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ACRYLIC ACID

CAS No: 79-10-7

EINECS No: 201-177-9

Summary Risk Assessment Report

2002 **Special Publication I.01.71**

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SUMMARY RISK ASSESSMENT REPORT

Final report, 2002

Germany

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

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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).

PREFACE

This report provides a summary, with conclusions, of the risk assessment report of the substance acrylic acid that has been prepared by Germany in the context of Council Regulation (EEC) No. 793/93 on the evaluation and control of existing substances.

For detailed information on the risk assessment principles and procedures followed, the underlying data and the literature references the reader is referred to the comprehensive Final Risk Assessment Report (Final RAR) that can be obtained from the European Chemicals Bureau[1]. The Final RAR should be used for citation purposes rather than this present Summary Report.

[1] European Chemicals Bureau – Existing Chemicals – http://ecb.jrc.it

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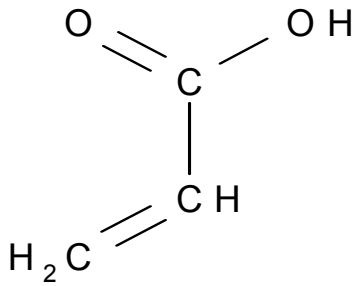
1 GENERAL SUBSTANCE INFORMATION

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
	0.02% w/w MEHQ as stabilizer in commercial Acrylic Acid

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) [2)]
Boiling point	141°C at 1,013 hPa	Merck Index (1996) [2)]
Density	1.0621 g/cm3 at 20°C	Merck Index (1996) [2)]
Vapour pressure	3.8 hPa at 20°C - (dynamic method)	BASF AG (1994a) [2)]
Surface tension	59.6 mN/m c=1g/l - (ring method)	Hüls AG (1995) [2)]
Water solubility	miscible in all ratios	Merck Index (1996) [2)]
Dissociation constant	pKa = 4.25	Weast (1989) [2)]
Partition coefficient	log Pow 0.46 at 25°C - (shake flask method)	BASF AG (1988) [2)]
Flash point	48-55°C	CHEMSAFE [2)]
Auto flammability	395°C - DIN 51794	CHEMSAFE [2)]
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
2) For references, see the comprehensive Final Risk Assessment Report that can be obtained from the European Chemicals Bureau: http://ecb.jrc.it

1.4 CLASSIFICATION

Classification and labelling according to the 28th ATP of Directive 67/548/EEC[2]:

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	

[2] 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).

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

2 GENERAL INFORMATION ON EXPOSURE

Acrylic acid (hereafter referred to as AA) is produced at five sites in tonnages of > 10,000 t/a, the production capacities per site range between 10,000 and 330,000 t/a. Additionally, data from three importers are included in the IUCLID-database. The total EU production capacity is estimated at 830,000 t/a for 1997/1998. The market trend is quite dynamic during the last decade with apparent annual growth rates about 5%.

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.

AA serves as an industrial intermediate product, i.e. it is either processed directly into a polyacrylate (industrial category IC 11) 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. Among the homo- and copolymers of AA, superabsorber polymers (SAP) are the most expansive use.

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 (IC 3). 99% of the acrylate esters are n-butyl, ethyl, methyl and 2-ethylhexyl acrylates, of which butyl acrylate predominates quantitatively. As for glacial AA, these acrylic esters serve as commercial products, which are further processed both on-site and by external downstream users.

3 ENVIRONMENT

3.1 ENVIRONMENTAL EXPOSURE

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 range 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.

The environmental behaviour of AA is determined by the following characteristics:

- The estimated range of atmospheric half-life is 39.6 hours to 6.5 days,
- AA is readily biodegradable. Hydrolysis is not significant at all tested pHs (3, 7, 11),
- AA is essentially non volatile,
- The average K_p value of 1.0 l/kg indicates no relevant adsorption onto sediment or soil.

Based on the physico-chemical properties of AA, hydrosphere and to a much lower extent air are the preferred target compartments for distribution and neither relevant bioaccumulation nor geoaccumulation are expected. In wastewater treatment plants (WWTPs), 87.3% of the substance is estimated to be removed entirely by biodegradation.

Predicted Environmental Concentrations (PECs) are calculated for the local aquatic environments of the production and processing sites using all site-specific information available. Data gaps are filled with default values proposed in the Technical Guidance Document (TGD). The resulting concentrations range between 0.043 µg/l and 0.77 µg/l with the exception of one production site (6.6 µg/l), which ceased manufacture of AA by October 1999.

AA is also used as external intermediate, i.e. more than half of the production quantity is sold within the EU (either as purified AA or as AA esters) to non producers/importers for processing at sites different to those considered above. The main use area for AA is the production of polymers, split to dry and wet polymerisation techniques and the production of SAP. From dry polymerisation no relevant releases via wastewater to hydrosphere have to be assumed.

The volumes of AA used for wet polymerisation have been provided for 13 European sites covering about 30% of AA externally processed. For the remaining ca. 70% of AA externally processed at unknown sites, a default calculation according to the TGD gives a local PEC significantly exceeding 0.1 mg/l. For all known sites where more than 500 t/a AA are handled, PECs are calculated based on site-specific use tonnages, site-specific information on wastewater treatment and dilution as far as available, and on default release factors. Resulting PECs range between 0.004 µg/l and 10 mg/l. It should be noted, that measured effluent concentrations range

up to >> 100 mg/l at known sites. With regard to received site-specific information, a processing volume of 10,000 t/a at one single site has to be assumed as a realistic worst case for unknown sites. For this generic site a PEC of 2.12 mg/l is calculated applying the TGD default parameters. Seven sites covering ca. 12% of the externally used tonnage confirmed zero release to hydrosphere as wastewater reutilization / recycling systems are employed.

For production of SAP, ca. 90% of the use volume is covered by site-specific emission data. Resulting PECs range to 0.97 µg/l at maximum. It should be noted, that two thirds of measured effluent concentrations exceeded 100 mg/l, reaching nearly 1.5 g/l at maximum, and that PECs around 1 µg/l result only because of high dilution. For almost 50% of the processing volume, zero emission to hydrosphere due to application of wastewater reutilization / recycling systems is confirmed. For the scenario of AA externally processed into SAP at unknown sites, a default calculation gives a local PEC of 0.34 mg/l.

Further exposure assessments are performed for leather finishing, textile finishing, paint formulation and application of water treatment agents, because the polymer emulsions may contain residual monomeric AA. The resulting PECs range between 0.2 µg/l and 1 µg/l.

No monitoring data for the aquatic environment are available.

AA measurements have been performed in drainage water from a tunnel construction site during application of a grouting agent containing magnesium diacrylate. High concentrations of AA were found in the drainage water during injection of the product and the resulting PEC for the local aquatic environment is estimated to range up to 280 µg/l, based on measured concentrations in the drainage water and site-specific dilution factors.

No PEC estimation is performed for the sediment compartment, since no relevant adsorption of AA onto sediment is expected.

For atmosphere one generic PEC estimation representing a realistic worst case for production and processing is performed. A local release amount of 36.3 t/a is estimated using the respective emission factors proposed in the TGD and this results in a concentration in air in the vicinity of the site of 34 µg/m^3. A refined scenario, estimating releases from external processing applying dry and wet polymerisation processes including SAP production by downstream users, results in a significantly lower PEC_{air} (9.3 µg/m^3).

Local exposure of atmosphere from manufacturing, formulation and use of polymers is expected to be significantly below the generic emissions calculated above for handling of monomeric AA and therefore additional quantification is not necessary.

Releases of AA to soil are expected to occur through atmospheric deposition after local release to atmosphere. The input through sludge application on agricultural soil is considered negligible, as AA does not partition to a significant extent to sewage sludge in WWTPs.

From the total annual deposition in vicinity of the generic worst-case site, the maximum equilibrium concentration in soil is calculated according to the procedure proposed in the TGD. The resulting bulk concentration in soil (natural soil and agricultural soil) is 2.4 µg/kg wwt, the respective porewater concentration is 2.4 µg/l.

The regional background concentrations calculated according to EUSES are low and do not contribute significantly to the local concentrations. The resulting values are:

$PECregional_{aquatic}$	=	0.40	µg/l
$PECregional_{air}$	=	0.002	µg/m^3
$PECregional_{agr.-soil}$	=	0.02	µg/kg (wwt)
$PECregional_{agr.-soil\ porewater}$	=	0.02	µg/l
$PECregional_{natural-soil}$	=	0.07	µg/kg (wwt)

3.2 EFFECTS ASSESSMENT

For fish, three valid results from acute tests are currently available. *Oncorhynchus mykiss* was found to be the most sensitive, the recorded 96-hour LC_{50} is 27 mg/l. For invertebrates, acute and long-term studies on *Daphnia magna* had been conducted and the most relevant EC value is a 21-d NOEC of 7 mg/l. Among four algae toxicity tests, the results of two independent guideline studies with *Scenedesmus subspicatus*, point particularly at specific algal sensitivity to AA. Derived from growth rate, the reported 72-hour NOECs are 30 and 31 µg/l, respectively.

The Predicted No Effect Concentration (PNEC) is derived from the lowest valid effect concentration, i.e. 30 µg/l in an algae test. Although long-term test results 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. $PNEC_{aqua}$ = 3 µg/l.

The derivation of a PNEC for microorganisms is based on results from four non-standard tests on cell multiplication inhibition with protozoa and bacteria. For the three protozoa tests NOEC values between 0.9 mg/l and 41 mg/l are reported, for the bacterial test with activated sludge a 30-min NOEC of 100 mg/l is reported. Applying an assessment factor of 1 for the protozoan species according to the TGD, the $PNEC_{microorganisms}$ is set at 0.9 mg/l for municipal plants. For industrial plants, a PNEC of 10 mg/l is derived, applying an assessment factor of 10 to the result with activated sludge.

There are no relevant results with benthic organisms available and there is no need for performing an indicative quantitative risk assessment for the sediment compartment, because AA shows no relevant adsorption and there are no monitoring data on AA concentrations in sediment available.

It is not possible to derive a PNEC for the atmospheric compartment due to the lack of experimental data.

Only one test on effects to terrestrial organisms is available. A respiration inhibition test with natural soil microflora revealed a 28-day NOEC of 100 mg/kg dwt. With an assessment factor of 1,000 a $PNEC_{soil}$ of 0.1 mg/kg would result. Additionally, the aquatic PNEC of 3 µg/l can be used and compared to the concentration in soil pore water in an indicative risk assessment for the soil compartment.

3.3 RISK CHARACTERISATION

The possible risks to microorganisms in wastewater treatment plants (WWTPs) are evaluated for industrial and municipal facilities. For all considered industrial scenarios the PEC/PNEC ratios are well below one and therefore no risk for the function of industrial WWTPs is expected. Regarding municipal WWTPs, PEC/PNEC ratios significantly above one are calculated for the downstream use scenarios of wet polymerisation (default and two known sites, based on measured effluent concentrations) and SAP production (default and highest site-specific figures).

According to the TGD and to a related Technical Recommendation, the present data give reasons for further testing needs to clarify the integrity of the native ciliate population in sewage sludge as a whole. However, it is accepted to postpone this testing need with regard to the conclusion for surface water (see below).

For surface water a comparison between PEC and PNEC for all relevant exposure scenarios is performed. Based on the updated site-specific data all producing sites reveal PEC/PNEC ratios clearly below one, except for site D, which ceased AA production by October 1999. There is at present no need for further information gathering or for limiting the risk beyond those measures which are already being applied **(conclusion (ii))**.

PEC/PNEC ratios above one are calculated for three out of eight known downstream user sites where > 500 t/a AA are used for wet polymerisation processes, and for the respective default scenarios, as well as for SAP production (default calculation for unknown sites). Therefore, a risk for the aquatic compartment has to be deduced on the basis of the present data. Risk reduction measures at the Community level are recommended **(conclusion (iii))**.

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

Although an improvement of exposure data would in principle be possible for the wet polymerisation scenarios including SAP production, e.g. by performing effluent measurements at the whole range of relevant sites, it is concluded that a sufficient and appropriate data basis cannot be acquired within an acceptable time frame and with acceptable efforts. It should be noted, that effluent concentrations up to >> 100 mg/l have been measured regularly both at wet polymerisation and SAP production sites. Moreover, with regard to the dynamic acrylate market, reliable PEC estimations are hampered by the variability of AA tonnages at individual sites. On the other hand, wastewater reutilization / recycling systems are known to result in zero emissions to the hydrosphere at a number of known sites. Plants applying such advanced process engineering would not require further consideration of risk reduction measures. Measures to be applied for limiting the risk to the local aquatic environment are supposed 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 drainage water and a risk for the local aquatic environment has to be deduced. The exposure assessment was based on measured concentration at a tunnel constructions site leading to a PEC/PNEC-ratio significantly above 1. A quantitative extrapolation to other construction sites seems difficult, but similar conditions might be anticipated. Data improvement is not the proposed option, because an environmentally safe handling of the grouting agent has to be achieved independently of local circumstances. Therefore, measures appropriate to local circumstances should be applied **(conclusion (iii))**.

Regarding all other processing and use scenarios, i.e. dry polymerisation, leather finishing, textile finishing, formulation of paints, and application of water treatment agents, no risk for the aquatic compartment is expected **(conclusion (ii))**.

From the current manufacturing and use of AA, no risk for the sediment compartment is expected **(conclusion (ii))**.

Due to the physical properties of AA, atmosphere is not regarded as a target of distribution. The short atmospheric half-lives of AA contribute further to low resulting concentrations in air. Therefore, adverse effects on organisms and abiotic effects on atmosphere, like global warming and ozone depletion are not expected from AA.

From an indicative risk assessment for the soil compartment no risk is deduced for the present data and there is no need for further testing and/or gathering of exposure information.

AA does not present indications of a bioaccumulation potential. A risk characterisation for secondary poisoning is not required **(conclusion (ii))**.

4 HUMAN HEALTH

4.1 HUMAN HEALTH (TOXICITY)

4.1.1 Exposure assessment

4.1.1.1 Occupational exposure

Acrylic acid (AA) 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.

The following occupational exposure limits are established for acrylic acid (ILO, 1994)[3]:

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/m³)

* De Nationale MAC lijst (Ministerie van Sociale Zaken en Werkgelegenheid, 1996)[3]

** Grænseværdier for stoffer og materialer (Arbejdstilsynet, 1996)[3]

With regard to inhalation exposure, exposure to acrylic acid in vapour form has to be considered. Concerning dermal exposure skin contact with the corrosive substance and with corrosive preparations (≥ 5% AA) is only assumed by single contacts. During use of non-corrosive preparations (< 5% AA) intermittent dermal contact has to be taken into account.

[3] For references, see the comprehensive Final Risk Assessment Report that can be obtained from the European Chemicals Bureau: http://ecb.jrc.it

The exposure assessment is based on measured data (limited), expert judgement and estimations according to the EASE model.

For occupational exposure, exposure 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.

Table 4.1 summarises the exposure data of acrylic acid.

Table 4.1 Summary of exposure data of acrylic acid

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

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

4.1.1.2 Consumer Exposure

Polymers manufactured with acrylic acid as co-monomer are used in consumer products (sanitary towels, pantyliners, and nappy pants). Furthermore products containing acrylic acid are used as adhesive or glue and in adhesive substances on the basis of solvents. Acrylic acid in sealing compounds is also used by consumers.

Inhalation exposure to UV-hardening adhesives

Assuming the use of 1 g UV-hardenable adhesive (content of acrylic acid monomer 6%) 4 times per year for 1 hour each, the consumer will be 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 (using the SCIES model).

Dermal 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 exposed to residual monomers of acrylic acid in homopolymerisates of acrylic acid which are used as "superabsorbents" in nappy pants.

The following amounts of acrylic acid in the residual dampness of nappies have been submitted: daytime: 0.36 µg, naptime: 0.43 µg, and 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 been calculated. Taking into account that 20% of acrylic acid is available for absorption (= 0.59 µg), that the body weight of newborn children is taken as 3.16 ± 0.35 kg, and that of a 1-year-old child is 9.74 ± 1.07 kg the dermal exposure of babies and 1-year-old children was calculated to be 0.18 µg/kg bw/d and 0.06 µg/kg bw/d, respectively.

4.1.1.3 Humans exposed via the environment

Humans can be exposed indirectly to acrylic acid via the environment mainly by intake via plant stems for the local and via drinking water for the regional scenario. An intake of 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 scenario, respectively.

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

Toxicokinetics, metabolism and distribution

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 of substance concentration. Mouse skin shows better permeability than human one. 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 citric acid cycle. Elimination preferably occurs as carbon dioxide (exhalation). Small amounts of 3-hydroxypropionic acid but no unmetabolised acrylic acid could be detected in urine. *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 lead to the formation of small amounts of mercapturic acid derivates.

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. 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 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 with a 10% aqueous solution of acrylic acid (pH 2.5) thus indicating that corrosive effects are not caused by the pH of the test substance. A dermal LD_{50} of approximately 640 mg/kg was determined for rabbits (with undiluted acrylic acid). 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. Inhalation LC_{50} values of 3.6 to >5.1 mg/l/4 hours have been determined. For classification, see Section 1.4.

Irritation/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.

Sensitisation

Pure acrylic acid does not show skin sensitising properties in animal sensitisation tests. However, skin sensitisation was observed in humans. This was attributed to oligomeric impurities in the raw material. Respiratory sensitisation has not been observed in humans.

Repeated dose toxicity

There is no information on the health effects in humans of repeated exposure to acrylic acid. 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 drinking water studies. 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, respectively.

Toxic effects of relevance were seen in inhalation studies in rats and mice. In a 90-day study, acrylic acid vapour induced degenerative lesions on the olfactory mucosa in mice at 5 ppm

(15 mg/m^3) and in rats at 75 ppm (221 mg/m^3). Mice seemed to be more sensitive than rats, thus a LOAEC of 5 ppm (15 mg/m^3) was derived for local effects. Long-term dermal exposure at concentrations >1% resulted in skin irritation.

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 data and taking into account data on structurally-related acrylic compounds, it is unlikely that acrylic acid is mutagenic *in vivo*.

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.

Toxicity for reproduction

In oral reproductive toxicity studies in 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 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. No prenatal developmental toxicity was observed (rats and rabbits, inhalation exposure). A NOAEC/developmental toxicity of 225 ppm (663 mg/m^3) was derived from the developmental toxicity study in rabbits.

4.1.3 Risk characterisation

4.1.3.1 Workers

For the purpose of the 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. The following report concentrates on the main points of concern with regard to the risk characterisation at workplaces.

Irritation/Corrosivity

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 "Repeated dose toxicity"). 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))**.

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 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. 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 species extrapolation from rodents to humans. Rodents show a nasal anatomy and respiratory physiology different from humans. 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 epithelium. However, the results are not sufficiently assessable due to insufficient data with regard to the uncertainty of the model and the sensitivity parameters. Therefore the calculation of the MOS is performed with the LOAEC of 5 ppm (15 mg/m^3) that resulted in slight effects in some mice (near to NAEC).

The LOAEC of 15 mg/m^3 is compared with the exposure information for scenarios with repeated daily inhalation exposure. 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))**.

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 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.

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. **(conclusion (iii)).**

Combined exposure (systemic effects)

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

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 **(conclusion (iii))**. This conclusion is also applicable to other scenarios where conclusion (iii) has been applied for repeated dose toxicity, systemic.

The conclusions of the occupational risk assessment are summarised in **Table 4.2**.

Table 4.2 Conclusions of the occupational risk assessment of acrylic acid

	Irritation/ Corrosivity (inhalation)	Repeated dose toxicity (local, inhalation)	Repeated dose toxicity (systemic, inhalation)	Repeated dose toxicity (combined exposure, systemic)
Chemical industry				
Production and further processing	**iii**	ii	ii	ii
Manufacture of adhesives (1-10% acrylic acid)	ii	ii	ii	ii
Industrial area				
Manufacture of adhesives (1-10% acrylic acid)				
- with LEV	ii	ii	ii	ii
- without LEV	**iii**	**iii**	**iii**	**iii**
Use of adhesives ≥ 5% acrylic acid (labelled as corrosive)				
- with LEV	**iii**	**iii**	ii	ii
- without LEV	**iii**	**iii**	**iii**	**iii**
< 5% acrylic acid (not labelled as corrosive)				
- with LEV	**iii**	**iii**	ii	ii
- without LEV	**iii**	**iii**	**iii**	**iii**
Decomposition during production of integrated circuits	ii	ii	ii	ii
Skilled trade				
Use of adhesives ≥ 5-10% acrylic acid (labelled as corrosive)	**iii**	ii	ii	ii
≤ 5% acrylic acid (not labelled as corrosive)	**iii**	ii	ii	ii

4.1.3.2 Consumers

Repeated dose toxicity

Following the exposure assessment consumers may be exposed to acrylic acid via inhalation using UV-hardening adhesives. This exposure does not reflect a realistic chronic exposure scenario. The LOAEC for local effects of 5 ppm (15 mg/m³) used for the margin of safety 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 been considered. Taking into account the worst-case exposure scenario (no real chronic exposure) and even the use of a LOAEC, the margin of safety is judged to be sufficient **(conclusion (ii))**.

Following the exposure assessment babies and smaller children may be exposed dermally to acrylic acid due to the use of nappy pants. This exposure results from the residual content of monomers in homopolymers of acrylic acid, which are used as "superabsorbents" in such products. The calculation of the dermal exposure of babies due to napkins leads to an internal exposure of 0.00018 mg/kg bw/d (uptake basis assuming that the bioavailability via the dermal

route is 100%). The NOAEL of 40 mg/kg bw/d was derived from a drinking water 90-day study in rats. The margin of safety 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))**.

Toxicity for reproduction

In oral reproductive toxicity studies in rats no effects on reproductive function (fertility) were observed, but some signs of postnatal developmental toxicity (retarded body weight gain of the pups) were observed following exposure of the parental generation. No gross abnormalities were seen in the offsprings. No prenatal developmental toxicity was observed in rats and rabbits after inhalation. Taking into account the low exposure it can be concluded that there is no concern **(conclusion (ii))**.

4.1.3.3 Humans exposed via the environment

Repeated dose toxicity

The main route of exposure is plant stems for the point source and the drinking water for the regional approach. For the risk characterisation the total daily intake for the local and the regional scenario (0.05 mg/kg bw/d and 15 ng/kg bw/d, respectively) are compared with an oral NOAEL of 40 mg/kg bw/d which was derived from the 90-day drinking water rat study. The margins of safety expressed by the magnitude between the calculated exposures and the NOAEL are considered to be sufficient for both scenarios. Thus, the substance is of no concern in relation to indirect exposure via the environment **(conclusion (ii))**.

Reproductive toxicity

In oral reproductive toxicity studies in rats no effects on reproductive function (fertility) were observed, but some signs of postnatal developmental toxicity (retarded body weight gain of the pups) were observed following exposure of the parental generation. No gross abnormalities were seen in the offsprings. No prenatal developmental toxicity was observed in rats and rabbits after inhalation. Taking into account the low exposure it can be concluded that the margin of safety for both scenarios is considered to be sufficient. Thus, there is no concern in relation to indirect exposure via the environment **(conclusion (ii))**.

4.2 HUMAN HEALTH (PHYSICO-CHEMICAL PROPERTIES)

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)**).

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.