According to Regulation (EC) No 1907/2006

Section 1 Identification of the substance/mixture and of the company/undertaking

1.1 Product identifier:

Identification on the label/Trade name:	acrylic acid
Additional identification:	acrylic acid
Identification of the product:	CAS#: 79-10-7 ; EC#: 201-177-9
Index Number:	607-061-00-8
REACH registration No.:	01-2119452449-31-XXXX

1.2 Relevant identified uses of the substance and uses advised against:

1.2.1 Identified uses:

ES 1: Manufacture and distribution of the substance

ES2: Manufacture of intermediates at production sites of substance (on-site) and at downstream user sites (off-site):esterification

ES3: Polymerization at production sites of substance (on-site) and at downstream user sites (off-site): superabsorber polymersand other polyacrylates

ES4: Other uses of substance as intermediateES5:

Use of substance as a laboratory agent

1.2.2 Uses advised against:

Not available.

1.3 Details of the supplier of the safety data sheet:

Supplier:	**Bloomchemag BV**
Address:	Sint-Antoniusstraat 16 b1, B-2400, Mol, Belgium
Contact person(E-mail):	info@bloomchemag.com / Corporate@bloomchemag.com
Telephone:	+91 7291970499

1.4 Emergency telephone Number:

+91 7291970499

Available outside office hours?	YES		NO	X

Section 2 Hazards Identification

2.1 Classification of the substance/mixture

2.1.1 Classification:

The substance is classified as following according to REGULATION (EC) No 1272/2008:

REGULATION (EC) No 1272/2008	
Hazard classes/Hazard categories	Hazard statement
Flam. Liquid 3	H226
Acute Tox. 4-oral	H302
Acute Tox. 3-dermal	H312
Acute Tox. 4-inhalation	H332
Skin Corr. 1A	H314
Eye Dam. 1	H318
STOT Single Exp. 3	H335
Aquatic Acute 1	H400

For full text of H- phrases: see section 2.2.

2.2 label elements

Hazard Pictograms:

Signal Word(S): Danger

Hazard Statement: H226: Flammable liquid and vapour.

H302: Harmful if swallowed.

H312: Harmful in contact with skin.

H332: Harmful if inhaled.

H314: Causes severe skin burns and eye damage.H335:

May cause respiratory irritation.

H400: Very toxic to aquatic life.

Precautionary statement P210 Keep away from heat/sparks/open flames/hot surfaces. — No smoking.

P233 Keep container tightly closed

P240 Ground/bond container and receiving equipment

P241 Use explosion-proof electrical/ventilating/lighting/equipment.P242

Use only non-sparking tools.

P243 Take precautionary measures against static dischargeP261

Avoid breathing dust/fume/gas/mist/vapours/spray P264 Wash

skin thoroughly after handling.

P270 Do not eat, drink or smoke when using this productP271

Use only outdoors or in a well-ventilated area P273 Avoid

release to the environment.

P280 Wear protective gloves/protective clothing/eye protection/face protection.P370 + P380

in case of fire: Evacuate area.

P301 + P312 IF SWALLOWED: call a POISON CENTER or doctor/physician IF you feel unwell.

P302 + P352 IF ON SKIN: wash with plenty of soap and water.

P304 + P340 IF INHALED: Remove victim to fresh air and Keep at rest in a position comfortable forbreathing

P312 Call a POISON CENTER or doctor/physician if you feel unwell.

P303 + P361 + P353 IF ON SKIN (or hair): Remove/Take off Immediately all contaminated clothing.Rinse SKIN with water/shower

P322 Specific measures

P330 Rinse mouth

P361 Remove/Take off immediately all contaminated clothingP363

Wash contaminated clothing before reuse.

P370 + P378 In case of fire: Use Water spray, foam, CO_2, dry powder. Fight large fire with alcoholresistant foam or water spray. for extinction.

P391 Collect spillage. Hazardous to the aquatic environment

P403 + P233 Store in a well-ventilated place. Keep container tightly closedP405 Store locked up

P501 Dispose of contents/container to in a licensed facility

2.3 Other hazards

Not available

Section 3 Composition/information on ingredients

Substance/Mixture: Substance

Ingredient(s):

Chemical Name	Registration No.	CAS No.	EC No.	Typical Concentration
Acrylic Acid	01-2119452449-31-xxxx	79-10-7	201-177-9	99.2%

Section 4 First aid measu res

4.1 Description of first aid measures:

Immediately remove contaminated clothing. If danger of loss of consciousness, place patient in recovery position and transportaccordingly. Apply artificial respiration if necessary. First aid personnel should pay attention to their own safety.

4.1.1 In case of inhalatio n:

Whilst protecting yourself remove the casualty from the hazardous area. Lay the casualty down in a quiet place and protect him against hypothermia. Provide fresh air, seek medical advice if necessary. Monitor breathing. In case of breathing difficulties have the casualty inhale oxygen. If the casualty is unconscious but breathing lay him in a stable manner on his side. Arrange medical treatment.

4.1.2 In case of skin con tact:

Relocate the casualty away from the source of danger. Take off all contaminated clothing immediately while protecting yourself. Immediately wash off and cleanse affected skin areas with plenty of water. Following massive, extensive contact, immediatelyplace the casualty under the emergency shower, wash off with plenty of water and only then remove clothes. Seek medical

advice independent of skin damage.

4.1.3 In case of eyes contact:

Rinse affected eye with widely spread lid for 15 minutes. Transport the casualty immediately to an eye doctor or into hospital. Continue eye bath during transportation.

4.1.4 In case of ingestion:

Rinse mouth with water and spit fluids out. Drink afterwards plenty of water in sips. Do not induce vomiting. Arrange medical treatment.

4.2 Most important symptoms and effects, both acute and delayed

Flammable liquid and vapour.

Harmful if swallowed.

Harmful in contact with skin.

Harmful if inhaled.

Causes severe skin burns and eye damage.

May cause respiratory irritation.

4.3 Indication of any immediate medical attention and special treatment needed

If skin irritation or rash occurs, get medical advice/attention.

Section 5 Fire-Fighting measures

5.1 Extinguishing media:

Suitable extinguishing media:	Water spray, foam, CO2, dry powder. Fight large fire with alcohol resistant foam or water spray. Do not use high volume water jet.
Unsuitable extinguishing media:	Not available.
5.2 Special hazards arising from the substance or mixture	Cool surrounding containers with water spray. If possible, take container out of dangerous zone. Heating causes a rise in pressure, risk of bursting and explosion. Spontaneous polymerization. Shut off sources of ignition. Beware of backfire. Stay on upwind side.
5.3 Special fire fighting methods and special protective actions for fire-fighters:	Wear self-contained breathing apparatus. Wear suitable, tightly sealed protectiveclothing. Full protective suit.

Section 6 Accidental release measures

6.1 Personal precautions, protective equipment and emergency procedures:

6.1.1 For non-emergency personnel:	Wear personal protective equipment (respiratory protection, eye protection, hand protection, body protection. Ensure sufficient ventilation. The hazardous area canonly be entered once suitable protective measures are implemented.
6.1.2 For emergency responders:	Keep sparks, flames, and other sources of ignition away. Keep material outof water sources and sewers. Build dikes to contain flow as necessary. Usewater spray to knock-down vapors. Neutralize spilled material with crushed limestone, soda ash, or lime.
6.2 Environmental Precautions:	Shut off all ignition sources. Evacuate area and warn affected surroundings. Do not

allow entrance in soil, stretches of water, ground water, drainage systems, and surface water.

6.3 Methods for Containment and Cleaningup: Use mechanical handling equipment. Pump off larger quantities. Dilute smaller quantities with plenty of water, neutralize if necessary with calcium carbamate or absorb spilt liquid with an absorbent (e.g. diatomite, vermiculite, sand). Fill into marked, sealable containers. Dispose according to regulations. Afterwards ventilate area and wash spill site. Inform responsible authorities if necessary.

6.4 Reference to other sections: See Section 7 for information on safe handling. See section 8 for information on personal protection equipment.See Section 13 for information on disposal.

Section 7 Handling and storage

7.1 Precautions for safe handling:

7.1.1 Protective measures: Use leak-proof equipment with exhaust for filling, refilling or transfer. Do not leave containers open. Avoid splashing. Fill into labelled container only. Use acid resistantutensils. Avoid skin and eye contact. Do not breathe in vapor or aerosols. Unintended, spontaneous polymerization can occur by overheating (especially localoverheating), photo-initiation (UV light), contamination, corrosion (Fe), stabilizer depletion and stabilizer deactivation (via oxygen depletion). Thawing of frozen product with tempered water between 20° and 35°C only.

Advice on protection against fire and explosions: Take precautionary measures against static charges. Keep away from ignition sources.

7.1.2 Advice on general occupational hygiene: Avoid contact with skin. Avoid inhalation of vapour. Eye wash fountains and safety showers must be easily accessible. Wash soiled clothing immediately.

7.2 Conditions for safe storage, includingany incompatibilities: Requirements for storage areas and storage containers: Protect from exposure to sunlight, from overheating/heating up or freezing. Recommended storage temperatures 15°C (min) – 25°C (max.). Keep under atmospheric oxygen (air), never use inert atmosphere: stabilizer is only effective in presence of oxygen. Observe max. shelf life of water free product. Suitable materials are: stainless steel, aluminium, polyethylene. Unsuitable materials are: iron, carbon-less (mild) steel, copper, brass and their alloys.

7.3 Specific end use(s): Not applicable.

Section 8 Exposure Controls/Personal Protection

8.1 Control parameters:

8.1.1 Occupational exposure limits: Not available

8.1.2 Additional exposure limits under the conditions of use: Not available

8.1.3 DNEL/DMEL and PNEC-Values:

DN(M)ELs for workers

Route	Type of effect	Hazard conclusion	Most sensitive endpoint
Inhalation	Systemic effects - Long-term		
Inhalation	Systemic effects - Acute		
Inhalation	Local effects - Long-term		irritation (respiratory tract)
Inhalation	Local effects - Acute		irritation (respiratory tract)
Dermal	Systemic effects - Long-term		
Dermal	Systemic effects - Acute		
Dermal	Local effects - Long-term		
Dermal	Local effects - Acute		skin irritation/corrosion
Eyes	Local effects		

DN(M)ELs for the general population

Route	Type of effect	Hazard conclusion	Most sensitive endpoint
Inhalation	Systemic effects - Long-term		
Inhalation	Systemic effects - Acute		
Inhalation	Local effects - Long-term	DNEL (Derived No Effect Level): 3.6 mg/m³	irritation (respiratory tract)
Inhalation	Local effects - Acute	DNEL (Derived No Effect Level): 3.6 mg/m³	irritation (respiratory tract)
Dermal	Systemic effects - Long-term		
Dermal	Systemic effects - Acute		
Dermal	Local effects - Long-term		
Dermal	Local effects - Acute	DNEL (Derived No Effect Level): 1 mg/cm²	skin irritation/corrosion
Oral	Systemic effects - Long-term		
Oral	Systemic effects - Acute		
Eyes	Local effects		

PNEC

Compartment	Hazard conclusion	Remarks/Justification

Freshwater	PNEC aqua (freshwater): 0.003 mg/L	Assessment factor: 10 Extrapolation method: assessment factor Due to the ready biodegradability of acrylic acid only effect valuesderived from studies with analytical monitoring should be used for the derivation of the PNEC. Such results are available from several acute tests conducted under standardised conditions for all three trophic levels. The LC50-/EC50-values for freshwater fish and invertebrates range from 27 mg/L (fish) to 47 mg/L (invertebrates). Two 21-day chroniclife-cycle studies with Daphnia magna are available. The NOECs for reproduction were 12 and 19 mg/L, respectively, and the NOECs for maternal survival were 3.8 and 7 mg/L, respectively (Huels, 1995; BAMM, 1996). Acute and long-term test results reveal that algae are the most sensitiveaquatic organisms. As their EC50 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. For growth rate reduction, the lowest EC10 value derived in two tests (BASF AG, 1994; Huels, 1995) 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. For the majority of test scenarios, growth rate was found to provide the most reliable estimate of "true" toxicity, featuring the advantages of lesssusceptibility to experimental disturbances, independence of test duration, better statistics, and immediate ecological relevance (Ratte, 1998). 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 EC10 values for PNEC derivation, provided that a smooth concentration-response curve is obtained as in the present tests.
Marine water	PNEC aqua (marine water): 0.0003 mg/L	Assessment factor: 100 Extrapolation method: assessment factor Due to the ready biodegradability of acrylic acid only effect valuesderived from studies with analytical monitoring should be used for the derivation of the PNEC. Such results are available from several acute tests conducted under standardised conditions for all three trophic levels. LC50-/EC50-values for freshwater and saltwater fish and

		invertebrates were between 20 and 240 mg/L, i.e. 27 mg/L (freshwater fish), 236 mg/L (marine fish),47 mg/L (freshwater invertebrates) and 97 mg/L (marine invertebrates). Thus, saltwater fish and invertebrates are not more sensitive to acrylic acid than freshwater fish and invertebrates.Two 21-day chronic life-cycle studies with Daphnia magna are available.The NOECs for reproduction were 12 and 19 mg/L, respectively and theNOECs for maternal survival were 3.8 and 7 mg/L, respectively (Huels, 1995; BAMM, 1996). Acute and long-term test results reveal that algae are the most sensitiveaquatic organisms. As their EC50 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. For growth rate reduction, the lowest EC10 value derived in two tests (BASF AG, 1994; Huels, 1995) was 0.03 mg/L for Scenedesmus subspicatus. Short-term tests with species from three trophic levels are available pluslong-term NOECs from two trophic levels (invertebrates and algae). Although long-term NOECs/EC10-values are available from only two trophic levels, an assessment factor of 100 can be proposed as recommended by the Guidance on information requirements and chemical safety assessment, Chapter R.10 (ECHA, May 2008) becauseof the comparatively high toxicity of acrylic acid to algae. The acute EC50 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.
Intermittent releases to water	PNEC aqua (intermittent releases): 0.0013 mg/L	Assessment factor: 100 Extrapolation method: assessment factor Short-term tests from all three trophic levels are available. Taking all available short-term tests with freshwater and saltwater species into consideration, the most sensitive species is Scenedesmus subspicatus with an EC50 of 0.13 mg/L. The recommended assessment factor for the derivation of a PNEC aqua (intermittent releases) is 100 (ECHA, May 2008).
Sediments (freshwater)	PNEC sediment (freshwater): 0.0236mg/kg sediment dw	Extrapolation method: partition coefficient Since no experimental data were available for sediment dwelling organisms, the PNEC sed was estimated using the equilibrium partitioning method as recommended by the Technical Guidance Document for Risk Assessment (ECB, 2003) and Guidance on information requirements and chemical safety assessment, Chapter

		R.10 (ECHA, May 2008). PNEC sediment in mg/kg sediment ww = 0.00514
Sediments (marine water)	PNEC sediment (marine water): 0.002346 mg/kg sediment dw	Assessment factor: 10 Extrapolation method: assessment factor Derived from PNEC sediment (frshwater), applying an assessmentfactor of 10.
Sewage treatment plant	PNEC STP: 0.9 mg/L	Assessment factor: 1 Extrapolation method: assessment factor For the derivation of the PNEC STP an activated sludge respiration inhibition test conducted according to standards (ISO 8192) has to be taken into account. The 30-min EC20 for domestic activated sludge was900 mg/L. An assessment factor of 100 was applied to this value leadingto a PNEC STP of 9 mg/L. But the most sensitive microorganism to acrylic acid was the protozoan Chilomonas paramaecium with a 48-hourTT of 0.9 mg/L. Although this species does not influence the degradationprocesses itself, it is necessary for a proper function of a WWTP. For thiskind of test result an assessment factor of 1 is proposed for the determination of PNEC STP. This value for the PNEC STP was used in th EU Risk Assessment (2002).
Soil	PNEC soil: 1 mg/kg soil dw	Assessment factor: 100 Extrapolation method: assessment factor A short-term test in Eisenia fetida with an LC50 > 1000 mg/kg dw and one long-term toxicity test with a NOEC of 100 mg/kg soil dw based on soil micro-flora (carbon-cycle) are available. An assessment factor of 100 is proposed by the Guidance on information requirements and chemical safety assessment, Chapter R.10 (ECHA, May 2008). The resulting PNEC soil is 1 mg/kg soil dw.
Air	No hazard identified:	
Secondary poisoning	PNEC oral: 0.03 g/kg food	Assessment factor: 30 Based on a log Kow value of 0.46, no bioaccumulation of acrylic acid in organisms is expected. Hence, secondary poisoning will not be animportant factor in the hazard assessment. Nevertheless, a PNEC oral was determined. Since no adequate data on acute or chronic toxicity towards birds are available, the PNEC oral for secondary poisoning was derived from a chronic drinking water study with rats (BASF AG 1987). The NOAEL (systemic) was 40 mg/kg bw corresponding to a NOEC (food) = 8E-04 kg/kgfood. The assessment factor for a 12-months study in mammals is

		30 resulting in a PNEC oral (mammal) = 2.7E-05 kg/kg food.	

8.2 Exposure controls

8.2.1Appropriate engineering controls:

Ventilation: Use local exhaust ventilation, or other engineering controls to maintain airborne levels below exposure limit requirements or guidelines. If there are no applicable exposure limit requirements or guidelines, general ventilation should be sufficient for most operations. Local exhaust ventilation may be necessary for someoperations.

8.2.2 Individual protection measures, such as personal protective equipment:

Eye/face protection:

Use chemical goggles. Chemical goggles should be consistent with EN 166 or equivalent. If exposure causes eye discomfort, use a full-face respirator.

Hand protection:

Use chemical resistant gloves classified under Standard EN374: Protective gloves against chemicals and micro-organisms. Examples of preferred glove barrier materials include: Butyl rubber. Ethyl vinyl alcohol laminate ("EVAL"). Viton. Examples of acceptable glove barrier materials include: Natural rubber ("latex"). Neoprene. Nitrile/butadiene rubber ("nitrile" or "NBR"). Polyvinyl alcohol ("PVA"). Polyvinyl chloride ("PVC" or "vinyl"). When prolonged or frequently repeated contactmay occur, a glove with a protection class of 6 (breakthrough time greater than 480minutes according to EN 374) is recommended. When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374) is recommended. NOTICE: The selection of a specific glove for a particular application and duration of use in a workplace should also take into account all relevant workplace factors such as, but not limited to: Other chemicals which may be handled, physical requirements (cut/puncture protection, dexterity, thermal protection), potential body reactions to glove materials,
as well as the instructions/specifications provided by the glove supplier.

Respiratory protection:

Respiratory protection should be worn when there is a potential to exceed the exposure limit requirements or guidelines. If there are no applicable exposure limit requirements or guidelines, wear respiratory protection when adverse effects, such as respiratory irritation or discomfort have been experienced, or where indicated byyour risk assessment process. In dusty or misty atmospheres, use an approved particulate respirator. For emergency conditions, use an approved positive-pressureself-contained breathing apparatus. Use the following CE approved air-purifying
respirator: Organic vapor cartridge with a particulate pre-filter, type AP2.

8.2.3 Environmental exposure controls:

Avoid discharge into the environment.
According to local regulations, Federal and official regulations.

Section 9 Physical and chemical properties

9.1 Information on basic physical and chemical properties

Appearance:	liquid
Colour:	colorless
Melting / freezing point/range (°C):	286.15 K at 101.3 kPa
Boiling point/range (°C):	414.15 K at 101.3 kPa
Flash point (°C):	48.5°C at 1013 hPa
Vapour pressure:	529 Pa at 298.15 K
Relative Density:	1.05 (d20/4)
Surface tension	69.6 mN/m at 20°C and 1000 mg/L
Water solubility (g/l):	1000 g/l at 25° C.
Partition coefficient n-octanol/water (log value):	Log Pow at 25°C: 0.46
Self-ignition temperature	711.2 K at 1013 hPa
Dissociation constant	4.26 at 25° C
Viscosity	1.149 mPa s (dynamic) at 25° C
Molecular Formula:	C3H4O2
Molecular Weight:	72.0627

9.2. Other information:

Flammability:	Flammable
Explosive properties :	Non explosive
Oxidising properties :	Non oxidizing
Granulometry :	Not available
Stability in organic solvents and identity of relevant degradation products :	Not available

Section 10 Stability and reactivity

10.1 Reactivity: Stable under recommended storage conditions. Hazardous Polymerization can occur.

10.2 Chemical stability: Stable under recommended storage conditions. Unstable at elevated temperatures. Hygroscopic.

10.3 Possibility of hazardous reactions: Hazardous Polymerization can occur. Elevated temperatures can cause hazardous polymerization. The inhibitor used with this monomer may separate if product becomes frozen. Protect from freezing. After freezing and thawing, hazardous polymerization can occur if thawed incorrectly. Polymerization can be catalyzed by:Absence of air. Free radical initiators. High temperature. Peroxides. Uninhibited monomer vapors can polymerize and plug relief devices.

10.4 Conditions to avoid: Avoid temperatures above 25 °C. Avoid temperatures below 15 °C. Exposure to elevated temperatures can cause product to decompose. Avoid static discharge. Avoid moisture. Do not blanket or purge with an inert gas to avoid depleting the oxygen concentration. The inhibitor used with this monomer can separate if product becomes frozen. Avoid direct sunlight or ultraviolet sources.

10.5 Incompatible materials: Avoid contact with oxidizing materials. Avoid contact with: Aldehydes. Amines. Anhydrides. Azides. Ethers. Free radical initiators. Halides. Iron oxides (rust). Mercaptans. Strong bases. Corrosive when wet. Avoid contact with metals such as:Brass. Copper. Mild steel. Avoid unintended contact with: Activated carbon. Aluminum oxide. Silica gel. Avoid contact with absorbent materials such as:
Clay-based absorbents. Avoid unintended contact with peroxides.

10.6 Hazardous decomposition products: Decomposition products depend upon temperature, air supply and the presence of other materials.

Section 11 Toxicological information

11.1 Toxicokinetics, metabolism and distribution

Following oral administration of [^{14}C]-Acrylic acid in rats and mice, a high percentage of the radiolabel (60 – 80 %) was rapidly absorbed and eliminated as $^{14}CO_2$ within 24 hours by both species. Excretion in urine and faeces accounted for 1-4 %, respectively. In rats, about 19-25 % of the acrylic acid-derived radioactivity remained in the tissues examined after 72 hr, mostly in adipose tissue and muscle. High-performance liquid chromatography (HPLC) analysis of rat urine and rat and mouse tissues indicated that absorbed AA was rapidly metabolized by the ß-oxidation pathway of propionate catabolism. No unchanged AA was detected; however, several metabolites that were more polar than AA were measured, including 3-hydroxypropionate.

The presented results are consistent with the incorporation of AA into a secondary pathway for propionic acid metabolism in which 3 -hydroxypropionate is an intermediate. In this pathway, AA is first converted to acrylyl-CoA which is subsequently oxidized to 3 -hydroxypropionate. 3 -Hydroxypropionate is, in turn, metabolized to acetate and CO_2 via malonic semialdehyde. The resultant acetate is then incorporated into intermediary metabolism. This pathway has been reported to be a major pathway for the metabolism of propionic acid in various insect and plant species, but is a secondary pathway in mammals.

On the other hand, reaction with reduced glutathion does not play a major role in the detoxification and metabolism of acrylic acid.

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. The CFD-PBPK model simulations indicated that the olfactory epithelium of the human nasal cavity is exposed to two- to threefold lower tissue concentrations of a representative inhaled organic acid vapour, acrylic acid, than the olfactory epithelium of the rodent nasal cavity when the exposure conditions are the same. The magnitude of this difference varies somewhat with the specific exposure scenario that is simulated. The increased olfactory tissue dose in rats relative to humans may be attributed to the large rodent olfactory surface area (greater than 50% ofthe nasal cavity) and its highly susceptible location (particularly, a projection of olfactory epithelium extending anteriorly in the dorsal meatus region). In contrast, human olfactory epithelium occupies a much smaller surface area (less than 5% of the nasal cavity), and it is in a much less accessible dorsal posterior location. In addition, CFD simulations indicated that human olfactory epithelium is poorly ventilated relative to rodent olfactory epithelium. These studies suggest that the human olfactory epithelium is protected from irritating acidic vapours significantly better than rat olfactory epithelium due to substantive differences in nasal anatomy and nasal air flow.

Discussion on bioaccumulation potential result:

<u>In Vivo Studies</u>:

C3H mice and Fischer 344 rats, respectively, were treated by gavage (40 or 150 mg/kg bw) with [1-^{14}C]-acrylic acid. Mice rapidly absorbedand 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 it was slower from fat. The disposition of orally administered acrylic acid in rats was similar to the results obtained from mice. High-performance liquid chromatography (HPLC) analysis of rat urine and rat and mouse tissues indicated that absorbed AA was rapidly metabolized by the ß-oxidation pathway of propionate catabolism. No unchanged AA was detected 1 h after oral administration; however,several metabolites that were more polar than AA were measured, including 3-hydroxypropionate. Neither AA nor its metabolites were detected at later times after oral administration (Black et al., 1995).

Sprague-Dawley 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%, muscle 6.5-7.5% and blood 0.8-1.1%) (De Bethizy et al., 1987).

The HPLC profile of metabolites observed in the urine of rats indicated two major metabolites. One of the major metabolites co-eluted with 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 leading to the conclusion that AA is not epoxidized to 2,3-epoxypropionic acid in vivo. Thisresult was supported by an in vitro study. Hepatic microsomes were prepared using conventional methods from rats and incubations werestarted 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 addition, Glutathione Depletion Studies were conducted in rats that were administered doses of 4, 40, 400 or 1000 mg/kg bw AA by gavage. One hour following oral administration of acrylic acid in rats a significant depletion of NPSH in the glandular stomach was reportedat doses above 4 mg/kg bw. In the forestomach NPSH depletion occurred at a dose of 1000 mg/kg bw. No significant effect of acrylic acidon NPSH in the blood or liver was observed (DeBethizy et al., 1987).

<u>In Vitro Studies</u>:

Dow Chemical (1979) have studied the metabolism of acrylic acid in rat tissue homogenates. Acrylic acid did not react with reduced glutathione either in the presence or absence of the soluble enzyme fraction. Non-protein sulfhydryl concentrations were not appreciably lower in blood after addition of acrylic acid in vitro (Dow Chemical, 1979; Miller et al., 1981).

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 of AA (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 2890, 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).

Computational Modeling Data:

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). This study provides a scientific basis for interspecies extrapolation of nasal olfactory irritants from rodents to humans. By using a series of short-term in vivo studies, in vitro studies with nasal explants, and computer modeling, regional nasal tissue dose estimates were made and comparisons of tissue doses between species were conducted. To make these comparisons, this study assumes that human and rodent olfactory epithelium have similar susceptibility to the cytotoxic effects of organic acids based on similar histological structure and common mode of action considerations. Interspecies differences in susceptibility to the toxic effects of acidic vapours are therefore assumed to be driven primarily by differences in nasal tissue concentrations that result from regional differences in nasal air flow patterns relative to the species-specific distribution of olfactory epithelium in the nasal cavity.

The rodent model uses two olfactory compartments to incorporate both the olfactory epithelium in the projection extending along the dorsalmeatus 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).

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 cm2/sec; air minute volumes: 250 mL/min (rat), 7500 mL/min (human); bloodflow to nasal cavity (human) estimated). 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).

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), 7500 mL/min (human)).

The acute inhalation, and in vitro studies have demonstrated that the nasal olfactory epithelium is the most sensitive tissue to the effects of inhalation exposure to organic acids and that the sustentacular cells are the most sensitive cell type of this epithelium. The CFD-PBPK model simulations indicated that the olfactory epithelium of the human nasal cavity is exposed to two- to threefold lower tissue concentrations of a representative inhaled organic acid vapour, acrylic acid, than the olfactory epithelium of the rodent nasal cavity when the exposure conditions are the same. The magnitude of this difference varies somewhat with the specific exposure scenario that is

simulated. The increased olfactory tissue dose in rats relative to humans may be attributed to the large rodent olfactory surface area (greater than 50% of the nasal cavity) and its highly susceptible location (particularly, a projection of olfactory epithelium extending anteriorly in the dorsal meatus region). In contrast, human olfactory epithelium occupies a much smaller surface area (less than 5% of thenasal cavity), and it is in a much less accessible dorsal posterior location. In addition, CFD simulations indicated that human olfactory epithelium is poorly ventilated relative to rodent olfactory epithelium. These studies suggest that the human olfactory epithelium is protected from irritating acidic vapours significantly better than rat olfactory epithelium due to substantive differences in nasal anatomy and nasal air flow.

Discussion on absorption rate:

The absorption of [^{14}C]-acrylic acid from acetone, water, and phosphate buffer was measured through human and mouse skin in vitro. Membranes were mounted in glass diffusion cells and acrylic acid was applied in each solvent at 0.01 %, 0.1 %, 1 %, and 4 %, respectively (100 µL/cm^2) under occlusive conditions. Samples were taken from the receptor solutions at recorded times, between 0 and 32 hr, and assayed for 14C content which was regarded as equivalent to acrylic acid. Steady state absorption rates were calculated to be between
0.007 µg/cm^2/hr (human, 0.01 % AA in phosphate buffer) and 201 µg/cm^2/hr (mouse, 4 % AA in acetone). Thus, absorption rates were influenced by the vehicle (acetone > water > phosphate buffer) and were proportional to the applied concentration in each vehicle. Mouseskin was 3 times more permeable than human skin under the conditions of this in vitro study (BAMM 1988).

C3H mice and Fischer 344 rats, respectively, were treated dermally (10 or 40 mg/kg bw in acetone) with [1-^{14}C]-acrylic acid. 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. Results from an in vitro experiment with rat skin (Frantz cell) showedthat at least 60 % of the applied dose evaporated and about 25% was absorbed, confirming the in vivo results. High-performance liquid chromatography (HPLC) analysis of rat urine and rat and mouse tissues indicated that absorbed AA was rapidly metabolized by the ß-oxidation pathway of propionate catabolism (Black et al., 1995).

Acute toxicity:

LD50(Oral, Rat):	ca. 1500 mg/kg bw
LD50(Dermal, Rabbit):	ca. 640 mg/kg bw
LC50(Inhalation, Rat):	> 5.1 mg/L air (analytical) (male/female)
Skin corrosion/Irritation:	Causes severe skin burns and eye damage.
Serious eye damage/irritation:	Causes serious eye damage.
Respiratory or skin sensitization:	Not classified
Germ cell mutagenicity:	Not classified
Carcinogenicity:	Not classified
Reproductive toxicity:	Not classified
STOT- single exposure:	May cause respiratory irritation.
STOT-repeated exposure:	Not classified

Aspiration hazard: Not classified

Section 12 Ecological information

12.1 Toxicity:

Acute toxicity		Time	Species	Method	Remarks
LC50	27 mg/L	96h	*Salmo gairdneri*	EPA OTS 797.1400	1 (reliable without restriction) key study experimental result
LC50	222 mg/L	96h	*Brachydanio rerio*	EU Method C.1	2 (reliable with restrictions) supporting study experimental result
LC50	236 mg/L	96h	*Cyprinodon variegatus*	OECD Guideline 203	2 (reliable with restrictions) supporting study experimental result
EC50	95 mg/L	48h	*Daphnia magna*	EPA OTS 797.1300	1 (reliable without restriction) key study experimental result
EC50	47 mg/L	48h	*Daphnia magna*	EU Method C.2	2 (reliable with restrictions) supporting study experimental result
EC50	97 mg/L	48h	*Mysidopsis bahia*	EPA OTS 797.1930	1 (reliable without restriction) key study experimental result
EC50	0.13 mg/L	72h	*Scenedesmus subspicatus*	EU Method C.3	1 (reliable without restriction) weight of evidence experimental result
EC50	0.205 mg/L	72h	*Scenedesmus subspicatus*	EU Method C.3	1 (reliable without restriction) weight of evidence experimental result
EC50	0.14 mg/L	72h	*Selenastrum capricornutum*	EPA OTS 797.1050	2 (reliable with restrictions) weight of evidence experimental result

12.2 Persistence and degradability: Readily biodegradable

12.3 Bioaccumulative potential: No indication for a potential for bioaccumulation

12.4 Mobility in soil: Very toxic to aquatic life.

12.5 Results of PBT&vPvB assessment: Not a PBT/vPvB.

12.6 Other adverse effects: Not available

Section 13 Disposal considerations

13.1 Waste treatment methods This product, when being disposed of in its unused and uncontaminated state should be treated asa hazardous waste according to EC Directive 91/689/EEC. Any disposal practices must be in compliance with all national and provincial laws and any municipal or local by-laws governing hazardous waste. For used, contaminated and residual materials additional evaluations may be required. Do not dump into any sewers, on the ground, or into any body of water.

Section 14 Transport information

	Land transport(ADR/RID)	Sea transport (IMDG)	Air transport (ICAO/IATA)
UN-Number:	UN2218	UN2218	UN2218
UN Proper shipping name:	Acrylic acid, stabilized	Acrylic acid, stabilized	Acrylic acid, stabilized
Transport hazard Class:	8+3	8+3	8+3
Packaging group:	II	II	II
Environmental hazards:	No	No	No
Special precautions for user:	See section 2.2	See section 2.2	See section 2.2
Transport in bulk according to Annex II of MARPOL 73/78 and the IBC Code	IBC 02	IBC 02	IBC 02

Section 15 Regulation information

15.1 Safety, health and environmental regulations/legislation specific for the substance or mixture

Relevant information regarding authorization: Not applicable.

Relevant information regarding restriction: Not applicable.

Other EU regulations: Employment restrictions concerning young person must be observed. Foruse only by technically qualified individuals.

Other National regulations: Not applicable

15.2 Chemical Safety Assessment has been carriedout? YES [X] NO [ ]

Section 16 Other information

16.1 Indication of changes

Version 1.1 Amended by (EU) 2015/830

Version 2.0 Placed exposure scenarios after section 16.

16.2 Training instructions:

Not applicable.

16.3 Further information:

This information is based upon the present state of our knowledge. This SDS has been compiled and is solely intended for this product.

16.4 Notice to reader:

Employers should use this information only as a supplement to other information gathered by them, and should make independent judgment of suitability of this information to ensure proper use and protect the health and safety of employees. This information is furnished without warranty, and any use of the product not in conformance with this Safety Data Sheet, or in combination with any other product or process, is the responsibility of the user.