

SIDS INITIAL ASSESSMENT PROFILE

CAS No.	108-65-6
Chemical Name	1-Methoxy-2-propyl acetate
Structural Formula	$\text{CH}_3\text{O}-\text{CH}_2-\text{CH}(\text{CH}_3)-\text{O}-\text{COCH}_3$

RECOMMENDATIONS

The chemical is currently of low priority for further work.

SUMMARY CONCLUSIONS OF THE SIAR**Human Health**

It is anticipated that rapid and extensive hydrolysis of 1-Methoxy-2-propyl acetate also known as 2-methoxy-1-methylethyl acetate (PMA) will occur *in vivo* following either oral, inhalation or dermal exposures to yield the corresponding glycol ether, propylene glycol monomethyl ether (PM). Thus, it is presumed that no substantial differences in the systemic toxicities of PM or PMA will exist. In particular, dermal testing with PMA in rats suggests that any effects arising from PMA would be overestimated by using PM toxicity data.

Acute toxicity of this chemical is low in rodents because LD₅₀ values are greater than 5,000 mg/kg by oral or dermal routes and greater than 10,800mg/m³ by inhalation.

This chemical is slightly irritating to the eye, but not to the skin. PMA is not skin-sensitising in guinea pigs.

In a oral rat study carried out according to the OECD combined repeated dose and reproductive/developmental toxicity screening test [OECD TG 422], a dose of 1,000 mg/kg/day of PMA exerted some effects in only male rats. Blood examination revealed decreases in glucose and inorganic phosphorus and an increase in relative weight of the adrenals was also noted in males. However, such changes were not observed in females. Histopathological examination revealed none of the alteration of tissues at the highest dose group for both sexes. As such changes in males were considered not to be adverse effect, a NOAEL was considered to be 1,000 mg/kg bw/day for both sexes.

An inhalation study conducted for 6 hr/day, 5 day/week for 2 weeks using rats and mice at doses of 300, 1,000 or 3,000 ppm (1.62, 5.39 or 16.18 mg/L) demonstrated that haematology and clinical chemistry analyses revealed no treatment-related effect. However, the kidneys of all male rats and two of five females in the 3,000 ppm-exposure group appeared to be slightly reticulated at necropsy. The change noted in these animals was a slight increase in the eosinophilic granularity of the proximal convoluted tubules of the kidneys. The same slight renal change was also observed in one of five male rats at 1,000 ppm. Another detectable effect in rats and mice was slight-to-moderate degeneration of olfactory epithelium in the nasal cavities. A NOAEL for inhalation toxicity in rats was established at 300 ppm (1.62 mg/L) for males and at 1,000 ppm (5.39 mg/L) for females, whereas a NOAEL for inhalation toxicity in mice was not established because the lowest dose at 300 ppm induced a minimum effect on the nasal cavity of mice. The change in nasal cavity is likely caused by acetic acid from PMA hydrolysis at the exposure site.

In reproductive/developmental oral toxicity study [OECD TG 422], there were no statistically significant adverse effects on reproductive parameters and no evidence of malformations at any doses. Likewise, in developmental/teratogenicity inhalation study, there were no statistically significant adverse effects on reproductive and teratogenic parameters at any doses, although some systemic toxicities were observed in dams at 2,000 and 4,000 ppm. A NOAEL was established at 1,000 mg/kg bw/day for reproductive/developmental toxicity by gavage and at 4,000 ppm (22,464 mg/m³) for developmental/teratogenicity toxicity by inhalation, respectively.

Two bacterial mutation tests, unscheduled DNA synthesis in rat hepatocytes and chromosomal aberration test *in vitro* show negative results.

PMA tested in the animal studies contained approximately a maximum of 2 % of the beta-isomer.

Environment

PMA is readily biodegradable (OECD TG 301F: 99 % after 28 days). This chemical is stable to chemical hydrolysis in water at pH 4 and 7, whereas it is hydrolyzed at pH 9 with half-life of 8.10 days at 25 °C.

The toxicity to aquatic plants (algae; *Selenastrum capricornutum*) was >1,000mg/L for EC₅₀ (72 hr) and NOEC (72 hr). The acute toxicity data in fish (medaka; *Oryzias latipes*) were >100 mg/L for 96h LC₅₀, 63.5 mg/L for 14d LC₅₀ and 47.5 mg/L for 14d NOEC. In *Daphnia magna*, EC₅₀ (48h) for acute toxicity and NOEC (21-d reproduction) for chronic toxicity were 373 mg/L and ≥100 mg/L, respectively. When assessment factor of 100 was applied to the 14d LC₅₀ for medaka and the chronic toxicity for *Daphnia*, PNECs were calculated as 0.635 and ≥1.0 mg/L, respectively. The lowest PNEC was thus determined to be 0.635 mg/L.

Exposure

The production volume in Japan was approximately 15,000 tonnes/year in use, while estimated global production is 100,000-500,000 tonnes/year according to IUCLID 1999. Commercially available PMA contains less than 0.5 % of the β-isomer. PMA has a variety of uses including as a solvent for paints, inks, lacquers, varnishes and cleaners, coatings and ink-removers, and as a pesticide inert.

Generic fugacity models (Level III Fugacity Model and Unit World Equilibrium Model) show this chemical would be distributed mainly to water if it was released into water.

As this chemical is contained as a solvent for specific paint products and used in industrial sites, user exposure may take place in the industry and consumer. PMA occurred in 366 chemical products on American market according to MSDS-OHS 2000.

NATURE OF FURTHER WORK RECOMMENDED

No recommendation.