METHYL BROMIDE (Group 3)

A. Evidence for carcinogenicity to humans (*inadequate*)

Two cohort studies mention exposure to methyl bromide. In both study populations, exposure to a great number of other chemical compounds occurred, and, therefore, the slight excesses of some cancers found cannot be interpreted in terms of exposure to methyl bromide[1].

B. Evidence for carcinogenicity to animals (*limited*)

In one 90-day study by oral administration in rats, methyl bromide was reported to produce squamous-cell carcinomas of the forestomach[1]. In a second, 25-week study, it was found that early hyperplastic lesions of the forestomach regressed after discontinuation of treatment; one early carcinoma (1/11) developed after 25 weeks of continuous treatment by gavage[2].

C. Other relevant data

No data were available on the genetic and related effects of methyl bromide in humans.

Micronuclei were induced in the bone-marrow and peripheral blood cells of rats and mice following exposure to methyl bromide by inhalation. After treatment of mice with methyl bromide by different routes, DNA methylation of liver and spleen was observed. Methyl bromide induced sister chromatid exchanges in human lymphocytes *in vitro* and mutation in mouse lymphoma cells *in vitro*. It did not induce unscheduled DNA synthesis in

rat hepatocytes. Methyl bromide induced sex-linked recessive lethal mutations in *Drosophila* and was mutagenic to plants and bacteria[3].

References

[1]*IARC Monographs*, *41*, 187-212, 1987

[2]Boorman, G.A., Hong, H.L., Jameson, C.W., Yoshitomi, K. & Maronpot, R.R. (1986) Regression of methyl bromide-induced forestomach lesions in the rat. *Toxicol. appl. Pharmacol.*, *86*, 131-139

[3]*IARC Monographs*, *Suppl. 6*, 386-388, 1987