SIDS INITIAL ASSESSMENT PROFILE

CAS No.	79-10-7
Chemical Name	2-Propenoic acid (Acrylic acid)
Structural Formula	$C_3H_4O_2$

RECOMMENDATIONS

The chemical is a candidate for further work (environment and consumer).
Risk reduction measures are recommended for the workers.

SUMMARY CONCLUSIONS OF THE SIAR

Human Health

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. The extent of absorption depends on pH and solvent with direct dependence on substance concentration. In mice acrylic acid is rapidly and completely metabolised mainly in liver and kidney via the normal catabolic pathways of beta-oxidation. Elimination preferably occurs as carbon dioxide.

Pure acrylic acid is a very reactive chemical and accordingly exhibits severe corrosive properties in contact with biological material. Thus, acrylic acid causes acute harmful effects by oral and dermal exposure. Oral LD50 values for rats cover a range from 140 up to 1400 mg/kg bw depending on the concentration of the test substance. An oral LD50 of 1350 mg/kg bw 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 LD50 of 640 mg/kg bw was determined for rabbits (with undiluted acrylic acid). Acute inhalation toxicity is low because acrylic acid interacts with humidity of the air prior to reaching the depth of the respiratory tract. LC50 values of 3.6 to >5.1 mg/l/4 hours have been determined.

Workplace data demonstrate that acrylic acid causes skin corrosion and irritation of the respiratory tract in humans. In tests with rabbits the pure acid caused severe burns to skin and eyes. Severe ocular damage caused by acrylic acid cannot be avoided by neutralizing the acid.

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

Repeated oral and inhalation exposure of acrylic acid to rats and mice resulted in dose related severe effects. Gavage on 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 subchronic and chronic drinking water studies. Reduced palatability (decreased water consumption) and unspecific signs of toxicity (decreased food consumption, body weight gain) at dosages >2000 ppm (100 mg/kg bw/d in male rats, 150 mg/kg bw/d in females) were observed. In a 90-day inhalation study, acrylic acid induced degenerative lesions on the olfactory mucosa in mice at 5 ppm (0.015 mg/l) and in rats at 75 ppm (0.221 mg/l). Mice seemed to be more sensitive than rats, thus a LOAEC of 5 ppm (0.015 mg/l) was derived for local effects. Long term dermal exposure at concentrations >1 % resulted in skin irritation.

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. In the mouse lymphoma assay small colonies were induced preferentially, thus 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.

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.

In oral studies on 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. No prenatal developmental toxicity was observed in rats and rabbits following inhalation exposure.

Environment

AA is fully miscible in water, has a vapour pressure of 3.8 hPa, and a log Pow of 0.46. The environmental behaviour of AA is determined by its range of 40 - 156 hours atmospheric half life and very low volatility. AA is readily biodegradable. Hydrolysis is not significant at all tested pHs (3, 7, 11). The average Kp 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 waste water treatment plants (WWTPs) 87.3 % of the substance are estimated to be removed entirely by biodegradation.

For fish, three valid results from acute tests are currently available. *Oncorhynchus mykiss* was found to be most sensitive, the recorded 96h-LC50 is 27 mg/l. For invertebrates, acute and long-term studies on *Daphnia magna* had been conducted. A 48h-EC50 of 47 mg/l and a 21d NOEC of 7 mg/l was obtained. 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 72h-EC50 is 0.13 mg/l and the 72h-EC10 is 0.03 mg/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. PNECaqua = 3 µg/l.

The derivation of a PNEC for microorganisms is based on results from tests on cell multiplication inhibition with protozoa and bacteria. For three protozoa tests NOEC values between 0.9 mg/l and 41 mg/l are reported, for a bacterial test with activated sludge a 30min-NOEC of 100 mg/l is reported. Applying an assessment factor of 1 for the protozoan species, the PNECmicroorganisms 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.

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 28d-NOEC of 100 mg/kg bw. With an assessment factor of 1,000 a PNECsoil of 0.1 mg/kg would result.

Exposure

In the European Union acrylic acid (hereafter referred to as AA) is produced and isolated as chemical intermediate. According to industry statements 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 either processed directly into a polyacrylate or polymerised via the intermediate stage of an acrylate ester.

Furthermore, acrylic acid is used as an ingredient and occurs as residual monomer in consumer products like adhesives, paints, binding agents and printing inks. Among the homo- and copolymerisates 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. 99% of the acrylate esters are n-butyl, ethyl, methyl and 2-ethylhexyl acrylates, of which butyl acrylate predominates quantitatively. Identical to glacial AA, these acrylic esters serve as commercial products, which are further processed both on-site and by external downstream users.

Releases of AA into the environment are to be expected during production and processing mainly via waste water and less amounts via exhaust gases. Regarding the formulation step, relevant releases may possibly occur during formulation of polymer dispersions. Residual monomeric AA-contents, which are the basis for release estimations from different polymeric products, are reported to range between 0.0002 and 0.2 %. 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.

Occupational exposure occurs during production and further processing and during manufacture and use of adhesives.

NATURE OF FURTHER WORK RECOMMENDED

This substance is agreed in the European Union Risk Assessment Programme under Regulation EEC/793/93, with the following conclusions:

Environment:

There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account:

Acrylic acid (AA) represents, based on the present data configuration, a potential risk to the local aquatic environment from wet polymerisation processes including wet production of SAP (super absorber polymers) by downstream users of monomeric AA (default calculations and three known sites).

Although an improvement of the data configuration (i.e. effluent measurements and/or site specific data on flow rates for the whole range of relevant sites) may in principle be possible, it is judged to be unlikely that representative monitoring data from the downstream users can be obtained sufficiently complete 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 have been reported. These data indicate that high effluent concentrations cannot be excluded, if certain types of process engineering are applied. On the other hand, application of waste water reutilization / recycling systems is known to warrant zero emission to hydrosphere at a number of downstream user sites which are processing about 50 % of AA used externally for SAP production and about 12 % of AA used externally in wet polymerisation processes. Measures to be applied for limiting the risk to the local aquatic environment are supposed to be also protective for municipal waste water 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. Quantitative extrapolation to other construction sites seems difficult, but similar conditions might be anticipated. Dependent on the local circumstances appropriate measures have to be chosen.

Acrylic acid (AA) represents, based on the present data configuration, a potential risk to municipal waste water treatment plants for the downstream use scenarios of SAP production (default calculation and highest site specific PECwwtp) and wet polymerisation (default calculation and two known sites). However, possible further testing to

refine the data configuration is postponed, since risk reduction measures necessary to remove concern for surface water will also cover the protection of municipal waste water treatment plants.

Human Health:

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

Worker: There is a need for limiting the risks; risk reduction measures which are already being applied shall be taken into account.

The occupational risk assessment comes to the conclusion that additional risk reduction measures are necessary for inhalation exposure in several scenarios. The relevant toxicological endpoints via inhalation are irritation and repeated dose toxicity (local and systemic effects).