ALDRIN (Group 3)

A. Evidence for carcinogenicity to humans (inadequate)

Specific mention of aldrin in analytical epidemiological studies is limited to reports of follow-up of two cohorts of men employed in its manufacture in plants where dieldrin (see p. 196) and endrin (and, in one, telodrin) were also manufactured¹⁻⁴. In the most recent report of the first of these cohorts³, 232 of 233 exposed workers were successfully followed from four to 29 (mean, 24) years, with duration of exposure to pesticides varying between four and 27 (mean, 11) years. There were nine deaths from cancer with 12 expected (standardized mortality ratio [SMR], 75; 95% confidence interval, 25-125). In the second cohort⁴, 90% of 1155 men were followed for 13 years or more. Mortality from all cancers was not increased (SMR, 82; 56-116), although there were apparent increases in mortality from cancers of the oesophagus, rectum and liver, based on very small numbers.

B. Evidence for carcinogenicity to animals (limited)

Aldrin was tested for carcinogenicity by the oral route in mice and rats. In mice, it produced malignant liver neoplasms^{1,5}. In rats, the incidence of thyroid tumours was increased in exposed animals in one study⁵, but this could not be clearly associated with treatment; three other studies in rats gave negative results^{1,6} and one was inadequate¹.

C. Other relevant data

No data were available on the genetic and related effects of aldrin in humans. It did not induce dominant lethal mutations in mice. In single studies, it induced chromosomal aberrations in bone-marrow cells of rats and mice, but no micronuclei in bone-marrow cells of mice treated *in vivo*. It induced chromosomal aberrations in cultured human lymphocytes; studies of DNA damage in human and rodent cells *in vitro* were inconclusive. Aldrin inhibited intercellular communication in both human and rodent cell systems. It did not induce sex-linked recessive lethal mutations in *Drosophila* but was mutagenic to yeast. It was not mutagenic to bacteria and did not induce breakage of plasmid DNA⁷.

References

- ¹IARC Monographs, 5, 25-38, 1974
- ²Van Raalte, H.G.S. (1977) Human experience with dieldrin in perspective. *Ecotoxicol. environ. Saf.*, 1, 203-210
- ³Ribbens, P.H. (1985) Mortality study of industrial workers exposed to aldrin, dieldrin and endrin. *Int. Arch. occup. environ. Health*, 56, 75-79
- ⁴Ditraglia, D., Brown, D.P., Namekata, T. & Iverson, N. (1981) Mortality study of workers employed at organochlorine pesticide manufacturing plants. *Scand. J. Work Environ. Health*, 7 (Suppl. 4), 140-146
- ⁵National Cancer Institute (1978) Bioassays of Aldrin and Dieldrin for Possible Carcinogenicity (Tech. Rep. Ser. No. 21; DHEW Publ. No. (NIH) 78-821), Bethesda, MD, US Department of Health, Education, and Welfare

⁶Deichmann, W.B., MacDonald, W.E. & Lu, F.C. (1979) Effects of chronic aldrin feeding in two strains of female rats and a discussion on the risks of carcinogens in man. In: Deichmann, W.B., ed., Toxicology and Occupational Medicine, New York, Elsevier/North-Holland, pp. 407-413

⁷IARC Monographs, Suppl. 6, 57-59, 1987