits, inhalation).

4.1.3.2 Workers

In **Table 4.6**, a summary of the effects which are relevant for the occupational risk assessment is given.

Table 4.6 Summary of effects relevant for the occupational risk assessment

Acrylic acid	Inhalation	Dermal
Acute toxicity	LC50 rat: 3,600 mg/m³/4 h	LD50 values for rabbits: 300 and 640 mg/kg bw
Irritation/Corrosivity	Respiratory tract irritant;	Pure substance: corrosive;
Sensitisation	No data; not suspected to be a respiratory tract sensitiser	Not a skin sensitizer (purified acrylic acid)
Repeated dose toxicity (local)	Respiratory tract irritant; LOAEC in mice: 5 ppm (15 mg/m³), a CFD/PBPK-model indicates a lower sensitivity of humans	Pure substance: corrosive; long term irritation threshold concentration between 4% and 1%
Repeated dose toxicity (systemic)	N(O)AEC (with and without extrapolation): 5 ppm (15 mg/m³)	Extrapolated NAEL greater than 600 mg/person/day
Mutagenicity	Unlikely to be mutagenic *in vivo*	
Carcinogenicity	Not suspected to be carcinogenic (experimental data)	
Fertility impairment	Not considered to be reprotoxic (experimental data)	
Developmental toxicity	Not considered to be reprotoxic (experimental data)	

For the purpose of risk assessment it is assumed that inhalation of vapour and skin contact are the main routes of exposure. Oral exposure it not considered to be a significant route of exposure.

4.1.3.2.1 Acute toxicity

Inhalation

Experimental investigation of the acute inhalation toxicity (rat, 4 h) of acrylic acid indicated a LC50 of 3,600 mg/m³. No lethality but local effects in the respiratory tract and hyperemia of the inner organs were observed at 2,970 mg/m³. Concerning respiratory tract irritation following exposure by inhalation please refer to "Irritation/Corrosivity/Inhalation" and '"Repeated dose toxicity/Inhalation (local)".

This value of 2,970 mg/m³ without lethality is much higher than the measured short-term value of 44.4 mg/m³ (assumed reasonable worst case) in the chemical industry. Therefore lethality due to inhalation exposure is not anticipated to occur.

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

Dermal

Dermal LD_{50} values of 300 mg/kg and 640 mg/kg bw are demonstrated for rabbits. Acute dermal toxicity is accompanied by corrosive effects to the skin.

The highest dermal exposure is estimated to be 42 mg/p/d (= 0.6 mg/kg/d) in the chemical industry and in industrial area when single skin contacts to acrylic acid are possible. Lethality due to skin contact is not anticipated to occur.

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

4.1.3.2.2 Irritation/Corrosivity

Dermal/Eyes

Acrylic acid led to severe burns of the skin. A 50% solution caused necrosis to rabbit skin after 1 minute and even a 10% solution led to irritation within 5 minutes. The repeated administration of a 4% solution to the skin of mice over 13 weeks (3 d/w) caused irritation while the administration of a 1% solution for 13 weeks or lifetime led to no effects. Acrylic acid caused also severe burns to the eyes. Irreversible changes were observed after application of the pure substance and a 10% solution to the eyes of rabbits. A 3% and a 1% solution led to irritation that reversed within 6 and 2 days respectively.

Acrylic acid is labelled with R 35 (Causes severe burns) and according to the preparations directive (88/279/EEC) a concentration of ≥ 10% should be labelled with R 35, a concentration of ≥ 5% and < 10% with R 34 and a concentration of ≥ 1% and < 5% with R 36/38. A 10% solution did not result in corrosivity as supposed by the labelling scheme, but the observation time of 5 minutes was too short, and corrosivity after a longer period is not excluded. In addition the 1% solution did not result in skin irritation. But overall the experimental data are not in a noteworthy contradiction to the above-mentioned labelling. The scheme is considered to be a pragmatic estimate of local effects of preparations.

Scenarios with corrosive AA preparations

In exposure scenarios where pure acrylic acid or corrosive preparations of acrylic acid (≥ 5% AA) are handled skin and eye contact is avoided by using suitable PPE. Potential exposure is assumed only by single contacts. These scenarios are all exposure situations in the chemical industry, the manufacture of adhesives in the industrial area and the use of adhesives with ≥ 5% AA in industrial and skilled trade applications.

Scenarios with irritant preparations

There are two scenarios where irritant preparations of acrylic acid (content < 5% AA) are handled. These are the use of adhesives with < 5% AA in the industrial area and skilled trade. In these situations it cannot be excluded that PPE is not worn. Skin and eye irritation may occur. Acute irritation (skin, eye) is a reversible adverse effect, which can be immediately recognised and prevented. These risk situations are not anticipated to result in additional types of risk reduction measures beyond those which are already being applied (e.g. appropriate hygiene measures).

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

Inhalation

Acute irritation testing revealed severe irritation of the respiratory tract of rats at 2,970 mg/m³ (4 h). A threshold for acute respiratory irritation is not described. With reference to the section on repeated dose toxicity, it is anticipated, that the respiratory tract irritation threshold for single exposure does not significantly differ from that for repeated exposure. This consideration implies that the assessment of local effects after repeated inhalation (see "Repeated dose toxicity") may be used for the assessment of short-term exposure as well. Experimental data concerning different exposure durations per day are not available. As a pragmatic approach it is assumed that the LOAEC of 5 ppm (6 h/d) is also appropriate to assess short-term exposure.

With regard to local chronic inhalation toxicity exposure situations in industrial areas like manufacture of adhesives (without LEV) and use of adhesives (with and without LEV) are evaluated as being of concern (see Section Repeated dose toxicity Inhalation (local) and **Table 4.7**). Additionally exposure situations either short-term or not occurring on a daily basis like production and further processing in the chemical industry (with an assumed reasonable worst case of 44.4 mg/m^3 as short-term value) and use of adhesives in skilled trade applications (exposure level: < 30 mg/m^3, shorter than shift length) give rise to concern with regard to acute respiratory irritation.

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

4.1.3.2.3 Sensitisation

Dermal

Acrylic acid is not considered to be a skin sensitiser. Dermal exposure of workers to acrylic acid is not anticipated to result in skin sensitisation.

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

Inhalation

Respiratory sensitisation has not been observed in humans and a sensitising potential was not observed in skin sensitisation testing. Overall acrylic acid is not suspected to be a respiratory sensitiser.

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

4.1.3.2.4 Repeated dose toxicity

Inhalation (local)

Vapours of acrylic acid are irritating to the upper respiratory tract. The assessment of its local irritation potency is based predominantly upon the available results of the 90-day inhalation studies in rats and mice. In both species tested degenerative changes to the olfactory epithelium were observed. For rats the NOAEC is 25 ppm, the LOAEC is 75 ppm. In mice, slight degeneration of olfactory epithelium was observed in some animals even at the lowest concentration of 5 ppm; at 25 ppm significant irritating effects were observed in almost all animals.

Comparison of results of the 2-week and 90-day inhalation studies with acrylic acid (see hazard assessment and discussion in WHO/IPCS Environmental Health Criteria 191, p 74-75) reveals that the effects caused by acrylic acid are largely determined by the exposure concentration and are relatively less affected by the duration of exposure in repeated exposure studies. Furthermore, results of a chronic inhalation study with the acrylic acid methyl ester indicate that most changes in the rat nasal mucosa developed during the first 12 months of exposure and increased only moderately with ongoing exposure up to 24 months (Reininghaus et al., 1991). In addition, comparison of methyl methacrylate chronic and subacute inhalation studies (see EU Risk Assessment Report for methyl methacrylate) supports the conclusion that progression of lesions of the olfactory epithelium might be minimal. Thus in conclusion, it may be assumed that the nasal irritation threshold for acrylic acid will not substantially change when extrapolation is made from experimentally-tested subchronic exposure to chronic exposure.

The main problem in the acrylic acid risk assessment is the species extrapolation from rodents to humans. Rodents show a nasal anatomy and respiratory physiology different from man. For instance, the architecture of nasal passages is more complex in rodents than in humans. These differences will influence the toxicokinetics of substances in the upper respiratory tract of species. A CFD/PBPK-model was constructed for interspecies (rat-human) extrapolation of acrylic acid tissue dose in the olfactory region of the nasal cavity. The model simulations indicate that under similar exposure conditions human olfactory epithelium is exposed to a 2-3-fold lower dose compared to rat olfactory presentation with regard to the uncertainty of the model and the sensitivity parameters (see Section 4.1.2.1). Therefore the calculation of both the direct and adjusted MOS is performed with the LOAEC of 5 ppm (15 mg/m³), that resulted in slight effects in some mice (near to NAEC).

The LOAEC of 15 mg/m³ is compared with the exposure information for scenarios with repeated daily inhalation exposure. For details see **Table 4.7**. The most critical exposure scenarios are found in the industrial area outside the chemical industry (manufacture of adhesives without LEV: MOS 0.4-2, use of adhesives without LEV: MOS 0.5, with LEV MOS 2-10). These MOS values are considered to be of concern.

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

Table 4.7 Repeated dose toxicity (inhalation/local and systemic and dermal contact/systemic): MOS values and conclusions

Area of production and use	Inhalation (local, systemic)				Dermal contact (systemic effects)		
	Shift-average value [mg/m³]	Direct MOS = adjusted MOS [1, 2)]	Conclusion		Shift-average value [mg/p/d]	Direct MOS, resp. adjusted MOS [3)]	Conclusion
			local	systemic			
Chemical Industry							
Production, further processing (filling, transfer, cleaning, maintenance, repair work)	3.0 [4)]	5	ii	ii	low [5)]	high	ii
Manufacture of adhesives (1 - 10% acrylic acid) (filling, transfer, cleaning, maintenance, repair work, drumming)	0.375 - 2.25 [6)]	7 - 40	ii	ii	low [5)]	high	ii
Industrial area							
Manufacture of adhesives (1 – 10% acrylic acid) (filling, transfer, cleaning, maintenance, repair work)	0.375 – 2.25 [6)] 7.5 – 37.5 [7)]	7 – 40 0.4 – 2	ii **iii**	ii **iii**	low [5)]	high	ii
Use of adhesives - ≥ 5% acrylic acid (handling, glueing, charging)	1.5 - 9 [6)] 30 [7)]	2 - 10 0.5	**iii** **iii**	ii **iii**	low [5)]	high	ii
- < 5% acrylic acid (handling, glueing, charging)	1.5 - 9 [6)] 30 [7)]	2 - 10 0.5	**iii** **iii**	ii **iii**	1 - 10.5 [8)]	14-150 resp. > 57-600	ii
Decomposition during production of integrated circuits	low [5)]	high	ii	ii	low [5)]	high	ii

1) LOAEC (local): 15 mg/m³
2) N(O)AEC (systemic): 15 mg/m³
3) NAEL 150 mg/p/d resp. > 600 mg/p/d
4) 90th percentile
5) expert judgement
6) EASE (inhalation, with LEV)
7) EASE (inhalation, without LEV)
8) EASE (dermal, without PPE

Inhalation (systemic)

There was no systemic toxicity in rats and male mice, systemic NOAEC therefore was 75 ppm. Because of lower body weight gain, the NOAEC for female mice was 5 ppm with a systemic LOAEC of 25 ppm. Overall, comparison of local (LOAEC of 5 ppm) and systemic dose responses shows that the toxic profile of acrylic acid is dominated by its local effects in the upper respiratory tract. The formal systemic LOAEC is 5 times greater than the local LOAEC, a clear systemic target organ was not found and it is not excluded, that lower body weight gain might be secondary to the predominant local effects at 25 ppm. A comparison of subchronic and chronic oral studies with acrylic acid shows that a specific duration adjustment is not necessary for systemic effects. So a MOS-calculation (direct and adjusted) of systemic effects could be based on the N(O)AEC of 5 ppm (= 15 mg/m³).

On the basis of this N(O)AEC the corresponding MOS values for repeated dose toxicity (systemic) are the same figures as the local MOS values the latter being based on the local LOAEL. Referring to the hazard assessment part of the report the lower body weight gain at 25 ppm is not described as secondary to local effects. So local and systemic risks need to be evaluated separately.

The MOS values calculated for the manufacture of adhesives without LEV (0.4-2) and use of adhesives without LEV (0.5) in industrial applications are evaluated as being of concern. Unlike the evaluation of local chronic inhalation risks the industrial area of use of adhesives with LEV is not considered of concern with regard to systemic toxicity. The different starting point for MOS calculation [LOAEC (local) resp. N(O)AEC (systemic)] has to be taken into consideration (results see **Table 4.7**).

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

Dermal (local)

Please refer to the preceding Section “Irritation/Corrosivity: Dermal/ Eyes”.

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

Dermal (systemic)

For chronic dermal contact to acrylic acid preparations which are not labelled as corrosive possible systemic toxicity needs to be discussed. The results of the 90-day inhalation studies and the drinking water studies may be used for route-to-route extrapolation.

In the inhalation studies (with nasal cavity toxicity as primary effect) lower body weight gain with a NOAEC of 5 ppm (15 mg/m³) was observed in female mice (respectively a NOAEC of 75 ppm for rats and male mice). The obstacles that complicate the identification of an independent systemic toxicity are already mentioned. Assuming a breathing volume of 10 m^3 per shift, an inhalation intake of 150 mg/person/day (based on female mice) respectively 2,250 mg/person/day (based on rats and male mice) may be calculated as dosage without systemic effects. The dose of 150 mg/person/day is used for the formal calculation of the direct MOS. For the calculation of an adjusted MOS route-specific differences in systemic availability should be considered due to differences in absorption. For a hydrophilic vapour like acrylic acid a nearly quantitative absorption (100%) via inhalation is assumed. In Section 4.1.2.1 dermal absorption rates of 12-26% are described. The highest value of about 25% is used for a calculation of route-specific

differences. A further duration adjustment is considered to be not necessary (see under Inhalation (systemic)). For the calculation of an adjusted MOS a dermal NAEL (human, chronic) of about 600 (150·4) mg/person/day is assumed.

For repeated oral administration a lowest NOAEL of 40 mg/kg/day (male rats) is derived from a drinking water study. Obviously the bad palatability led to a lower drinking water intake, which was considered to result in lower food consumption and body weight gain. Other effects, observed at higher doses of the drinking water studies, were not indicative of a clear target organ and might be partly based on the above-mentioned effects (for details see Section 4.1.2.6). Additionally a gavage study was available, but the effects observed in that study were attributed to high peak concentrations. An oral exposure schedule, which does not result in high peak concentrations, (that is the drinking water study) is considered more appropriate for the dermal risk assessment. The formal NOAEL (40 mg/kg/day) is to be used for a calculation of the direct MOS and as a starting point for extrapolation, but a more relevant dose might be higher (perhaps 3-fold; range of 150 mg/kg/day). Metabolic rate scaling requires an extrapolation factor of 1/4 resulting in an anticipated human oral NAEL of 10 mg/kg/day (700 mg/person/day). For a route-to-route extrapolation specific differences in systemic availability should be considered due to differences in absorption. According to Section 4.1.2.1, the absorption after oral administration should be nearly quantitative. Dermal absorption is considered to be about 25%. A specific duration adjustment is not applied for acrylic acid. Based on the oral study a dermal NAEL (human, chronic) of 2,800 (700·4) mg/person/day is estimated.

The NOAEC/NOAEL of both the inhalation and the drinking water study have their limitations as starting point for an assessment of eventual systemic effects after dermal application. The formal NOAEC of the inhalation study (5 ppm) is derived from female mice, the NOAECs for male mice and rats were clearly higher. In addition it is not excluded, that the observed effect (lower body weight gain) is secondary to the predominant local effect. The formal NOAEL of the oral study (40 mg/kg/day) is based on effects that were related to the bad palatability and should not be relevant for the dermal route. More relevant doses might be about 3-fold higher, but a clear systemic target organ toxicity is not described in the drinking water studies. The toxicity profile of acrylic acid (oral and inhalation) is clearly influenced by the route of administration and the main weakness of both studies is the debatable appropriateness of the observed dose-response relationship to be extrapolated to the dermal route. A strong scientific argument to prefer the oral or inhalation study as a starting point is not available and erring possibly on the side of caution the inhalation study resulting in a lower NAEL is selected for the dermal risk assessment.

The NAEL used for the calculation of the direct MOS is 150 mg/p/d, for the adjusted MOS: > 600 mg/p/d.

For scenarios with repeated daily dermal exposures the highest exposure level is calculated to be 1-10.5 mg/p/d (industrial area: use of adhesives with <5% acrylic acid). The calculated MOS values are 14-150 (direct MOS), resp.>57-600 (adjusted MOS). Details see **Table 4.7**. Therefore systemic health risks by chronic dermal exposure are not considered of concern.

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

Combined exposure (systemic effects)

Systemic health effects due to combined exposure (inhalation and dermal contact) are to be assessed in addition to route-specific risk estimates.

The MOS values for combined exposure are calculated by the formula:

$$\frac{1}{MOS_{comb.}} = \frac{1}{MOS_{inh.}} + \frac{1}{MOS_{derm.}}$$

One scenario with daily repeated combined inhalation and dermal exposure is found in the industrial area.

The results of the calculations for combined exposure are presented for "Use of adhesives" as an example in **Table 4.8**.

Table 4.8 Combined exposure (repeated dose toxicity, systemic)

Exposure scenario	MOS $_{inhalation}$*	Conclusion	MOS $_{dermal}$*	Conclusion	MOS $_{combined}$	Conclusion
Industrial area: use of adhesives (< 5% acrylic acid)	 2 with LEV 0.5 without LEV	 ii **iii**	 57 57	 ii ii	 1,9 0.5	 ii **iii**

* Lowest MOS values of ranges are used

The MOS for combined exposure is mainly determined by the MOS for inhalation exposure. The conclusions are identical with those for isolated inhalation exposures. The conclusion for the scenario "use of adhesives (< 5% AA), ind. area, without LEV" is **conclusion (iii)**. Thus no relevant additional risk by combined exposure is expected. This conclusion is also applicable to other scenarios where conclusion (iii) has been applied for repeated dose toxicity, systemic (see **Table 4.9**).

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

4.1.3.2.5 Mutagenicity

With reference to Section 4.1.2.7, acrylic acid is unlikely to be mutagenic *in vivo*. Corresponding risks at workplaces are not anticipated to occur.

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

4.1.3.2.6 Carcinogenicity

There is no evidence that acrylic acid administered orally to rats or applied dermally to mice is carcinogenic. Human data on carcinogenicity is not available. In conclusion, acrylic acid is not

suspected to be a carcinogenic agent. Based on these data carcinogenic effects are not anticipated to occur.

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

4.1.3.2.7 Toxicity for reproduction

Based on experimental data, acrylic acid is not considered to be a reproductive toxicant (fertility impairment and developmental toxicity). The oral rat NOAEL for reproductive function is 460 mg/kg/day. The rat NOAEC for developmental toxicity is reported to be 360 ppm (1,060 mg/m^3). Comparison of these data on reproductive toxicity with threshold levels for general local and systemic toxicity (anticipated NAEC for respiratory tract irritation slightly below 5 ppm; oral NOAEL of 40 mg/kg/d for systemic effects in the rat drinking water study) shows that toxicity for reproduction is not anticipated to occur.

Corresponding occupational risks are not anticipated to occur.

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

4.1.3.2.8 Conclusions of the occupational risk assessment

The conclusions of the occupational risk assessment are summarised in **Table 4.9**.

Table 4.9 Conclusions of the occupational risk assessment of acrylic acid

	Acute toxicity (inh., dermal)	Irritation/ Corrosivity (dermal)	Irritation/ Corrosivity (inh.)	Sensiti-sation (inh., dermal)	Repeated dose tox. (local, inh.)	Repeated dose tox. (system., inh.	Repeated dose tox. (local, system. dermal)	Repeated dose tox. (combined exposure, systemic)	Muta-genicity	Carcino-genicity	Repro-ductive toxicity
Chemical industry											
Production and further processing	ii	ii	**iii**	ii	ii	ii	ii	ii	ii	ii	ii
Manufacture of adhesives (1-10% acrylic acid)	ii	ii	ii	ii	ii	ii	ii	ii	ii	ii	ii
Industrial area											
Manufacture of adhesives (1 - 10% acrylic acid)											
- with LEV	ii	ii	ii	ii	ii	ii	ii	ii	ii	ii	ii
- without LEV	ii	ii	**iii**	ii	**iii**	**iii**	ii	**iii**	ii	ii	ii
Use of adhesives ≥ 5% acrylic acid (labelled as corrosive)											
- with LEV	ii	ii	**iii**	ii	**iii**	ii	ii	ii	ii	ii	ii
- without LEV	ii	ii	**iii**	ii	**iii**	**iii**	ii	**iii**	ii	ii	ii
< 5% acrylic acid (not labelled as corrosive)											
- with LEV	ii	ii	**iii**	ii	**iii**	ii	ii	ii	ii	ii	ii
- without LEV	ii	ii	**iii**	ii	**iii**	**iii**	ii	**iii**	ii	ii	ii
Decomposition during production of integrated circuits	ii	ii	ii	ii	ii	ii	ii	ii	ii	ii	ii
Skilled trade											
Use of adhesives ≥ 5 - 10% acrylic acid (labelled as corrosive)	ii	ii	**iii**	ii	ii	ii	ii	ii	ii	ii	ii
≤ 5% acrylic acid (not labelled as corrosive)	ii	ii	**iii**	ii	ii	ii	ii	ii	ii	ii	ii

4.1.3.3 Consumers

4.1.3.3.1 Acute Toxicity

Following the exposure assessment, consumers are not expected to be exposed to acrylic acid in the range of doses which can be derived from acute oral or dermal toxicity figures based on animal LD_{50} values (oral: 140 up to 1,400 mg/kg bw and dermal: 300-640 mg/kg bw). Therefore the substance is of no concern in relation to acute oral or dermal toxicity.

Consumer exposure may occur as a result of the inhalation exposure to adhesives (up to peak concentrations of 0.542 µg/l as calculated applying the SCIES model). Following inhalation exposure in rats, acrylic acid has demonstrated a LC_{50} value of 3,600 µg/l/4h.

Acute toxicity is dominated by chemical interactions with water and/or biological material. Acrylic acid causes acute harmful effects by the oral, inhalation and dermal routes of exposure. Concerning an acute inhalation exposure scenario, the margin of safety is assumed to be sufficient.

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

4.1.3.3.2 Irritation/Corrosivity

Acrylic acid is known to cause skin corrosion and irritation of the respiratory tract in humans. The substance causes severe burns to skin and eyes of animals. Necrosis to rabbit skin is already caused by a 50% aqueous substance solution after 1 minute of exposure.

According to the severe local corrosive properties acrylic acid is classified as corrosive, C and labelled with the R phrase R 35, causes severe burns. Provided the safety advice in accordance with the classification is followed current risk reduction measures are considered sufficient.

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

4.1.3.3.3 Sensitisation

Pure acrylic acid does not show skin sensitisating properties. The sensitising properties of previously available acrylic acid samples (commercial grade) was attributed to an impurity (α,β-diacryloxypropionic acid). This impurity could not be detected in current commercial acrylic acid.

There is no information available on the potential of acrylic acid to produce respiratory sensitization in animals. However, respiratory sensitization has not been observed in humans.

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

4.1.3.3.4 Repeated dose toxicity

Following the exposure assessment consumers may be exposed to an average concentration of 0.384 mg/m^3 (1 hour) with a possible peak value of 0.542 mg/m^3 using UV-hardening adhesives. This exposure does not reflect a realistic chronic exposure scenario. However, during use of nappy pants babies and smaller children may be exposed dermally to acrylic acid. This exposure results from the residual content of monomers in homopolymers of acrylic acid which are used as "superabsorbents" in such products.

Studies in experimental animals have shown that the toxicity of acrylic acid is dominated by its local irritation effects irrespective of the manner of application. Prolonged inhalation (14 and 90 days) of concentrations from 5 ppm or higher in mice (15 mg/m^3) and from 75 ppm or higher in rats (221 mg/m^3) induced degeneration of the olfactory mucosa. A NOAEC for local effects of 25 ppm (resp. 74 mg/m^3) was derived from the 90-day study on rats, whereas no data on NOAEL are available in mice. In this species a LOAEC of 5 ppm (15 mg/m^3) could be derived. Acrylic acid causes severe mucosal damage to the stomach after repeated gavage administration of >150 mg/kg bw/d, but not after application via drinking water at similar or higher doses (NOAEL 40 mg/kg bw/d (male rats), resp. 83 mg/kg bw/d (female rats)). Long-term dermal exposure of acrylic acid at concentrations of >1% resulted in skin irritation whereas no skin effect was evident at 1% (cf. dermal carcinogenicity studies in Section 4.1.2.8).

Following oral, dermal or inhalation administrations no other systemic toxic effects were detected except premature deaths and tubular degeneration/necrosis in the kidneys which were evident after gavage administration of dosages >150 mg/kg bw/d in a rat 3-month study. Effects did not occur in drinking water studies at similar or higher doses. After prolonged inhalation exposure a NOAEC for systemic effects of 5 ppm (15 mg/m^3) was derived for female mice and of 75 ppm (221 mg/m^3) for male mice as well as female and rats.

For the decision on the appropriateness of MOS, the following aspects regarding the critical effect as well as exposure have been considered and taken into account:

Overall confidence in the database

The data taken into account for performing the risk characterisation have been evaluated with regard to their reliability, relevance and completeness according to Section 3.2 of the TGD. The data were published in peer-reviewed journals or submitted to the Competent Authority in private reports being adequately detailed and in accordance with internationally recognised guidelines and to GLP.

The findings of all studies are not contradictory so that the judgement can be based on the database.

There are no reasons to assume limited confidence.

Uncertainty arising from the variability in the experimental data

The studies cited above allow a conclusion on the NOAEC/LOAEC of severe health effects during inhalation (degenerative lesions of the olfactory mucosa) from four studies. There is only one valid study to derive a NOAEC for local irritation on rats. Comparing the local effect concentrations, mice seem to be more sensitive than rats without sex preference. Thus, a LOAEC was derived from one single mice study, which was well performed and the results were in conformity with the findings of the other studies.

Three oral 90-day studies on rats allow the derivation of a NOAEL for systemic effects. The range varied from 40 mg/kg bw/d to 331 mg/kg bw/d. The NOAEL of 40 mg/kg bw/d (decreased body weight gain) was derived from the drinking water study (BASF, 1987) which was well performed and the results were in conformity with the findings of the other studies.

There are no reasons to assume a special extent of uncertainty which have to be taken into account.

Intra- and interspecies variation

Hitherto available data on kinetics of acrylic acid do not allow to calculate the intraspecies and interspecies variability by applying modern approaches. However, the present data give no hint on a particular high variability in kinetics. The variability of the data on the toxicodynamics has been described above and seems not to justify an increased MOS.

Nature and severity of the effect

The main effect considered as "critical effect" is degeneration of the olfactory mucosa (irreversible, serious health effect).

There are no reasons to assume that the effects shown in the animal experiments are limited to the species tested, thus being of no relevance for humans. Because of the seriousness of the effect there is concern, which has to be expressed in the magnitude of the MOS.

Dose response relationship

In rats as well as mice no steep dose-response relationship is observed for the irritation effects at the olfactorium. Due to the fact that the LOAEC from the most sensitive species is used for MOS considerations we are of the opinion that no further reasons are given for requiring a higher MOS.

Differences in exposure (route, duration, frequency and pattern)

Inhalation and oral route

Following the exposure assessment, the consumer may be exposed to acrylic acid via inhalation, oral exposure can be neglected. The described human exposure scenario (usage of an adhesive) does not represent a real chronic scenario. The LOAEC used for the discussion of the MOS regarding this application is derived from a 90-day inhalation study in mice. Because acrylic acid acts directly and locally at the nasal cavity, systemic effects have not to be considered.

Dermal route

The estimated dermal body burden with an assumed absorption of 100% is compared with an oral NOAEL from a 90-day study.

There are no reasons to assume that special concern can be derived neither from this procedure nor from the available toxicokinetic information; concerning different routes inasmuch as absorption was set at 100%.

Human population to which the quantitative and/or qualitative information on exposure applies

Following the inhalation exposure there is no reason to assume a special risk for elderly, children or other people suffering from special diseases like obesity or persons with high bronchial reactivity.

Regarding the dermal exposure special concern has to be directed to infants, in particular premature babies.

Other factors

There are no other factors known requiring a peculiar margin of safety.

MOS for inhalation exposure scenario

During application of an UV-hardening adhesive for one hour (4 times per year) the consumer may be exposed to an average concentration of 0.384 mg/m^3 with a possible peak value of 0.542 mg/m^3. This exposure does not reflect a realistic chronic exposure scenario.
Therefore, the margin of safety between the

estimated exposure level of	0.384 mg/m^3
and the	
LOAEC for local irritation effects of	15 mg/m^3

is about 40 which is judged to be sufficient because a worst-case exposure scenario and a LOAEC for mice as more sensitive species are taken into consideration.

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

MOS for dermal exposure scenario

The calculation of the dermal exposure of babies due to nappies leads to an internal exposure of 0.00018 mg/kg bw/d (uptake basis assuming the bioavailability via the dermal route is 100%). The margin of safety between the

estimated exposure level of	0.00018 mg/kg bw/d
and the	
oral NOAEL of	40 mg/kg bw/d

is about 200,000 which is judged to be sufficient, even if the special considerations on premature babies as population at risk and route-to-route extrapolation are taken into consideration.

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

MOS for oral exposure scenario

The oral uptake is negligible.

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

4.1.3.3.5 Mutagenicity

Acrylic acid is non-mutagenic in Salmonella and CHO cells (HPRT locus) but clearly mutagenic in the mouse lymphoma assay and in the *in vitro* chromosomal aberration test. However, *in vivo* acrylic acid did not produce mutagenic effects in either rat bone marrow cells or mouse germ cells after oral administration. Based on the present results and taken into account data on structurally-related acrylic compounds, it is unlikely that acrylic acid is mutagenic *in vivo*.

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

4.1.3.3.6 Carcinogenicity

Studies on experimental animals indicate that acrylic acid is not carcinogenic in animals. There are no cancer data available with respect to human exposure.

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

4.1.3.3.7 Toxicity for reproduction

In oral reproductive toxicity studies (rats), no effects on reproductive function (fertility) were observed, but some signs of postnatal developmental toxicity (retarded body weight gain of the pups) were seen following exposure of the parental generation. No gross abnormalities were observed in the offsprings. No prenatal developmental toxicity was observed (rats and rabbits, inhalation).

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

4.1.3.4 Humans exposed via the environment

Indirect exposure via the environment is estimated using both a regional and a point source model.

The main route of exposure is via the drinking water for the regional exposures and stem/air for the point source approach.

Following these data, a total daily dose of 50 µg/kg bw/d is calculated for the local scenario and of 15.1 ng/kg bw/d for the regional one. In a repeated dose toxicity study (male rats, oral, 90-day study) the NOAEL was 40 mg/kg bw/d.

Comparison indirect exposure - Local scenario/NOAEL

$$\frac{\text{Indirect exposure}}{\text{NOAEL}} = \frac{40\ \text{mg/kg bw/d}}{0.050\ \text{mg/kg bw/d}} = 800$$

The margin of safety for systemic effects between the calculated local exposure and the NOAEL is considered to be sufficient. 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 or testing or risk reduction measures beyond those which are being applied already.

Comparison indirect exposure - Regional scenario/NOAEL

$$\frac{\text{Indirect exposure}}{\text{NOAEL}} = \frac{40\ \text{mg/kg bw/d}}{0.000015\text{mg/kg bw/d}} \approx 2 \cdot 10^6$$

On the basis of these data the margin of safety for the regional scenario is considered to be sufficient. 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 or testing or 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

See Section 4.1.1.2.

4.2.2 Effects assessment

4.2.2.1 Explosivity

Acrylic acid is not explosive.

4.2.2.2 Flammability

Acrylic acid is flammable.

4.2.2.3 Oxidising potential

In view of its chemical structure, acrylic acid is not expected to have an oxidising potential.

4.2.3 Risk characterisation

4.2.3.1 Workers

Acrylic acid is flammable. If it is heated above its flash point, an explosive atmosphere may be formed (lower explosion limit: 2.4% (vol.); upper explosion limit: 15.9%(vol.) according to manufacturer). In order to exclude any possible hazard to workers, the national regulations on handling flammable liquids and on the prevention of explosions must be observed.

Adequate worker protection measures must be observed. Risk reduction measures beyond those which are being applied already are not considered necessary.

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

5 RESULTS

5.1 ENVIRONMENT

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

Acrylic acid (AA) presents, based on the present data, a risk to the environment around point sources. A potential risk to municipal wastewater treatment plants is identified for the downstream use scenarios of super absorber polymers (SAP) production (based on default calculation and highest site-specific PEC_{wwtp}) and wet polymerisation (based on default calculation and known sites L, Q).

Since the $PNEC_{microorganisms}$ is derived from single species tests with ciliated protozoa, there is a need for further data reflecting the integrity of the native ciliate population in sewage sludge as a whole. However, since risk reduction measures are necessary to remove concern for surface water (see below), these measures will also cover the protection of municipal wastewater treatment plants, and additional testing is not required.

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

This conclusion applies to effects on sediment, atmosphere, soil, and secondary poisoning. Conclusion (ii) applies also to the aquatic compartment regarding all production sites, the processing scenario (dry polymerisation), and the relevant use scenarios (leather finishing, textile finishing, formulation of paints and application of water treatment agents).

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

Acrylic acid (AA) presents, based on the present data, a risk to the environment around point sources.

A potential risk to the local aquatic environment is identified from wet polymerisation processes including wet production of SAP (super absorber polymers) by downstream users of monomeric AA (based on default calculations and known sites N, O, Q).

Although an improvement of the data (i.e. effluent measurements and/or site specific data on flow rates) may in principle be possible, it is judged to be unlikely that sufficiently complete representative monitoring data from the downstream users can be obtained with reasonable expenditure of time and money. For certain known SAP production sites and wet polymerisation sites, regular effluent concentrations up to 100 mg/l AA and significantly more have been reported. These data indicate that high effluent concentrations cannot be excluded, even if certain types of process engineering are applied. On the other hand, application of wastewater reutilization / recycling systems is known to result in zero emissions to the hydrosphere at a number of downstream user sites, processing about 50% of AA used externally for SAP production and about 12% of AA used externally in wet polymerisation processes. For sites applying this kind of technique, no further risk reduction measures are deemed necessary.

Measures applied for limiting the risk to the local aquatic environment are presumed to be also protective for municipal wastewater treatment plants.

During the use of a grouting agent containing magnesium diacrylate high concentrations of AA are released via the drainage water. The exposure assessment was based on measured effluent concentrations at a tunnel construction site. A quantitative extrapolation to other construction sites seems difficult, but similar conditions might be anticipated. Measures appropriate to local circumstances should be applied.

5.2 HUMAN HEALTH

5.2.1 Human health (toxicity)

5.2.1.1 Workers

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

This conclusion is reached because of:

- concerns for respiratory tract irritation and corrosivity as a consequence of single inhalation exposure arising from production and processing, production of adhesives containing the substance and use of adhesives containing the substance (industrial area and skilled trade),
- concerns for local effects as a consequence of repeated inhalation exposure arising from production and use of adhesives containing the substance,
- concerns for general systemic toxicity as a consequence of repeated inhalation exposure arising from production and use of adhesives containing the substance.

5.2.1.2 Consumers

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

5.2.1.3 Humans exposed via the environment

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

5.2.2 Human health (risks from physico-chemical properties)

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

6 REFERENCES

Aquateam (1999). Sluttrapport. Utslipp knyttet til bruk av kjemiske tetningsmidler i Romeriksporten. Rapport Nr 99-010. Utarbeidet for NSB Gardermobanen, April 1999, 57 pp.

Arbejdstilsynet (1996). Grænseværdier for stoffer og materialer. At-anvisning Nr.3.1.0.2. København Ø.

Archer G, Horvath MK (1991). Adsorption and desorption of acrylic acid to soils; Report No 3193-88-0214-EF-001. Ricerca, Inc (1991) zitiert in Tyler et al.

Ashby J, Richardson CR, Tinwell H (1989). Inactivity of ethyl acrylate in the mouse bone marrow micronucleus assay. Mutagenesis 4, 283 – 285.

BAMM (1988). Bushy Run Research Center. Acrylic acid: Acute vapor inhalation toxicity test in rats. Project Report 51-577 Draft.

BAMM (1990). Letter of 14.12.1990, with Final Report.

BAMM (1991). Bushy Run Research **C**enter, unpublished report, 04.10.1991.

BAMM (1993). Technical Committees, FYI 19.02.1993.

Barrow CS (1984). Species differences in toxicology of the nasal passages: Acrylic acid and Dimethylamine. CIIT Activities 4, 1-5.

Barrow CS (1986). Quantititation of Nasal "dose" with formaldehyde, acrylic acid, and dimethylamine. In: Toxicology of the nasal passages. (CS Barrow, ed). Hemisphere Publishing Corporation, Washington, New York, London, pp 113-122.

BASF AG (1958). Bericht über die biologische Prüfung der reinen und rohen Acrylsäure. Report No. VII/365-366, 21.07.1958.

BASF AG (1977). Abteilung Toxikologie, unpublished report 77/238.

BASF AG (1979). Bericht über die Prüfung der akuten dermalen Toxizität von Acrylsäure rein an der Rückenhaut weißer Kaninchen. Report for the substance 78/520 from 08.01.1979.

BASF AG (1980). Bestimmung der akuten Inhalationstoxizität LC50 von Acrylsäure rein als Dampf bei 4stündiger Exposition.

BASF AG (1983). Abteilung Toxikologie, unpublished report (80/386), Project No. 37 R0386/8017, 30.12.1983.

BASF AG (1987). Abteilung Toxikologie, unpublished report, Project-No. 35C0380/8250), 28.04.1987.

BASF AG (1988). Analytisches Labor; unveröffentlichte Untersuchung (J.Nr. 129304/01 vom 02.09.88).

BASF AG (1989). Abteilung Toxikologie, unpublished report (82/380), 30.03.1989.

BASF AG (1992). Technische Information Monomere, Acrylsäure rein. Ausgabe TI/ED 1330.

BASF AG (1993). Bestimmung der Atmungshemmung mit Belebtschlamm von Acrylsäure rein im Kurzzeitatmungshemmtest, Labor Ökologie; unveröffentlichte Untersuchung, Bericht vom 18.01.93 (Projektnr. 92/2641/08/2).

BASF AG (1993a). Unpublished information, 15.02.1993, 02.09.1994.

BASF AG (1994a). Sicherheitsdatenblatt Acrylsäure rein (22.08.1994).

BASF AG (1994b). Bestimmung der Hemmwirkung von Acrylsäure rein auf die Zellver-mehrung der Grünalge Scenedesmus subspicatus, Labor Ökologie; Unveröffentlichte Untersuchung vom 04.07. bis 07.07.1994: (Projektnummer 94/0840/60/1).

BASF AG (1994c). Abteilung Toxikologie, unpublished report (Project No.: 71R0114/92011), 12.01.1994.

BASF AG (1997). Personal communication, 14.02.1997.

BASF AG (1998). Department of Toxicology, Report: Acrylic acid glacial. Acute dermal irritation / corrosion in rabbits. Unpublished report, 24 June 1998.

Basic Acrylate Monomer Manufactures (1991). Washington D.C., AA-4-AA-5.

Bauer JR W (1991). Acrylic Acid and Derivatives in: Kirk-Othmer, Encayclopedia of chemical Technology, 4. Aufl., Band 1, 287-314.

Black KA et al. (1994). The Toxicologist 14, 430.

Black KA, Finch L, Frederick CB (1993). Metabolism of acrylic acid to carbon dioxide in mouse tissues. Fundam. Appl. Toxicol. 21, 97-104.

Black KA, Beskitt JL, Finch L et al. (1995). Disposition and metabolism of acrylic acid in C3H mice and Fischer 344 rats after oral or cutaneous administration. J. Toxicol. Environ. Health 45, 291-311.

Black KA, Finch L (1995). Acrylic acid oxidation and tissue-to-blood partition coefficients in rat tissues. Toxicol. Lett. 78, 73-78.

Bowman JH (1990). Acute flow-through toxicity of glacial acrylic acid to Rainbow Trout (Salmo gairdneri), ABC Final Report 37343 und ABC Protocol No. 8007-PMN.

Bringmann G (1978b). Bestimmung der Schadwirkung wassergefährdender Stoffe gegen Protozoen, I. Bakterienfressende Flagellaten, Zeitschrift für Wasser- und Abwasser-Forschung 11(6), 210-215.

Bringmann G et al. (1980). Bestimmung der Schadwirkung wassergefährdender Stoffe gegen Protozoen, III. Saprozoische Flagellaten, Zeitschrift für Wasser- und Abwasser-Forschung 13(5), 170-173.

Bringmann G, Kühn R (1977). Grenzwerte der Schadwirkung wassergefährdender Stoffe gegen Bakterien (Pseudomonas putida) und Grünalgen (Scenedesmus quadricauda) im Zellvermehrungshemmtest, Zeitschrift für Wasser- und Abwasser-Forschung, 10(3/4), 87-98.

Bringmann G, Kühn R (1978a). Grenzwerte der Schadwirkung wassergefährdender Stoffe gegen Blaualgen (Microcystis aeruginosa) und Grünalgen (Scenedesmus quadricauda) im Zellvermehrungshemmtest, Vom Wasser 50, 45-60.

Bringmann G, Kühn R (1980). Zeitschrift für Wasser- und Abwasser-Forschung 1, 26-31.

Bringmann G, Kühn R (1982). Ergebnisse der Schadwirkung wassergefährdender Stoffe gegen Daphnia magna in einem standardisierten Testverfahren, Zeitschrift für Wasser- und Abwasser-Forschung, 15(1), 1-6.

BUA-Stoffbericht (1995). 160, Acrylsäure (2-Propensäure), Gesellschaft Deutscher Chemiker, Beratergremium für umweltrelevante Altstoffe, S Hirzel, Wissenschaftliche Verlagsgesell-schaft.

Buckley LA, James RA, Barrow CS (1984). Differences in nasal cavity toxicity between rats and mice exposed to acrylic acid vapors. The Toxicologist 4, 1.

Burgess D (1989). Acute flow-through toxicity of acrylic acid to Daphnia magna, Analytical bio-chemistry Laboratories, Inc., Report #37344.

Bush ML, Frederick CB, Kimbell JS, Ultman JS (1998). A CFD-PBPK hybrid model for simulating gas and vapor uptake in the rat nose. Toxicol. Appl. Pharmacol. 150, 133-145.

Bushy Run Research Center (1993). Developmental toxicity evaluation of inhaled acrylic acid vapor in New Zealand White Rabbits. Unpublished report 92N1008, 24.06.1993.

Cameron TP, Rogers-Back AM, Lawlor TE, Harabell JW, Seifried HE, Dunkel VC (1991). Genotoxicity of multipfunctional acrylates in the Salmonella/mammalian-microsome assay and mouse lymphoma TK+/- assay. Environ. Molecul. Mutagen. 17, 264-271.

Carpenter CP, Weil CS, Smith HF (1974). Range-finding toxicity data: List VIII. Toxicol. Appl. Pharmacol. 28, 313-319.

Celanese Corp. (1986). Chromosomal aberration test. Unpublished report (T4901), 338, 28.07.1986.

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

CITI (1992). Biodegradation and Bioaccumulation Data of Existing Chemicals Based on the CSCL Japan, edited by Chemicals Inspection & Testing Institute Japan, published by Japan Chemical Industry Ecology-Toxicology & Information Center.

Conde-Salazar L, Guimaraens D, Romero LV (1988). Occupational allergic contact dermatitis from anaerobic acrylic sealants. Contact Dermatitis 18, 129-132.

DeSesso JM (1993). The relevance to humans of animal models for inhalation studies of cancer in the nose and upper airways. Quality Assurance: Good Practice, Regulation, and Law 2 (3), 213-231.

D'Souza WR, Francis WR (1988). Vehicle and pH effects on the dermal penetration of acrylic acid: *in vitro-in vivo* correlation. Toxicologist 8, 209.

Daecke C, Schaller S, Schaller J, Goos M (1993). Contact urticaria from acrylic acid in Fixomull^{R} tape. Contact Dermatitis 29, 216-217.

DeBethizy JD, Udinsky JR, Scribner HE, Frederick CB (1987). The disposition and metabolism of acrylic acid in male Sprague-Dawley rats. Fund. Appl. Toxicol. 8, 549-561.

DePass LR, Woodside MD, Garman RH, Weil CS (1983). Subchronic and reproductive toxicology studies on acrylic acid in the drinking water of the rat. Drug Chem. Toxicol. 6, 1-20.

Douglas MT (1991). Assessment of ready biodegradability of acrylic acid (Closed bottle test); Report No BMM 1/913229 Huntingdon Research Centre, Ltd., England.

Dow Chemical Company (1979). Toxicology Research Laboratory, unpublished reports, 04.09.1979 and 30.11.1979.

Dow Chemical Co. (1979a). A comparison of single-dose oral LD50's for SPB: (sprague-Dawley) rats and CD F (Fischer 344-Derived) rats. Report No. 8EHQ-0592-3831.

Dow Chemical Co. (1979b). A comparison of single-dose oral LD50's for SPB: (Sprague-Dawley) rats and CD F (Fischer 344-Derived) rats. Report No. 8EHQ-0592-3831.

EC (European Comission) (1996). Technical Guidance Documents for the Risk Assessment of Notified New and Existing Substances.

ECETOC (1995). European Centre for Ecotoxicology and Toxicology of Chemicals. Acrylic Acid. CAS No. 79-10-7. Joint Assessment of Commodity Chemicals No. 34. ECETOC, Brussels.

Elf Atochem (1998). α-β-Diacryloxypropionic acid (DAPA) – Confirmation of absence of DAPA in commercial acrylic acid (complementary study), Carling, 08.06.1998.

Engelhardt G, Klimisch HJ (1983). n-Butyl-acrylate: Cytogenic investigations in the bone marrow Chinese hamsters and rats after 4-day inhalation. Fundam. Appl. Toxicol. 3, 640-641.

Environmental Health Criteria 191 (1997). Acrylic acid, WHO, Genf 1997.

EPDLA (European Polymer Dispersion and Latex Association) (1998). Environmental releases of acrylic acid - Data from polymer dispersion plants. Preliminary Version, 1 page. (30 July 1998).

Fh-ITA (1994). Fraunhofer-Institut für Toxikologie und Aerosolforschung. Micronucleus assay with Methyl Acrylate (unpublished).

Finch L, Frederick CB (1992). Rate and route of oxidation of acrylic acid to carbon dioxide in rat liver. Fundam. Appl. Toxicol. 19, 498-504.

Forbis AD (1989). Acute toxicity of Acrilic acid to Selenastrum capricornutum Printz, Analytical bio-chemistry Laboratories, Inc., Report #37345.

Fowler JF (1990). Immediate contact hypersensitivity to acrylic acid. Dermatologic Clinics 8, 193-195.

Fox M, GibsonT (1990). Mulach R, Sasano T, CEH Marketing research report, SRI International.

Frantz SW et al. (1994). The Toxicologist, 14, 430.

Frederick CB, Bush ML, Lomax LG, Black KA, Finch L, Kimbell JS et al. (1998). Application of a hybrid computational fluid dynamics and physiologically-based inhalation model for interspezies dosimetry extrapolation of acidic vapors in the upper airways. Accepted for publication in Toxicol. Appl. Pharmacol.

Frith CH, Wiley LD (1981). Morphologic classification and correlation of incidence of hyperplastic and neoplastic hematopoietic lesions in mice with age. J. Gerontology 36, 534-545.

Grasso P (1987). Persistent organ damage and cancer production in rats and mice. Arch. Toxicol. Suppl. 11, 75-83.

Lühr HP, Grunder HT, Stein D, Körkemeyer K, Borchardt B (1997). Produkte und Verfahren zur Sanierung von Abwasserkanälen. FB 779, Schriftenreihe der Bundesanstalt für Arbeitsschutz und Arbeitsmedizin,Wirtschaftsverlag, NW, Bremerhaven.

Hachiya N, Taketani A, Takizawa Y (1982). Mutagenicity of environmental substances. Nippon Koshu Eisei Zasshi 29, 236-239.

Hawkins DR, Kirkpatrick D, Aikens PJ, Saxton JE (1992). The metabolism of acrylic acid in soil under aerobic conditions; Report No HRC/R&H 93A/920625. Huntingdon Research Centre Ltd., England.

Hellwig J, Deckardt K, Freisberg KO (1993). Subchronic and chronic studies of the effects of oral administration of acrylic acid to rats. Food Chem. Toxicol. 31, 1-18.

Hellwig J, Gembardt C, Murphy SR (1997). Acrylic acid: Two-generation reproduction toxicity study in Wistar rats with Continuous administration in the drinking water. Food Chem. Toxicol. 35, 859-868.

Henricks-Eckerman ML, Engström B, Anäs (1990). Thermal degradation products of steel protective paints, Am. Ind. Hyg. Assoc. J. 51, 241-244.

Hoechst Celanese Corp. (1992). Potassium acrylate - 60% neutralized solution. Project no. 3859.

Hoechst (1994). Written comments from of Hoechst AG, June 1994.

Hüls (1995). Determination of the surface tension. Unpublished test report (Report No. AN-ASB 0066, 28.06.1995).

Hüls (1995a). Bestimmung der Abbaubarkeit von Acrylsäure im DOC-DIE-AWAY-Test, unveröffentlichte Untersuchung DDA-107.

Hüls (1995b). Bestimmung der akuten Wirkungen von Acrylsäure gegenüber Fischen, unveröffentlichte Untersuchung FK 1333.

Hüls (1995c). Bestimmung der Auswirkungen von Acrylsäure auf das Schwimmverhalten von Daphnia magna, unveröffentlichte Untersuchung DK – 661.

Hüls (1995d). Bestimmung der Auswirkungen von Acrylsäure auf die Reproduktionsrate von Daphnia magna, unveröffentlichte Untersuchung DL - 164.

Hüls (1995e). Bestimmung der Auswirkungen von Acrylsäure auf das Wachstum von Scenedesmus subspicatus, unveröffentlichte Untersuchung AW-413.

ILO (1994). ILO-CIS Chemical Database, Updated Jan. 94, Genf.

Inter-Company Acrylate Study Group (1981). Update (1984).

Inter-Company Acrylate Study Group (1982). Bushy Run Research Center, unpublished report (Project-Report 45-512).

International Research and Development Corporation (1994). Unpublished report, IRDC Study No. 285-053, Draft Report; zusätzlich Report No. 93R-199 (pathologisch-histologische Befunde) vom 22.07.1994.

Ishidate M (1988). Data book of chromosomal aberration test *in vitro*. Elsevier, Amsterdam.

Juhnke I, Lüdemann D (1978). Ergebnisse der Untersuchungen von 200 chemischen Verbindungen auf akute Fischtoxizität mit dem Goldorfebntest: Z. f. Wasser- und Abwasserforsch. 11, 161.

Kliemt G (1995). Arbeitsplätze mit Gefahrstoffbelastung und hohem Frauenanteil. Fb 713. Bundesanstalt für Arbeitsschutz.

Kligerman AD, Atwater AL, Bryant MF, Erexson GL, Kwanyuen P, Dearfield KL (1991). Cytogenetic studies of ethyl acrylate using C57BL/6 mice. Mutagenesis 6, 137-141.

Klimisch HJ, Hellwig J (1991). The prenatal inhalation toxicity of acrylic acid in rats. Fundam. Appl. Toxicol. 16, 656-666.

Kutzman RS, Meyer GJ, Wolf AP (1982). The biodistribution and metabolic fate of [^{11}C] acrylic acid in the rat after acute inhalation exposure or stomach intubation. J. Toxicol. Environ. Health 10, 969-979.

Lomax LG, Brown DW, Frederick CB (1994). Regional histopathology of the mouse nasal cavity following two weeks of exposure to acrylic acid for either 6 or 22 hours per day. The Toxicologist 14, 312.

Majka J, Knobloch K, Sterkiewicz J (1974). Bewertung der akuten und subakuten Toxizität der Acrylsäure (Translation from Polish). Medycyna Pracy. Dwumiesiecznik. ROK XXV - 1974. Panstwowy Zaklad Wydawnictw Lekarskich; 427-435.

McCarthy KL, Thomas WC, Aardema MJ, Seymour JL, Putman DL, Yang LL, Curren RD, Valencia R (1992). Genetic toxicology of acrylic acid. Food Chem. Toxicol. 30, 505-515.

Merck Index (1996). The Merck Index. 12th edition. Merck & Co., Inc., Whitehouse Station, NJ.

Miller RR, Ayres JA, Jersey GC, McKenna MJ (1981a). Inhalation toxicity of acrylic acid. Fund. Appl Toxicol. 1, 271-277.

Miller RR, Ayres JA, Jersey GC, McKenna MJ (1981b). Results of a 90-day acrylic acid vapor inhalation study with rats and mice. The Toxicologist 1, 83.

Miller RR, Ayres JA, Rampy LW, McKenna MJ (1981c). Metabolism of acrylate esters in rat tissue homogenates. Fund. Appl. Toxicol. 1, 410-414.

Ministerie van Sociale Zaken en Werkgelegenheid (1996). De Nationale MAC-lijst. P145, Sdu Uitgevers, Den Haag.

Moore MM, Amtower A, Doerr CL, Brock KH, Dearfield KL (1988). Genotoxicity of acrylic acid, methyl acrylate, ethyl acrylate, methyl methacrylate, and ethyl methacrylate in L5178Y mouse lymphoma cells. Environ. Molec. Mutag. 11, 49-63.

Morita T, Norihide A, Awogi T, Sasaki YF, Sato S, Shimada H, Sutou S, Suzuki T, Wakata A, Sofuni T, Hayashi M (1997). Evaluation of the rodent micronucleus assay in the screening of IARC carcinogens (Groups 1, 2A and 2B). The summary report of the 6th collaborative study by CSGMT/FEMS.MMS. Mutation Res. 389, 3-122.

Neeper-Bradley T, Fowler E, Pritts I, Tyler T (1997). Developmental toxicity study of inhaled acrylic acid in New Zealand White rabbits. Food Chem. Toxicol. 35, 869-880.

Niinimaa et al. (1980). The switching point from nasal to oronasal breathing. Respiratory Physiology 42, 61-67.

Nyholm N (1999). Algal toxicity test reports on acrylic acid (AA), methacrylic acid (MAA), and methyl methacrylate (MMA) - a critical evaluation of selected test reports. Study contracted by the Danish EPA, Study report dated 27-02-1999, 7 pp.

Pahren HR, Bloodgood DE (1961). Biological Oxidation of Several Vinyl Compound, JWPCF 33(3), 233-238.

Przybojewska B, Dziubaltowska E, Kowalski Z (1984). Genotoxic effects of ethyl acrylate and methyl acrylate in the mouse evaluated by the micronucleus test. Mutation Res. 135, 189-191.

Rao KS, Betso E, Olson KJ (1981). A collection of guinea pig sensitization test results - grouped by chemical class. Drug Chem. Toxicol. 4, 331-351.

Ratte HT (1998). Influence of the growth pattern on the EC50 of cell number, biomass integral and growth rate in the algae growth inhibition test. Draft 25-Sep-98.

Reichert JK (1993). Umweltforschungsplan des BMU, Forschungsberichts-Nr. 102 06 223, Untersuchung der organischen Belasung von Abwässern von Galvanikbetrieben und Leiterplattenherstellern, Universität Aachen.

Reininghaus W, Koestner A, Klimisch HJ (1991). Chronic toxicity and oncogenicity of inhaled methyl acrylate and n-butyl acrylate in Sprague-Dawley rats. Food Chem. Toxicol. 29, 329-339.

Rohe D (1995). Märkte und Unternehmen, Acrylsäure, Chemische Industrie 1-2/95, pp 12-13.

Rohm & Haas Co. (1994). Report No. 93R-199, 17.10.1994.

Sadtler NA (1960). Sadtler Standard Spectra, Philadelphia, PA, Sadtler Research Lab., Nr.2994 UV.

Schumann H, Obst K, Friedrich C, Pattard M, Pluta HJ, Hahn J (1997). Umweltverträglichkeit von Chemikalien zur Abwasserbehandlung. UBA-Texte 39/97, Forschungsbericht 102 06 518. ISSN 0722-186X.

Shah JF (1990). A hydrolysis study of 14C-Acrylic Acid, Report No 3196-88-0209-EF-001. Ricerca.

Shelton DR, Tiedje JM (1984). General Method for Determination Anaerobic Biodegradation Potential, Applied and Environmental Microbiology 47(4), 850-857.

SRI (1991). Directory of Chemical Producers, Western Europe: Acrylic Acid, SRI International, 899.

Subramaniam R, Richardson RB, Morgan KT, Kepler G, Kimbell JS, Guilmette RA (1998). Computational fluid dynamics simulations of inspiratory airflow in the human nose and nasopharynx. Inhal. Toxicol. 10, 91-120.

SRI (1996). Acrylic Acid. p. 714 in: 1996/97 Directory of Chemical Producers, Europe. SRI Consulting, Menlo Park, CA.

Swenberg JA, Gross EA, Randall HW (1987). Localization and quantitation of cell proliferation following exposure to nasal irritants. In: Toxicology of the nasal passages. Hemisphere Publishing Corporation, Washington New York, London, pp 291-300.

Tegeris AS, Balmer MF, Garner FM, Thomas WC, Murphy SR, McLaughlin JE, Seymour JL (1988). A 13-week skin irritation study with acrylic acid in 3 strains of mice. The Toxicologist 8, 127.

TEGEWA Polyelectrolyte Producers Group (1999). Information on use of acrylic acid in water treatment agents, including residual monomeric AA concentration and specific emission data provided by producers of flocculent agents. Fax dated 17-02-1999.

TSCATS (1979). OTS 0529492, Old Doc I.D. 41034 B2-37A, New Doc I.D. 44-7229258, Dow Chem. Co.

TSCATS (1986). OTS 0520695, New Doc I.D. 86-890001183, Dow Chem. Co., Toxicology Research Laboratory, Final Report 10.12.1979, 15.10.1986.

TSCATS (1986). OTS 0520853, New Doc I.D. 86-8900013538, Rohm & Haas Co.

TSCATS (1988). OTS 0520855, New Doc I.D. 86-8900013556, Rohm & Haas Co.

TSCATS (1988). OTS 0520856, New Doc I.D. 86-8900013568, Rohm & Haas Co.

TSCATS (1992a). OTS 0510541-2, Old I.D. 8EHQ-0692-0592, New Doc I.D. 89-920000108, Bushy Run Res. Ctr., Supplement.

TSCATS (1992b). OTS 0537283, Old Doc I.D. 8EHQ-0592-3831, New Doc I.D. 88-920002473, Dow Chem.

Tyler TR, Murphy SR, Hunt EK (1993). Health effect assessment of the basic acrylates. Basic Acrylic Monomer Manufacturers Association (BAMM); CRC Press, Boca Raton.

Työterveyslaitos (1998). Finnish Institute for Occupational Health; written communication from 08.12.1998.

Union Carbide, Bushy Run Research Center (1993a). Unpublished report (Project-No. 92N1007), 15.06.1993.

Union Carbide, Bushy Run Research Center (1993b). Unpublished report (Project-No. 92N1008), 24.06.1993.

Vodicka P, Gut I, Vodickova L (1986). Effects of selected derivatives of acrylic acid in six-hour inhalation exposure of rats: elimination of thioethers and modification of glycemia. Prac. Lek. 38, 407-413.

Voullaire E, Kliemt J (1995). Gefahrstoffe in Klein- und Mittelbetrieben: Neue Wege überbetrieblicher Unterstützung; FB 703, Schriftenreihe der Bundesanstalt für Arbeitsschutz und Arbeitsmedizin,Wirtschaftsverlag, NW, Bremerhaven.

Waegemackers THJM, van der Walle HB (1984). alpha,beta-Diacryloxypropionic acid, a sensitizing impurity in commercial acrylic acid. Dermatosen 32, 55-58

Weast RC (ed.) (1988-1989). Handbook of Chemistry and Physics. 69th edition. CRC Press Inc., Boca Raton, FL, C-673.

Wiegand HJ, Schiffmann D, Henschler D (1989). Non-genotoxicity of acrylic acid and n-butyl acrylate in a mammalian cell system (SHE cells). Arch. Toxicol. 63, 250-251.

Winkelmann D et al. (1989). Imaging technology, in: Ullmanns Encyclopedia of Industrial Chemistry, 5th edition, Vol. A 13, VCH Verlagsgesellschaft mbH, Weinheim, 637-642.

Winter SM, Sipes IG (1993). The disposition of acrylic acid in the male Sprague-Dawley rat following oral or topical administration. Food Chem. Toxic. 31, 615-621.

Winter SM, Weber GL, Gooley PR, MacKenzie NE, Sipes IG (1992). Identification and comparison of the urinary metabolites of [1,2,3-$^{13}C_3$]acrylic acid and [1,2,3-$^{13}C_3$]propionic acid in the rat by homonuclear ^{13}C nuclear magnetic resonance spectroscopy. Drug Metab. Dispos. 20, 665-672.

Zeiger E, Anderson B, Haworth S, Lawlor T, Mortelmans K, Speck W (1987). Salmonella Mutagenicity Tests III: Results from the testing of 255 chemicals. Environ. Mutagenesis 9, 1-110.

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 III of Directive 67/548/EEC)
CA	Chromosome Aberration
CA	Competent Authority
CAS	Chemical Abstract Services
CEC	Commission of the European Communities
CEN	European Standards Organisation / European Committee for Normalisation
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 III of Directive 67/548/EEC)

EASE	Estimation and Assessment of Substance Exposure Physico-chemical properties [Model]
EbC50	Effect Concentration measured as 50% reduction in biomass growth in algae tests
EC	European Communities
EC10	Effect Concentration measured as 10% effect
EC50	median Effect Concentration
ECB	European Chemicals Bureau
ECETOC	European Centre for Ecotoxicology and Toxicology of Chemicals
ECVAM	European Centre for the Validation of Alternative Methods
EDC	Endocrine Disrupting Chemical
EEC	European Economic Communities
EINECS	European Inventory of Existing Commercial Chemical Substances
ELINCS	European List of New Chemical Substances
EN	European Norm
EPA	Environmental Protection Agency (USA)
ErC50	Effect Concentration measured as 50% reduction in growth rate in algae tests
ESD	Emission Scenario Document
EU	European Union
EUSES	European Union System for the Evaluation of Substances [software tool in support of the Technical Guidance Document on risk assessment]
F(+)	(Highly) flammable (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
FAO	Food and Agriculture Organisation of the United Nations
FELS	Fish Early Life Stage
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 t/a)
IARC	International Agency for Research on Cancer
IC	Industrial Category
IC50	median Immobilisation Concentration or median Inhibitory Concentration
ILO	International Labour Organisation
IPCS	International Programme on Chemical Safety
ISO	International Organisation for Standardisation
IUCLID	International Uniform Chemical Information Database (existing substances)
IUPAC	International Union for Pure and Applied Chemistry
JEFCA	Joint FAO/WHO Expert Committee on Food Additives

JMPR	Joint FAO/WHO Meeting on Pesticide Residues
Koc	organic carbon normalised distribution coefficient
Kow	octanol/water partition coefficient
Kp	solids-water partition coefficient
L(E)C50	median Lethal (Effect) Concentration
LAEL	Lowest Adverse Effect Level
LC50	median Lethal Concentration
LD50	median Lethal Dose
LEV	Local Exhaust Ventilation
LLNA	Local Lymph Node Assay
LOAEL	Lowest Observed Adverse Effect Level
LOEC	Lowest Observed Effect Concentration
LOED	Lowest Observed Effect Dose
LOEL	Lowest Observed Effect Level
MAC	Maximum Allowable Concentration
MATC	Maximum Acceptable Toxic Concentration
MC	Main Category
MITI	Ministry of International Trade and Industry, Japan
MOE	Margin of Exposure
MOS	Margin of Safety
MW	Molecular Weight
N	Dangerous for the environment (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC
NAEL	No Adverse Effect Level
NOAEL	No Observed Adverse Effect Level
NOEL	No Observed Effect Level
NOEC	No Observed Effect Concentration
NTP	National Toxicology Program (USA)
O	Oxidizing (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
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 III of Directive 67/548/EEC
SAR	Structure-Activity Relationships
SBR	Standardised birth ratio
SCE	Sister Chromatic Exchange
SDS	Safety Data Sheet
SETAC	Society of Environmental Toxicology And Chemistry
SNIF	Summary Notification Interchange Format (new substances)
SSD	Species Sensitivity Distribution
STP	Sewage Treatment Plant
T(+)	(Very) Toxic (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
TDI	Tolerable Daily Intake
TG	Test Guideline
TGD	Technical Guidance Document
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 Organization
WWTP	Waste Water Treatment Plant
Xn	Harmful (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)
Xi	Irritant (Symbols and indications of danger for dangerous substances and preparations according to Annex III of Directive 67/548/EEC)

Appendix A1 Distribution and fate

Substance: Acrylic Acid

melting point: $MP := 286 \cdot K$

vapour pressure: $VP := 380 \cdot Pa$

water solubility: $SOL := 1000000 mg \cdot l^{-1}$

part. coefficient octanol/water: $LOGP_{OW} := 0.46$

molecular weight: $MOLW := 0.07206 kg \cdot mol^{-1}$

gas constant: $R := 8.3143 J \cdot mol \cdot K^{-1}$

temperature: $T := 293 \cdot K$

conc. of suspended matter in the river: $SUSP_{water} := 15 \cdot mg \cdot l^{-1}$

density of the solid phase: $RHO_{solid} := 2500 kg \cdot m^{-3}$

volume fraction water in susp. matter: $Fwater_{susp} := 0.9$

volume fraction solids in susp.matter: $Fsolid_{susp} := 0.1$

volume fraction of water in sediment: $Fwater_{sed} := 0.8$

volume fraction of solids in sediment: $Fsolid_{sed} := 0.2$

volume fraction of air in soil: $Fair_{soil} := 0.2$

volume fraction of water in soil: $Fwater_{soil} := 0.2$

volume fraction of solids in soil: $Fsolid_{soil} := 0.6$

aerobic fraction of the sediment comp.: $Faer_{sed} := 0.1$

product of CONjunge and $SURF_{air}$: $product := 10^{-4} \cdot Pa$

distribution air/water: Henry-constant

$$HENRY := \frac{VP \cdot MOLW}{SOL} \qquad HENRY = 0.027 \cdot Pa \cdot m^3 \cdot mol^{-1}$$

$$\log\left(\frac{HENRY}{Pa \cdot m^3 \cdot mol^{-1}}\right) = -1.563$$

$$K_{air\_water} := \frac{HENRY}{R \cdot T} \qquad K_{air\_water} = 1.124 \cdot 10^{-5}$$

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

$a := 0.52$ (a,b from TGD, p. 539)

$b := 1.02$ $K_{OC} := 10^{a \cdot LOGP_{OW} + b} \cdot l \cdot kg^{-1}$ $K_{OC} = 18.164 \cdot l \cdot kg^{-1}$

Suspended matter

$Kp_{susp} := 1 \cdot l \cdot kg^{-1}$

$K_{susp\_water} := Fwater_{susp} + Fsolid_{susp} \cdot Kp_{susp} \cdot RHO_{solid}$ $K_{susp\_water} = 1.15$

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

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

Sediment

$Kp_{sed} := 1 \cdot l \cdot kg^{-1}$

$K_{sed\_water} := Fwater_{sed} + Fsolid_{sed} \cdot Kp_{sed} \cdot RHO_{solid}$ $K_{sed\_water} = 1.3$

Sludge

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

Soil

$Kp_{soil} := 1 \cdot l \cdot kg^{-1}$

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

$K_{soil\_water} = 1.7$

Elimination in STPs

rate constant in STP: k = 1 d^{-1} elimination P = f (k, logpow, logH) = 87.3

fraction directed to surface water $Fstp_{water}$ = 12.7

biodegradation in different compartments

surface water

$kbio_{water} := 4.7 \cdot 10^{-2} \cdot d^{-1}$ (cTGD, table 5)

soil

$DT50bio_{soil} := 15 \cdot d$ (see RAR)

$kbio_{soil} := \frac{\ln(2)}{DT50bio_{soil}}$ $kbio_{soil} := 0.047 \cdot d^{-1}$

sediment

$kbio_{sed} := \frac{\ln(2)}{DT50bio_{soil}} \cdot Faer_{sed}$ $kbio_{sed} = 0.005 \cdot d^{-1}$

degradation in surface waters

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

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

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

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

Atmosphere

calculation of CONjunge * SURFaer for the OPS-model

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

$VP := wenn(MP > 285 \cdot K, VPL, VP)$ $VP = 389.162 \cdot Pa$

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

$Fass_{aer} = 2.57 \cdot 10^{-7}$

degradation in the atmosphere

$kdeg_{air}$ = 0.0175 h^{-1} (see AOP-calculation)

Appendix A2 Calculation of PEC_{local} for aquatic compartment during production and processing of chemicals at one site

Status: TGD, ESD, IC-3

chemical: Acrylic Acid, generic scenario for production/processing

Production volume: $T_1 := 330000\,t \cdot a^{-1}$

Processing volume: $T_2 := 330000\,t \cdot a^{-1}$

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

Emission faktor for processing (TGD, tab. A3.3): $f_2 := 0.7\,\%$

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

Duration of emission for processing (TGD, tab. B3.2): $Temission_2 := 300\,d \cdot a^{-1}$

Fraction of emission directed to water:
(SimpleTreat, k: 1 h-1; logH:-1.56 ; $logK_{ow}$: 0.46) $Fstp_{water} := 12.7\,\%$

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

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

Emission per day:

$$Elocal_{water} := \frac{T_1 \cdot f_1}{Temission_1} + \frac{T_2 \cdot f_2}{Temission_2} \qquad Elocal_{water} = 1.1 \cdot 10^4 \ kg \cdot d^{-1}$$

Concentration in surface water:

$$Clocal_{water} := \frac{Elocal_{water} \cdot Fstp_{water}}{V \cdot FACTOR} \qquad Clocal_{water} = 269.5 \ \mu g \cdot l^{-1}$$

Appendix A3 Default exposure estimation of $Clocal_{water}$

Status: TGD, Tables A and B

stage of life cycle: default processing (polymerisation), wet process
IC/UC/MC:11/33/III

Total annual tonnage of chemical: $TONNAGE := 10000\,t \cdot a^{-1}$

Release factor (A-table: A3.10): $f_{emission} := 0.01$

Fraction of main source (B-table: B3.9): $Fmainsource := 1$

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

Duration of emission (B-table: B3.9): $Temission := 300\,d \cdot a^{-1}$

Fraction of emission directed to water:
(SimpleTreat; k: 1 h^{-1}; logPow: 1.38; logH:1.4) $Fstp_{water} := 12.7\,\%$

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

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

Emission per day:

$$Elocal_{water} := \frac{TONNAGE \cdot Fmainsource \cdot f_{emission}}{Temission} \qquad Elocal_{water} = 333.33\,kg \cdot d^{-1}$$

Influent concentration:

$$Clocal_{inf} := \frac{Elocal_{water}}{EFFLUENT_{stp}} \qquad Clocal_{inf} = 1.67 \cdot 10^{5}\ \mu g \cdot l^{-1}$$

Effluent concentration:

$$Clocal_{eff} := Clocal_{inf} \cdot Fstp_{water} \qquad Clocal_{eff} = 2.12 \cdot 10^{4}\ \mu g \cdot l^{-1}$$

Concentration in surface water:

$$Clocal_{water} := \frac{Clocal_{eff}}{FACTOR \cdot DILUTION} \qquad Clocal_{water} = 2.12 \cdot 10^{3}\ \mu g \cdot l^{-1}$$

Annual average local concentration in water:

$$Clocal_{water\_ann} := Clocal_{water} \cdot \frac{Temission}{365\,d \cdot a^{-1}} \qquad Clocal_{water\_ann} = 1.74 \cdot 10^{3}\ \mu g \cdot l^{-1}$$

Appendix A4 Calculation of PEC_{local} for the aquatic compartment - leather production industry

Status: TGD, ESD, IC-7

Mass of processed goods per day:	$W1 := 15 \cdot t \cdot d^{-1}$	$d := 86400\,s$
		$a := 365 \cdot d$
mass of AA-monomer used per mass of good: (see RAR)	$W2 := 0.2 \cdot kg \cdot t^{-1}$	$\mu g := 10^{-9} \cdot kg$
Degree of 'fixation':	$F := 95 \cdot \%$	
Participation factor on production per day	$A := 100 \cdot \%$	
Waste water flow of wwtp:	$EFFLUENT_{stp} := 2000 \cdot m^3 \cdot d^{-1}$	
Fraction of emission directed to water:	$Fstp_{water} := 12.7 \cdot \%$	
Dilution factor (TGD):	$DILUTION := 10$	
Factor (1+Kp*SUSPwater):	$FACTOR := 1$	

Emission per day:

$$Elocal_{water} := W1 \cdot W2 \cdot (1 - F) \cdot A \qquad Elocal_{water} = 0.15 \bullet kg \cdot d^{-1}$$

Influent concentration:

$$Clocal_{inf} := \frac{Elocal_{water}}{EFFLUENT_{stp}} \qquad Clocal_{inf} = 0.07 \bullet mg \cdot l^{-1}$$

Effluent concentration:

$$Clocal_{eff} := Clocal_{inf} \cdot Fstp_{water} \qquad Clocal_{eff} = 0.00952 \bullet mg \cdot l^{-1}$$

Concentration in surface water:

$$Clocal_{water} := \frac{Clocal_{eff}}{DILUTION \cdot FACTOR} \qquad Clocal_{water} = 0.952 \bullet \mu g \cdot l^{-1}$$

$Clocal_{water}$=PEC_{local} for $PEC_{regional}$=0

Appendix A5 Calculation of PEC_{local} for aquatic compartment for textile finishing industry

Status: TGD, ESD, IC-13

Mass of good processed per day: $W1 := 3 \cdot t \cdot d^{-1}$ $\mu g := 10^{-9} \cdot kg$

Mass of substance used per mass of good: $W2 := 0.2 \cdot kg \cdot t^{-1}$

Degree of fixation: $F := 95 \cdot \%$

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

Fraction of emission directed to water: $Fstp_{water} := 12.7 \cdot \%$

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

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

Emission per day:

$Elocal_{water} := W1 \cdot W2 \cdot (1 - F)$ $Elocal_{water} = 0.03 \bullet kg \cdot d^{-1}$

Influent concentration:

$Clocal_{inf} := \frac{Elocal_{water}}{EFFLUENT_{stp}}$ $Clocal_{inf} = 0.015 \bullet mg \cdot l^{-1}$

Effluent concentration:

$Clocal_{eff} := Clocal_{inf} \cdot Fstp_{water}$ $Clocal_{eff} = 0.002 \bullet mg \cdot l^{-1}$

Concentration in surface water :

$Clocal_{water} := Clocal_{eff} \cdot DILUTION^{-1} \cdot FACTOR^{-1}$ $Clocal_{water} = 0.19 \bullet \mu g \cdot l^{-1}$

Appendix A6 Default calculation of PEC_{local} for the hydrosphere

Status: TGD for Existing Substances / EUSES

AA

formulation of paints

tables: A2.1, B2.3

tonnage: $T := 8 \cdot tonne \cdot a^{-1}$

release factor (A2.1): $r := 0.003$

fraction of main source (B2.3): $f := 1$

waste water flow of the WWTP: $Q := 2000 \cdot m^3 \cdot d^{-1}$

number of days for releases: $d := 300 \cdot d \cdot a^{-1}$

$$C_{inf} := \frac{T \cdot r \cdot f}{Q \cdot d} \qquad C_{inf} = 0.04 \cdot mg \cdot l^{-1}$$

Elimination in WWTP related to SIMPLETREAT:

$P := 87.3 \cdot \%$ P = f (biodegradation, log pow, log H)

$$C_{eff} := C_{inf}(1 - P) \qquad C_{eff} = 5.08 \cdot 10^{-3} \cdot mg \cdot l^{-1}$$

Calculation of C-local:

partition coefficient for susp.matter: $K_{p\_susp} := 1 \cdot kg^{-1} \cdot l$

concentration of suspended matter: $c_{susp} := 15 \cdot mg \cdot l^{-1}$

dilution factor for receiving surface water: $D := 10$

$$C_{local} := \frac{C_{eff}}{\left(1 + K_{p\_susp} \cdot c_{susp}\right) \cdot D}$$

$$C_{local} = 0.508 \cdot \mu g \cdot l^{-1}$$

Appendix A7 Atmosphere (OPS model) - generic approach

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

(generic approach)

$d := 24 \cdot h$

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 release at the local main source at life cycle stage:

$Fmainsource := 1$

number of days per year for the emission:

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

release to air during life cycle stage: (330000 * 0.00011)

$RELEASE := 36.3 \cdot t \cdot a^{-1}$

local emission to air:

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

$$Elocal_{air} = 121 \cdot kg \cdot d^{-1}$$

fraction of the emission to air from STP:

$Fstp_{air} := 0$

local emission rate to STP during emission episode:

$Elocal_{water} := 11 \cdot t \cdot d^{-1}$

local emission to air from STP during emission episode:

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

$$Estp_{air} = 0 \cdot kg \cdot d^{-1}$$

local concentation in air during emission episode:

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

$$Clocal_{air} = 0.034 \cdot mg \cdot m^{-3}$$

annual average concentration in air, 100m from point source:

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

$$Clocal_{air\_ann} = 0.028 \cdot mg \cdot m^{-3}$$

regional concentration in air:

$$PECregional_{air} := 0 \cdot mg \cdot m^{-3}$$

annual average predicted environmental concentration in air.

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

$$PEClocal_{air\_ann} = 0.028 \bullet mg \cdot m^{-3}$$

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

$$Fass_{aer} := 2.6 \cdot 10^{-7}$$

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 $mg*m^{-2}*d^{-1}$

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

total deposition flux during emission episode:

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

$$DEPtotal = 0.048 \bullet mg \cdot m^{-2} \cdot d^{-1}$$

annual average total depostion flux:

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

$$DEPtotal_{ann} = 0.04 \bullet mg \cdot m^{-2} \cdot d^{-1}$$

Appendix A8 Atmosphere (OPS-model) - external processing (wet and dry polymerisation)

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

$d := 24 \cdot h$

External processing (wet and dry polymerisation)

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 release at the local main source at life cycle stage:

$Fmainsource := 1$

number of days per year for the emission:

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

release to air during life cycle stage: (10000 * 0.001)

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

local emission 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:

$Fstp_{air} := 0$

local emission rate to STP during emission episode:

$Elocal_{water} := 3300 \cdot tonne \cdot d^{-1}$

local emission to air from STP during emission episode:

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

$Estp_{air} = 0 \cdot kg \cdot d^{-1}$

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:

$$PECregional_{air} := 0 \cdot mg \cdot m^{-3}$$

annual average predicted environmental concentration in air.

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

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

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

$$Fass_{aer} := 2.6 \cdot 10^{-7}$$

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}

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

total deposition flux during emission episode:

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

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

annual average total depostion flux:

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

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

Appendix A9 Exposure of soil

Chemical: Acrylic acid, generic production and processing

Input:

annual average total deposition flux: $DEPtotal_{ann} := 0.040 \cdot mg \cdot m^{-2} \cdot d^{-1}$

soil-water partitioning coefficient: $K_{soil\_water} := 1.7$

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

air-water partitioning coefficient: $K_{air\_water} := 1.1 \cdot 10^{-5}$

rate constant for for removal from top soil: $kbio_{soil} := 0.047 \cdot d^{-1}$

PECregional: $PECregional_{natural\_soil} := 0 \cdot mg \cdot kg^{-1}$

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:

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

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

	$\frac{Clocal_{soil_i}}{ppm}$
$Clocal_{soil}$ =	0.0024
$Clocal_{agr.soil}$ =	0.0024
$Clocal_{grassland}$ =	0.0047

	$\frac{PEClocal_{soil_i}}{ppm}$
$PEClocal_{soil}$ =	0.00242
$PEClocal_{agr.soil}$ =	0.00242
$PEClocal_{grassland}$ =	0.00469

Indicating persistency of the substance in soil

initial concentration after 10 years:

$\frac{C_{soil\_10_i}}{ppm}$
0.00242
0.00242
0.00469

initial concentration in steady-state situation:

$$C_{soil\_ss_i} := \frac{D_{air_i}}{k_i} + Csludge_{soil\_1_i} \cdot \left(\frac{1}{1 - \exp\left(365 \cdot d \cdot k_i \right)} \right)$$

$\frac{C_{soil\_ss_i}}{ppm}$
0.00242
0.00242
0.00469

fraction of steady-state in soil achieved:

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

$\overline{Fst\_st_i}$

concentration in pore water

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

	$\frac{Clocal_{soil\_porew_i}}{mg \cdot l^{-1}}$
$Clocal_{soil\_porew}$ =	0.00242
$Clocal_{agr.soil\_porew}$ =	0.00242
$Clocal_{grassland\_porew}$ =	0.00469

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

	$\frac{PEClocal_{soil\_porew_i}}{mg \cdot l^{-1}}$
$PEClocal_{soil\_porew}$ =	0.00242
$PEClocal_{agr.soil\_porew}$ =	0.00242
$PEClocal_{grassland\_porew}$ =	0.00469

concentration in ground water

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

Appendix A10 Calculation of continental and regional PECs

SimpleBox2.0a - calculation of continental and regional PECs

- adaptation to TGD (1996) / Umweltbundesamt (06/98)

INPUT - AA

Parameter names acc. SimpleBox20	Unit	Input	Parameter names according Euses
Physicochemical properties			
COMPOUND NAME	[-]	AA	Substance
MOL WEIGHT	[g.mol^{-1}]	72,06	Molecular weight
MELTING POINT	[° C]	13	Melting Point
VAPOUR PRESSURE(25)	[Pa]	380	Vapour pressure at 25°C
log Kow	[log10]	0,46	Octanol-water partition coefficient
SOLUBILITY(25)	[mg.l^{-1}]	1,000,000	Water solubility
Distribution - Partition coefficients			
- Solids water partitioning (derived from K_{oc})			
Kp(soil)	[l.kg$_d^{-1}$]	1	Solids-water partitioning in soil
Kp(sed)	[l.kg$_d^{-1}$]	1	Solids-water partitioning in sediment
Kp(susp)	[l.kg$_d^{-1}$]	1	Solids-water partitioning in suspended matter
- Biota-water			
BCF(fish)	[l.kg$_w^{-1}$]	0,49	Bioconcentration factor for aquatic biota
Degradation and Transfromation rates			
- Characterisation and STP			
PASSreadytest	[y / n]	y	Characterisation of biodegradability
- Environmental <u>Total</u> Degradation			
kdeg(air)	[d^{-1}]	$4{,}20 \cdot 10^{-01}$	Rate constant for degradation in air
kdeg(water)	[d^{-1}]	$4{,}70 \cdot 10^{-02}$	Rate constant for degradation in bulk surface water
kdeg(soil)	[d^{-1}]	$4{,}70 \cdot 10^{-02}$	Rate constant for degradation in bulk soil
kdeg(sed)	[d^{-1}]	$2{,}30 \cdot 10^{-03}$	Rate constant for degradation in bulk sediment
Sewage treatment (e.g. calculated by SimpleTreat)			
- Continental			
FR(volatstp) [C]	[-]	$0{,}00 \cdot 10^{+00}$	Fraction of emission directed to air (STPcont)
FR(effstp) [C]	[-]	$1{,}27 \cdot 10^{-01}$	Fraction of emission directed to water (STPcont)
FR(sludgestp) [C]	[-]	$0{,}00 \cdot 10^{+00}$	Fraction of emission directed to sludge (STPcont)
- Regional			
FR(volatstp) [R]	[-]	$0{,}00 \cdot 10^{+00}$	Fraction of emission directed to air (STPreg)
FR(effstp) [R]	[-]	$1{,}27 \cdot 10^{-01}$	Fraction of emission directed to water (STPreg)
FR(sludgestp) [R]	[-]	$0{,}00 \cdot 10^{+00}$	Fraction of emission directed to sludge (STPreg)

Release estimation			
- Continental			
Edirect(air) [C]	[t.y^{-1}]	277	Total continental emission to air
STPload [C]	[t.y^{-1}]	973	Total continental emission to wastewater
Edirect(water1) [C]	[t.y^{-1}]	77,4	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}]	54	Total continental emission to air
STPload [R]	[t.y^{-1}]	218	Total continental emission to wastewater
Edirect(water1) [R]	[t.y^{-1}]	8,6	Total continental emission to surface water
Edirect(soil3) [R]	[t.y^{-1}]	0	Total continental emission to industrial soil
Edirect(soil2) [R]	[t.y^{-1}]	0	Total continental emission to agricultural soil

OUTPUT - AA

Parameter names acc. SimpleBox20	Unit	Output	Parameter names according Euses
Physicochemical properties			
COMPOUND NAME	[-]	AA	Substance
Output			
- Continental			
PECsurfacewater (total)	[mg.l^{-1}]	$3{,}39 \cdot 10^{-05}$	Continental PEC in surface water (total)
PECsurfacewater (dissolved)	[mg.l^{-1}]	$3{,}39 \cdot 10^{-05}$	Continental PEC in surface water (dissolved)
PECair	[mg.m^{-3}]	$3{,}28 \cdot 10^{-07}$	Continental PEC in air (total)
PECagr.soil	[mg.kg$_{wwt}^{-1}$]	$3{,}29 \cdot 10^{-06}$	Continental PEC in agricultural soil (total)
PECporewater agr.soil	[mg.l^{-1}]	$3{,}29 \cdot 10^{-06}$	Continental PEC in pore water of agricultural soils
PECnat.soil	[mg.kg$_{wwt}^{-1}$]	$1{,}12 \cdot 10^{-05}$	Continental PEC in natural soil (total)
PECind.soil	[mg.kg$_{wwt}^{-1}$]	$1{,}12 \cdot 10^{-05}$	Continental PEC in industrial soil (total)
PECsediment	[mg.kg$_{wwt}^{-1}$]	$3{,}15 \cdot 10^{-05}$	Continental PEC in sediment (total)
- Regional			
PECsurfacewater (total)	[mg.l^{-1}]	$3{,}96 \cdot 10^{-04}$	Regional PEC in surface water (total)
PECsurfacewater (dissolved)	[mg.l^{-1}]	$3{,}96 \cdot 10^{-04}$	Regional PEC in surface water (dissolved)
PECair	[mg.m^{-3}]	$2{,}04 \cdot 10^{-06}$	Regional PEC in air (total)
PECagr.soil	[mg.kg$_{wwt}^{-1}$]	$2{,}05 \cdot 10^{-05}$	Regional PEC in agricultural soil (total)
PECporewater agr.soil	[mg.l^{-1}]	$2{,}05 \cdot 10^{-05}$	Regional PEC in pore water of agricultural soils
PECnat.soil	[mg.kg$_{wwt}^{-1}$]	$6{,}95 \cdot 10^{-05}$	Regional PEC in natural soil (total)
PECind.soil	[mg.kg$_{wwt}^{-1}$]	$6{,}95 \cdot 10^{-05}$	Regional PEC in industrial soil (total)
PECsediment	[mg.kg$_{wwt}^{-1}$]	$3{,}73 \cdot 10^{-04}$	Regional PEC in sediment (total)

Appendix A11 Indirect exposure via the environment

(TGD, Chapter 2)

Input

chemical properties

	$\log K_{OW} := 0.46$
octanol-water partitioning coefficient [-]	$K_{OW} := 10^{\log K_{OW}}$
Henry - partitioning coefficient [Pa*m^3*mol^{-1}]	$HENRY := 0.027 \cdot Pa \cdot m^3 \cdot mol^{-1}$
air-water partitioning coefficient [-]	$K_{air\_water} := 1.12 \cdot 10^{-5}$
fraction of the chemical associated with aerosol particles [-]	$F_{ass\_aer} := 2.6 \cdot 10^{-7}$
half-life for biodegration in surface water [d]	$DT_{50\_bio\_water} := 15 \cdot d$

environmental concentrations

annual average local PEC in surface water (dissolved) [mg$_{chem}$ * l$_{water}^{-1}$]	$PEClocal_{water\_ann} := 270 \cdot \mu g \cdot l^{-1} \cdot \frac{300}{365}$
annual average local PEC in air (total) [mg$_{chem}$ * m$_{air}^{-3}$]	$PEClocal_{air\_ann} := 28 \cdot \mu g \cdot m^{-3}$
local PEC in grassland (total), averaged over 180 days [mg$_{chem}$ * kg$_{soil}^{-1}$]	$PEClocal_{grassland} := 0.00469 mg \cdot kg^{-1}$
local PEC in porewater of agriculture soil [mg$_{chem}$ * l$_{porewater}^{-1}$]	$PEClocal_{agr\_soil\_porew} := 0.00242 mg \cdot l^{-1}$
local PEC in porewater of grassland [mg$_{chem}$ * l$_{porewater}^{-1}$]	$PEClocal_{grassland\_porew} := 0.00469 mg \cdot l^{-1}$
local PEC in groundwater under agriculture soil [mg$_{chem}$ * l$_{water}^{-1}$]	$PEClocal_{grw} := 0.00242 mg \cdot l^{-1}$
regional PEC in surface water (dissolved) [mg$_{chem}$ * l$_{water}^{-1}$]	$PECregional_{water} := 0.396 \mu g \cdot l^{-1}$
regional PEC in air (total) [mg$_{chem}$ * m$_{air}^{-3}$]	$PECregional_{air} := 2 \cdot 10^{-3} \cdot \mu g \cdot m^{-3}$
regional PEC in agriculture soil (total) [mg$_{chem}$*kg$_{soil}^{-1}$	$PECregional_{agr\_soil} := 0.02 \cdot \mu g \cdot kg^{-1}$
regional PEC in porewater of agriculture soils [mg$_{chem}$*l$_{water}^{-1}$	$PECregional_{agr\_soil\_porew} := 0.02 \cdot \mu g \cdot l^{-1}$

Definition of the concentrations used for indirect exposure

$C_{water_{local}} := PEClocal_{water\_ann}$

$C_{air_{local}} := PEClocal_{air\_ann}$

$C_{grassland_{local}} := PEClocal_{grassland}$

$C_{agr\_porew_{local}} := PEClocal_{agr\_soil\_porew}$

$C_{grass\_porew_{local}} := PEClocal_{grassland\_porew}$

$C_{grw_{local}} := PEClocal_{grw}$

$C_{water_{regional}} := PECregional_{water}$

$C_{air_{regional}} := PECregional_{air}$

$C_{grassland_{regional}} := PECregional_{agr\_soil}$

$C_{agr\_porew_{regional}} := PECregional_{agr\_soil\_porew}$

$C_{grass\_porew_{regional}} := PECregional_{agr\_soil\_porew}$

$C_{grw_{regional}} := PECregional_{agr\_soil\_porew}$

Results of calculation

$DOSE_{tot_{local}} = 0.05 \circ \frac{mg}{kg_{bw} \cdot d}$

$RDOSE_{drw_{local}} = 11.83 \circ \%$

$RDOSE_{air_{local}} = 11.19 \circ \%$

$RDOSE_{stem_{local}} = 76.6 \circ \%$

$RDOSE_{root_{local}} = 0.02 \circ \%$

$RDOSE_{meat_{local}} = 1.13 \bullet 10^{-3} \circ \%$

$RDOSE_{milk_{local}} = 0.02 \circ \%$

$RDOSE_{fish_{local}} = 0.33 \circ \%$

$DOSE_{tot_{regional}} = 1.51 \bullet 10^{-5} \circ \frac{mg}{kg_{bw} \cdot d}$

$RDOSE_{drw_{regional}} = 74.78 \circ \%$

$RDOSE_{air_{regional}} = 2.83 \circ \%$

$RDOSE_{stem_{regional}} = 19.56 \circ \%$

$RDOSE_{root_{regional}} = 0.7 \circ \%$

$RDOSE_{meat_{regional}} = 7.61 \bullet 10^{-4} \circ \%$

$RDOSE_{milk_{regional}} = 0.01 \circ \%$

$RDOSE_{fish_{regional}} = 2.11 \circ \%$

Appendix B1 Simulation of consumer exposure to UV-hardening adhesives

Substance:
Acrylic acid

CAS:
79-10-7

Computer model used:
EPA-model: SCIES
(Screening-Level Consumer Inhalation Exposure Software)

Category of consumer products:
UV-hardening adhesives

Results:
User potential dose rate from inhalation:
7.70 Milligramm/yr
= 13.0 Mikrogramm/day
= 216 Nanogramm/kg b.w. and day

Generic Product

Annual Frequency of Use:	4 Events/Year
Mass of Product:	1.000 grams
Duration of Use:	1.000 Hours
Zone 1 Volume:	40.000 cubic meters
Whole House Volume:	292.000 cubic meters
House Air Exchange Rate:	0.200 room air exchanges/hr
User Inhalation Rate:	1.300 cubic meters/hr (during use)
Non-User Inhalation Rate:	1.100 cubic meters/hr (& User after use)
Molecular Weight:	72.060 g/mole
Vapour Pressure:	7.725E+00 torr
Weight Fraction:	0.060
Starting Time:	12:00 NOON

OUTPUT SUMMARY

Evaporation Time:	0.347 Hours
Release Time:	1.000 Hours (Duration of Exposure)
Duration Following Each Use:	2189.000 Hours
Interval Between Uses:	2190.000 Hours
User Potential Dose Rate From Inhalation:	4.706 mg/yr
Non-User Potential Dose Rate From Inhalation:	2.937 mg/yr

	Average (mg/m^3)	Peak (mg/m^3)
Concentration in zone of release		
During period of use	0.384	0.542
During period after use	0.000	0.448
Concentration in Zone 2		
During period of use	0.052	0.119
During period after use	0.000	0.154
Concentration to which User and Non-User are exposed		
Person Using Product (user)	0.000	0.542
Person Not Using Product (non-user)	0.000	0.154

HOURLY ACTIVITY PATTERN

User:	111111234542467422744411
Non-User:	111111132442476422644411
Hour:	03 06 09 15 18 21 24

START HOUR

European Commission

EUR 19836 EN **European Union Risk Assessment Report acrylic acid, Volume 28**

Editors: B.G. Hansen, S.J. Munn, C. Musset, M. Luotamo, S. Pakalin, J. de Bruijn, F. Berthault, S. Vegro, G. Pellegrini, R. Allanou, S. Scheer.

Luxembourg: Office for Official Publications of the European Communities

2002 – VIII pp., 126 pp. – 17.0 x 24.0 cm

Environment and quality of life series

ISBN 92-894-1272-0

Price (excluding VAT) in Luxembourg: EUR 20.50

The report provides the comprehensive risk assessment of the substance acrylic acid. It has been prepared by Germany in the frame of Council Regulation (EEC) No. 793/93 on the evaluation and control of the risks of existing substances, following the principles for assessment of the risks to man and the environment, laid down in Commission Regulation (EC) No. 1488/94.

The evaluation considers the emissions and the resulting exposure to the environment and 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 acrylic acid concludes that there is a concern for workers. For consumers and human exposed via the environment the risk assessment concludes that there is at present no concern.

The risk assessment for the environment concludes that there is concern for the aquatic ecosystem, and a need for further information on risks for microorganisms in the sewage treatment plant. The information requirement is postponed since risk reduction measures to protect surface water will also protect municipal wastewater treatment plants.

The conclusions of this report will lead to risk reduction measures to be proposed by the Commission's Committee on risk reduction strategies set up in support of Council Regulation (EEC) 793/93.

14

LB-NA-19836-EN-C

The mission of the JRC is to provide customer-driven scientific and technical support for the conception, development, implementation and monitoring of EU policies. As a service of the European Commission, the JRC functions as a reference centre of science and technology for the Union. Close to the policy-making process, it serves the common interest of the Member States, while being independent of special interests, private or national.

European Commission – Joint Research Centre
Institute for Health and Consumer Protection
European Chemicals Bureau (ECB)

European Union Risk Assessment Report

acrylic acid

CAS No: 79-10-7 EINECS No: 201-177-9

Series: 1st Priority List Volume: 28

Price (excluding VAT) in Luxembourg: EUR 20.50

OFFICE FOR OFFICIAL PUBLICATIONS
OF THE EUROPEAN COMMUNITIES
L – 2985 Luxembourg

ISBN 92-894-1272-0

9 789289 412728