BASIC ACRYLIC MONOMER MANUFACTURERS, INC.

SUBSTANCE REVIEW: N-BUTYL ACRYLATE

(Last Updated: 5/7/12)

Disclaimer

Substance	Acronym	CAS Number
n-Butyl acrylate	nBA	141-32-2

Physicochemical Properties

Property	Value
Physical state at 20°C and 1013 hPa	Liquid Color: colorless Odor: pungent
Melting / freezing point	-64.6°C
Boiling point	147°C at 1013.25 hPa
Relative density	0.9 g/cm^3 @ atmospheric pressure
Vapor pressure	5 hPa at 22.5°C
Surface tension	not surface active
Water solubility	1.7 g/l at 20°C
Partition coefficient n-octanol/water (log value)	Log Pow = 2.38 at 20°C
Flash point	37°C
Flammability	Flammable upon ignition. The substance has no pyrophoric properties and does not liberate flammable gases on contact with water.
Explosive properties	non explosive
Self-ignition temperature	565 K 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	not applicable
Viscosity	0.88 mPa.s at 20°C: (dynamic)

Environmental Fate

In contact with water, nBA will hydrolyze very slowly. Photodegradation in air will proceed slowly, too. In water, sewage treatment plants and soil rapid degradation is expected, since n-BA was readily biodegradable in an OECD 310-Screening test. Based on an experimental log Pow and calculated BCF, a potential for bioaccumulation is not expected. Adsorption of nBA to the solid soil phase is not expected. The estimated Koc indicates that nBA will exhibit a medium to high mobility in soil.

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

Ecotoxicity

When evaluated as a group, the acrylate esters have similar ecotoxicity data.LC50 values in freshwater fish ranged from 1.81 and 5.2 mg/L, EC50 values in freshwater invertebrates (Daphnia magna) were between 1.3 and 8.74 mg/L, and EC50 values in freshwater algae were between 1.71 and 14.6 mg/L, respectively. Thus, effect values were all in the same range of concentrations with Daphnia magna as the most sensitive freshwater species by a narrow margin. A 21-day chronic life-cycle study with Daphnia magna is available with ethyl acrylate with a respective NOEC of 0.19 mg/L, and another with nBA with a NOEC of 0.136 mg/L. In addition, several NOEC values from studies in algal species are available ranging from 0.45 to 3.85 mg/L.

HUMAN HEALTH EFFECTS

Acute Toxicity
n-Butyl acrylate is of low toxicity after a single ingestion and after a short-term skin contact. NBA is of moderate toxicity after a short-term inhalation.

- Oral: LD50 = 3150 mg/kg bw (rat)
- Dermal: LD50 2000 - 3024 mg/kg bw (rabbit)
- Inhalation: LC50 = 10.3 mg/L (rat)

Irritation/Sensitization
n-Butyl acrylate is irritating to eyes, respiratory system and skin. After repeated skin contact with nBA sensitization is possible.

Repeated Dose Toxicity
In a chronic (2 years) repeated dose study by the inhalation route a LOAEC of 86 mg/m³ was determined in rats for local effects (nasal mucosa irritation). The respective NOAEC for systemic effects was 773 mg/m³. After subchronic exposure by vapor inhalation over 3 months the NOAEC for systemic and toxic effects was 570 mg/m³ and the LOAEC was 1110 mg/m³ based on body weight decrease, clinico-chemical changes, and changed organ weights. The NOAEC for local effects (histological changes in the nasal mucosa and olfactory epithelium) was 111 mg/m³ and the LOAEC is 570 mg/m³.

Following a 90-day oral administration of nBA in the drinking water, the
NOAEL was 84 (male) and 111 (female) mg/kg bw/d for F344 rats, respectively. This dose level administered in drinking water approaches the maximum solubility limit of nBA in water and thus was the highest concentration that could be reasonably given.

Genetic Toxicity

In vitro, nBA was negative in the Ames Assay with and without metabolic activation. An in vitro micronucleus assay and an in vitro UDS assay in Syrian hamster embryo fibroblasts were both negative without metabolic activation. In vivo, nBA showed no genotoxic (cytogenetic) effects after vapor inhalation exposure in rats and hamsters.

Developmental/Reproductive Toxicity

The results of subchronic and chronic animal studies gave no indication of a fertility impairing effect caused by nBA. In addition, there are data from the structural analogue methyl acrylate. In a two-generation study in which groups of rats were whole-body exposed to methyl acrylate vapors, no effects on reproductive function (i.e. fertility) were observed. The NOAEC for reproductive function was 75 ppm (= ca. 268 mg/m³).

At concentrations where no maternal toxicity was observed, nBA did not cause developmental toxicity or teratogenicity. A developmental toxicity study in rabbits conducted with the structural analogue methyl acrylate did not give any indication for a developmental toxic or teratogenic effect. No teratogenicity occurred after inhalation of nBA in animal studies.

In high maternal toxic vapor concentrations only, nBA caused fetotoxic effects. After oral gavage at very high doses to mice which caused severe maternal toxicity, nBA caused malformations. Those effects are considered secondary to the maternal toxicity.

Carcinogenicity

n-Butyl acrylate showed no evidence of carcinogenicity in a 2-year vapor inhalation study in Sprague-Dawley rats up to the highest tested dose (135 ppm = 773 mg/m³) and in a lifetime skin painting study in C3H/HeJ mice at approx. 8 mg/kg bw.

Toxicokinetics

After oral and i.v. administration, nBA was rapidly absorbed and metabolized in male rats. The major portion of n-BA was hydrolyzed by carboxyesterase to acrylic acid and n-butanol. The subsequent metabolism follows that for acrylic acid, and involves metabolism to carbon dioxide via the propionate degradation pathway (acrylic acid --> 3-hydroxypropionic acid --> malonyl semialdehyde --> acetyl S CoA --> --> tricarboxylic acid cycle --> --> CO_2). Metabolism of n-butanol proceeds via the alcohol and aldehyde dehydrogenase pathway. A smaller portion of the administered n-BA was conjugated with endogenous GSH to be subsequently excreted as mercapturic acids in the urine.

Disclaimer

This document is not intended to be comprehensive. It is provided solely as background information and should not substitute for an up-to-date Safety Data Sheet or research should specific regulatory or other legal questions arise. It is not intended to be a statement of legal requirements when using or handling acrylates. Although the information is believed to be accurate as of the last update, new information may become available and regulations frequently change, and no warranty, expressed or implied, is made concerning the contents. In addition, many states and localities adopt their own regulations, which are not covered by this summary or on the BAMM website. In all events, the user should consult applicable laws and regulations, as well as their supplier's Safety Data Sheet, for current information and requirements. **NO WARRANTY OF FITNESS FOR ANY PARTICULAR PURPOSE, WARRANTY OF MERCHANTABILITY, OR ANY OTHER WARRANTY, EXPRESS OR IMPLIED, IS MADE CONCERNING THE INFORMATION PROVIDED HEREIN.**