BASIC ACRYLIC MONOMER MANUFACTURERS, INC.

SUBSTANCE REVIEW: ACRYLIC ACID

(Last Updated 5/7/12)

Disclaimer

Substance	Acronym	CAS Number
Acrylic acid	AA	79-10-7

Physicochemical Properties

Property	Results
Physical state at 20°C and 1013 hPa	Liquid Odor: acrid, pungent Color: colorless
Melting / freezing point	13°C
Boiling point	141°C at 1013 hPa
Relative density	1.05 g/cm^3
Vapor pressure	5.29 hPa at 25°C
Surface tension	69.6 mN/m at 20°C and 1000 mg/L
Water solubility	1000 g/l at 25°C
Partition coefficient n-octanol/water (log value)	0.46 @ 25°C
Flash point	48.5°C at 1013 hPa
Flammability	Flammable liquid cat. 3 The substance has no pyrophoric properties and does not liberate flammable gases on contact with water.
Explosive properties	non explosive
Self-ignition temperature	438°C at 1013 hPa
Oxidizing properties	no oxidizing properties
Granulometry	not applicable
Stability in organic solvents and identity of relevant degradation products	not applicable
Dissociation constant	4.26 at 25°C
Viscosity	1.149 mPa.s (dynamic) at 25°C

Environmental Fate

In contact with water, AA will be essentially stable to hydrolysis. Photodegradation in air will proceed slowly.

In surface water, sewage treatment plants and soil rapid degradation is expected, since AA was readily biodegradable in several OECD 301-Screening tests. In a well-documented study on biodegradation in soil, performed according to U. S. EPA Pesticide Assessment Guidelines, Subdivision N, § 162- 1, AA was rapidly metabolized under aerobic conditions. After 3 days no AA was detected in soil extracts. The half-life for AA under these conditions was estimated to be less than 1 day. From the presented simulation test in soil it can be concluded that AA is readily biodegradable in this soil type (sandy loam).

Based on an experimental log Pow and subsequently calculated BCF, a potential for bioaccumulation is not expected. Adsorption of AA to the solid soil phase is not expected.

Fugacity model calculations (Mackay Level I) revealed the hydrosphere as the main target compartment for distribution which is also indicated by the substance's physicochemical properties.

Ecotoxicity

The LC50-/EC50-values for freshwater fish and invertebrates range from 27 mg/L (fish) to 47 mg/L (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 the NOECs for maternal survival were 3.8 and 7 mg/L, respectively.

Acute and long-term test results reveal that algae are the most sensitive aquatic organisms. Their algal EC50 and NOEC values are more than two orders of magnitude lower than those for species of other trophic levels indicating a specific toxicity to algae.

For growth rate reduction, the lowest EC10 value derived in two tests was 0.03 mg/L for Scenedesmus subspicatus. The respective values based on biomass reduction are < 0.01 mg/L.

HUMAN HEALTH EFFECTS

Acute Toxicity

Acrylic acid is of moderate toxicity after a single ingestion and after short-term inhalation. AA is of low toxicity after short-term skin contact to non-corrosive concentrations.

- Oral: LD50 = 146-1405 mg/kg bw (rat) depending on the concentration tested
- Dermal: LD50 > 2000 mg/kg bw (rabbit)
- Inhalation: LC50 > 5.1 mg/L (rat, vapor saturated atmosphere)

Irritation/Sensitization

Acrylic acid is highly corrosive to skin and eyes. AA may be irritating/corrosive to the respiratory tract. AA does not cause skin sensitization in animals. Respiratory sensitisation has not been observed in humans

Repeated Dose Toxicity

In a subchronic study with Fischer 344 rats following administration of AA in the drinking water for 90 days, the NOAEL was determined to be 83 mg/kg bw/day. Following chronic exposure to AA in the drinking water, the NOAEL for male Wistar rats was 40 mg/kg bw/day and female rats was 375 mg/kg bw/day. In a 90-days subchronic study in rats and mice by the inhalation route, the NOAEC for local effects on the nasal epithelium was 74 mg/m^3 in rats. No NOAEC for local effects was derived in mice, the respective LOAEC was 15 mg/m^3. The systemic NOAEC in rats and male mice was 221 mg/m^3, and in female mice 15 mg/m^3.

Genetic Toxicity

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

Developmental/Reproductive Toxicity

In oral reproductive toxicity studies (rats) no effects on reproductive function (i.e. fertility) were observed. The NOAEL for reproductive function was 460 mg/kg bw/d.

Following administration of AA in the drinking water to Wistar rats some signs of postnatal developmental toxicity (retarded body weight gain of the pups) were seen, however only 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 an OECD TG 2-generation study in rats. No prenatal developmental toxicity was observed (rats and rabbits, inhalation), even at concentration levels that produced some signs of maternal toxicity. No specific teratogenic potential could be revealed for dose levels up to and including 360 ppm (rats) (= approx. 1.08 mg/L) and 225 ppm (rabbits) (= approx. 0.673 mg/L), respectively. According to the present database AA does not show any potential to cause toxicity to reproduction.

Carcinogenicity

Acrylic acid showed no evidence of carcinogenicity in a 2-year drinking water study in Wistar rats up to the highest dose tested of 78 mg/kg bw/day. In two dermal carcinogenicity studies in three mice strains (C3H/HeJ, C3H/HeN Hsd BR and Hsd:(ICR)BR), the frequency of skin tumors was not elevated compared to the vehicle controls.

Toxicokinetics

Following oral administration of [^{14}C]-AA 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 AA-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 glutathione does not play a major role in the detoxification and metabolism of AA.

A hybrid CFD-PBPK inhalation model was constructed with the aim to evaluate the relationship between inhaled AA vapor 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 vapor, AA, 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 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 vapors significantly better than rat olfactory epithelium due to substantive differences in nasal anatomy and nasal air flow.

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