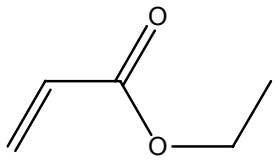
SIDS INITIAL ASSESSMENT PROFILE

CAS No.	140-88-5
Chemical Name	Ethyl Acrylate (2-Propenoic Acid, Ethyl Ester)
Structural Formula	

SUMMARY CONCLUSIONS OF THE SIAR

Human Health

Ethyl acrylate is readily absorbed from the gastrointestinal tract and upper respiratory tract. Rapid metabolism occurs by two primary routes, carboxylesterase mediated hydrolysis of the ester linkage to acrylic acid and ethanol, and conjugation with glutathione. Both pathways serve to detoxify ethyl acrylate. Approximately 60% of the administered dose is excreted within 8 hours as CO_2.

Ethyl acrylate is slightly toxic following acute oral, dermal and inhalation exposure: LD_{50} rat (oral) = 1120 mg/kg body weight; LC_{50} rat (inhalation; vapor; 4 hour exposure) = 2180 ppm (9 mg/L); LD_{50} rabbit (dermal) = 3049 mg/kg body weight. In standard primary irritation studies, ethyl acrylate is a strong skin and eye irritant. Ethyl acrylate is considered likely to be a sensitizer and exposure to ethyl acrylate may result in cross-sensitization with other acrylates and methacrylates.

Repeated-dose studies confirm the irritant properties of ethyl acrylate with localized irritation, often severe, occurring at the site of contact for oral dosing, including forestomach tumors following chronic gavage dosing, and metaplasia or atrophy of the olfactory epithelium following inhalation exposure at concentrations greater than 5 ppm (0.02 mg/L). Repeated dose studies indicate that systemic toxicity, manifested primarily as body weight reduction, from oral or inhalation exposure to ethyl acrylate for periods up to 2 years, is minimal. No systemic toxicity was observed in oral (gavage or drinking water) studies below approximately 100 mg/kg/day for 90 days or 2 years. The LOAEC for systemic toxicity (decreased body weight) following 2 years of inhalation exposure was 75 ppm (0.31 mg/L) and the NOAEC for systemic toxicity was 25 ppm (0.10 mg/L). Nasal irritation, a site of contact effect, occurred at this concentration. The NOAEC for nasal irritation after 2 years of exposure was 5 ppm. Except for localized irritant effects, repeated exposure to ethyl acrylate in animal studies did not result in overt toxicity or specific organ toxicity.

Ethyl acrylate was not mutagenic to bacteria (*Salmonella* reverse mutation assay) *in vitro* and was not clastogenic in *in vivo* mouse micronucleus assays. Positive mutagenic activity in some *in vitro* assays occurred only at concentrations resulting in significant cell death. These assays are, therefore, considered inadequate for evaluation of the mutagenic potential of ethyl acrylate. Overall, ethyl acrylate is considered to pose no mutagenic hazard based on the available data.

Ethyl acrylate was not carcinogenic following exposure via inhalation at the highest concentration of 75 ppm (0.31 mg/L) or via drinking water at the highest concentration of 2000 ppm. Although forestomach tumors were observed in chronic gavage studies conducted by the National Toxicology Program (NTP), use of these studies for classification of ethyl acrylate carcinogenicity was determined by NTP to be scientifically unjustifiable. This was based on the conclusion that the forestomach tumors are not considered relevant and were seen only when ethyl acrylate was administered by gavage at high concentrations that induced marked local irritation and cellular proliferation. Therefore, ethyl acrylate was not considered to be carcinogenic in experimental animals; these same data were used in 1986 by IARC to classify ethyl acrylate in category 2B. Ethyl acrylate is scheduled for reevaluation by IARC.

Ethyl acrylate had no effect on reproductive organs following repeated exposures via oral or inhalation routes for up to 2 years. In a well-conducted inhalation study exposing pregnant rats from gestation day 6 to 20, maternal and fetal body weights were reduced only at the highest concentration and no developmental toxicity or teratogenicity was observed. The NOAEL for maternal and fetal toxicity was 100 ppm (0.41 mg/L) and the LOAEL was 200 ppm (0.82 mg/L). The NOAEL for developmental toxicity and teratogenicity was 200 ppm (0.82 mg/L), the highest exposure concentration tested. Based on the available animal studies, ethyl acrylate is not toxic to reproduction or development and is not teratogenic.

Environment

The water solubility of ethyl acrylate is 15 g/L (25°C) and specific gravity is 0.9234 g/cm^3 at 20°C. The measured log Kow is 1.18. The vapor pressure is 38 hPa at 20°C. The melting point is – 71.2°C and the boiling point is 99.4°C.

Ethyl acrylate is photodegraded by reaction with hydroxyl radicals in the atmosphere with a half-life of 11.8 hours (calculated). The hydrolysis rate of ethyl acrylate is pH dependent with hydrolysis rates of <1% and <2% after 28 days at pH 3 and pH 7, respectively. The hydrolysis half-life at pH 11 is 182 minutes.

Distribution modeling using Mackay Level I indicates that the main target compartment will be air (90%) with the remainder partitioning into water (10%). Fugacity model level III calculations with 100% of the ethyl acrylate release to air (from Toxic Release Inventory data) gives comparable results; the levels are: 94% (air), 5.6% (water), <1% (soil) and <0.1% (sediment).

A low bioaccumulation potential is expected based on the partition coefficient and other physical/chemical parameters. Ethyl acrylate attained 57.3% degradation within 28 days in a closed bottle test according to OECD Test Guideline 301 D. In a CO_2-Headspace test according to ISO 14593 (identical to OECD Test Guideline 310) ethyl acrylate was readily biodegradable (96% degradation after 28 days).

Ethyl acrylate is acutely toxic to aquatic organisms. The 96-hour LC_{50} for rainbow trout was 4.6 mg/L (measured), the 48-hour EC_{50} for *Daphnia magna* was 7.9 mg/L (measured) and the 96-hour EC_{50} value (growth rate) for algae (*Selenastrum capricornutum*) was 5.5 mg/L (measured). In a 21-day chronic study with *Daphnia magna*, the EC_{50} for mortality was 0.5 mg/L and the NOEC was 0.19 mg/L (measured).

Exposure

The production volume of ethyl acrylate is estimated to be 50,000 to 100,000 tonnes per year in Europe and 250,000 to 500,000 tonnes per year in North America. Ethyl acrylate is produced and primarily used in closed systems. Its principle use is for the production of homopolymers and copolymers with other monomers (e.g. acrylic acid and its salts, amides, etc.), which are then used in a variety of products including paints, binders, polishes and adhesives.

Environmental releases are minimal. In 2001, US TRI reporting indicated that virtually all ethyl acrylate releases were to the air compartment (185,000 pounds).

Extensive occupational exposure monitoring records are available which indicate that 8 hr TWAs for a variety of operations are below the regulatory/guideline values. End-use consumer products contain only trace levels of acrylic acid and esters (as a result of polymerization). Therefore, consumer exposure to acrylate monomers is low.

RECOMMENDATION AND RATIONALE FOR THE RECOMMENDATION AND NATURE OF FURTHER WORK RECOMMENDED

The chemical is currently of low priority for further work. The chemical possesses properties indicating a hazard for human health (possible skin sensitization; skin, eye and nasal irritation) and the environment. Based on data presented by the Sponsor Country, exposure to humans and the environment is anticipated to be low, and therefore this chemical is currently a low priority for further work. Countries may desire to investigate any exposure scenarios that were not presented by the Sponsor country.