

**SIDS INITIAL ASSESSMENT PROFILE**

<b>CAS No.</b>	67-64-1
<b>Chemical Name</b>	Acetone
<b>Structural Formula</b>	CH <sub>3</sub> -CO-CH <sub>3</sub>

**RECOMMENDATIONS**

The chemical is currently of low priority for further work.

**SUMMARY CONCLUSIONS OF THE SIAR****Human Health**

The acute toxicity is low. Acetone is not a skin irritant or sensitizer but is a defatting agent to the skin. Acetone is an eye irritant. The subchronic toxicity of acetone has been examined in mice and rats that were administered acetone in the drinking water and again in rats treated by oral gavage. Acetone-induced increases in relative kidney weight changes were observed in male and female rats used in the oral 13-week study. Acetone treatment caused increases in the relative liver weight in male and female rats that were not associated with histopathologic effects and the effects may have been associated with microsomal enzyme induction. Hematologic effects consistent with macrocytic anemia were also noted in male rats along with hyperpigmentation in the spleen. The most notable findings in the mice were increased liver and decreased spleen weights. Overall, the no-observed-effect-levels in the drinking water study were 1% for male rats (900 mg/kg/d) and male mice (2258 mg/kg/d), 2% for female mice (5945 mg/kg/d), and 5% for female rats (3100 mg/kg/d). For developmental effects, a statistically significant reduction in foetal weight, and a slight, but statistically significant increase in the percent incidence of later resorptions were seen in mice at 15,665 mg/m<sup>3</sup> and in rats at 26,100 mg/m<sup>3</sup>. The no-observable-effect level for developmental toxicity was determined to be 5220 mg/m<sup>3</sup> for both rats and mice. Teratogenic effects were not observed in rats and mice tested at 26,110 and 15,665 mg/m<sup>3</sup>, respectively. Lifetime dermal carcinogenicity studies in mice treated with up to 0.2 mL of acetone did not reveal any increase in organ tumor incidence relative to untreated control animals.

The scientific literature contains eight different studies that have measured either the neuro-behavioural performance or neurophysiological response of humans exposed to acetone. Effect levels ranging from about 600 to greater than 2375 mg/m<sup>3</sup> have been reported. Neurobehavioral studies with acetone-exposed employees have recently shown that 8-hr exposures in excess of 2375 mg/m<sup>3</sup> were not associated with any dose-related changes in response time, vigilance, or digit span scores. Clinical case studies, controlled human volunteer studies, animal research, and occupational field evaluations all indicate that the NOAEL for this effect is 2375 mg/m<sup>3</sup> or greater.

**Environment**

Acetone has been tested in a wide variety of aquatic and terrestrial species. Acute toxicity to fish ranges from an LC<sub>50</sub> of 6,070 mg/L for Brook trout to 15,000 mg/L for Fathead minnow. The lowest LC<sub>50</sub> for aquatic invertebrates is 2,100 mg/L, ranging to 16,700 mg/L. The NOEC's for toxicity to aquatic plants range from 5,400-7,500 mg/L. The chronic NOEC for Daphnia is 1,660 mg/L. Tests using Ring-neck pheasant and Japanese quail produced no adverse effects at 40,000 mg/kg. In summary, ecotoxicity testing shows that acetone exhibits a low order of toxicity.

An assessment factor of 100 was used to calculate a predicted no effect concentration (PNEC) for acetone in an

aqueous environment, because acute toxicity data were available for algae, crustaceans, and fish. The lowest PNEC value for these species was calculated to be 21 mg/L when using the  $LC_{50}$  value of 2100 mg/L for marine brine shrimp.

### **Exposure**

Worldwide production capacity of acetone was 3.8 million tonnes in 1995 with the actual volume produced being somewhat less at 3.7 million tonnes. Production capacity in the United States constituted about 33% (1.3 million tonnes) of the global capacity, while Western Europe and Asia (including Japan) were about 31% (1.2 million tonnes) and 19% (0.7 million tonnes), respectively. Major end uses of acetone can be divided into three separate categories as: i) a chemical feedstock, ii) a formulating solvent for commercial products, and iii) an industrial process solvent. Acetone can be found in wide variety of consumer and commercial products but only a few are known to contain high concentrations. These include paints and paint-related products, such as paint thinners, finger nail polish removers, automotive waxes and tar removers.

PECs have been derived based on the results from air and water monitoring data. The  $PEC_{local}$  (2500  $\mu\text{g/L}$  [water], 10,000  $\mu\text{g/m}^3$  [air]) and  $PEC_{global}$  (50  $\mu\text{g/L}$  [water], 10  $\mu\text{g/m}^3$  [air]) values are intended to represent plausible worst case environment concentrations on a global and regional scale.

High concentrations of acetone can be detected in a variety of occupational environments (up to 2876  $\text{mg/m}^3$  at cellulose acetate factory). The predominant route of both occupational and consumer exposure is through vapor inhalation. The estimated human exposure (EHE) value for workplace employees is 1780  $\text{mg/m}^3$ . Using a USEPA modelling programme entitled SCIES (Screening Consumers Inhalation Exposure Software), a scenario intended to represent a likely indoor consumer use of a product (45 min application of a spray contact adhesive that contained 21% acetone) predicted a short-term exposure (EHE) value of 900  $\text{mg/m}^3$  for the consumer use of the product.

### **NATURE OF FURTHER WORK RECOMMENDED**

None recommended.