SIDS INITIAL ASSESSMENT PROFILE

CAS No.	75-01-4
Chemical Name	Vinyl Chloride
Structural Formula	CH_2=CHCl

RECOMMENDATIONS

This chemical is currently of low priority for further work in the SIDS Program as human exposures are controlled due to the chemical's genotoxicity and cancer hazard and based upon OECD risk reduction measures.

SUMMARY CONCLUSIONS OF THE SIAR

Human Health

The primary route of exposure for vinyl chloride is by inhalation. Vinyl chloride is rapidly and well absorbed following inhalation or oral exposure, and is bioactivated by the liver. The acute toxicity (rat oral LD_{50} >4000 mg/kg; rat and mouse inhalation LC_{50} 390,000 mg/m^3 and 294,000 mg/m^3 respectively) is low. Anesthetic effects have been reported in humans at levels of 12000 ppm (30,720 mg/m^3 for a five minute exposure period. The NOAEL for inhalation exposure to rats, rabbits, guinea pigs or dogs is 50 ppm (128 mg/m^3) for 6 months. For oral repeated dose, the critical target organ is the liver (liver cell polymorphism) with a lifetime NOAEL in the rat of 0.13 mg/kg/day. Vinyl chloride (and/or its metabolites) produces DNA adducts and has been positive in gene mutation and chromosomal aberration assays. Chromosomal aberrations have also been observed in peripheral lymphocytes of exposed workers in some studies. Long term exposure in experimental animals and humans causes liver cancer (angiosarcoma). Vinyl chloride is a known human carcinogen. Cancer of the lymphopoietic system, connective tissues, and soft tissue have been associated with vinyl chloride exposure in some studies, but not others. In a combined reproductive/developmental study in rats the NOAEL for reproductive/developmental effects was 1,100 ppm (2816 mg/m^3), the highest dose tested. Human studies have not linked vinyl chloride exposure with negative reproductive outcomes.

Environment

Vinyl Chloride has a vapor pressure of 3330 hPa at 20^{0}C, a water solubility value of 1.1 g/l at 20^{0}C and a log P_{ow} of 1.58 at 22^{0}C. In the soil and water microorganism study, vinyl chloride was biodegraded at 30% after 40 days and 99% after 108 days, and has a low bioaccumulation potential. Environmental releases of vinyl chloride are almost exclusively to the air compartment. Fugacity modeling indicates that of the vinyl chloride released >99% will remain in the air compartment. The dominant removal process in the atmosphere is photoxidation with a calculated half-life of 2.2 – 2.7 days. The 96 hour LC_{50} ranges from 210 to > 1000mg/l for fish (four studies). The estimated QSAR value for algae EC_{50} (96hr) is 118 mg/L and the LC_{50} (48 hr) for daphnia is 196 mg/L. Toxic concentrations of vinyl chloride are not expected to be reached in aquatic systems based on low emissions, low bioaccumulation potential and high volatility.

Exposure

Vinyl chloride is a gas, which is manufactured in closed systems as an industrial intermediate - mainly for the production of polyvinyl chloride (PVC) and vinyl copolymers. North American production capacity in 1999 was

about 8.344 million metric tons and global capacity was 30.022 million metric tons. Workplace exposure is tightly controlled in the U.S. and other OECD countries. The most likely route for consumer and environmental exposure is inhalation of residual vinyl chloride monomer (VCM) present in PVC products, however, residual monomer levels in these products are highly regulated and tightly controlled to very low levels. Such products include food packaging, medical devices, PVC pipe, wire coatings, automotive interiors, exterior siding, interior vinyl floors, wall and furniture coverings, and toys. Vinyl chloride is present in the air near production facilities generally at levels <0.1 mg/m^3, and in ground water generally below the 0.001 ppm detection limit.

NATURE OF FURTHER WORK RECOMMENDED

No recommendation.