

Aldrin and Dieldrin

Guideline

The maximum acceptable concentration (MAC) for aldrin and dieldrin in drinking water is 0.0007 mg/L (0.7 µg/L).

Identity, Use and Sources in the Environment

Aldrin (C₁₂H₈Cl₆) and dieldrin (C₁₂H₈Cl₆O) are both non-systemic chlorinated hydrocarbon insecticides. They were used in Canada for more than 25 years to control insects in crops and in domestic, forestry and industrial situations.¹

Periodic re-evaluations of aldrin and dieldrin by Agriculture Canada resulted in their decreased use, and registered uses of the compounds were very limited after the mid-1970s. The last registered use of aldrin and dieldrin in Canada by licensed pest control operators was restricted to subterranean termite control. No sales of aldrin or dieldrin in Canada have been reported since 1984. The last known manufacturer of aldrin and dieldrin, Shell International Chemical Co. (UK), ceased its production of the pesticides in 1989.^{2,3} Registration of aldrin and dieldrin in Canada was discontinued on 31 December 1990.¹

Aldrin and dieldrin have low water solubilities (0.027 mg/L for aldrin at 27°C and 0.186 mg/L for dieldrin at 20°C). Their vapour pressures at 20°C are 10.0 × 10⁻³ Pa for aldrin and 4.1 × 10⁻⁴ Pa for dieldrin. Reported log octanol-water partition coefficients for aldrin (7.4) and dieldrin (6.2)⁴ demonstrate their high potential for bioaccumulation.

In the soil, aldrin is converted by epoxidation to dieldrin. Dieldrin is more stable and highly persistent in the environment.⁵ Aldrin and dieldrin are both strongly sorbed to soil, especially to organic matter. Furthermore, aldrin and dieldrin are resistant to leaching,⁶⁻⁸ which leads to a low potential for groundwater contamination.

Long-range atmospheric transport has introduced measurable quantities of various organochlorine

compounds, including dieldrin, in the Canadian Arctic.⁹ Dieldrin's presence in the gas phase or on small particles with long atmospheric residence times, its low water solubility and its high biological and chemical stability enable it to move in the atmosphere from warmer to colder regions.^{9,10} The presence of dieldrin in Canadian regions where it has never been used raises concerns about our inability to control the movement of this persistent chemical in the environment.

Exposure

During 1976, samples of Ottawa drinking water were collected and analysed for organochlorine pesticides. The results showed low levels of aldrin (≤0.006 µg/L) and dieldrin (≤0.004 µg/L).¹¹ In the Niagara region, dieldrin was detected at three locations from 1979 to 1982 in both raw and treated water (detection limit not reported); frequencies of detection of up to 6% were reported, and the maximum concentration (0.015 µg/L) was recorded in a sample from Niagara-on-the-Lake.¹² From 1978 to 1985, trace levels of dieldrin were reported in four of 531 samples from 15 Alberta municipalities using surface water supplies (detection limit 0.01 µg/L).¹³

Low levels of aldrin and dieldrin (≤0.001 µg/L) were reported at 16 offshore stations in Lake Superior in June 1983.¹⁴ Two of 1400 samples collected from 38 sites in the Prairies from 1971 to 1977 contained detectable levels of aldrin (detection limit not reported); the maximum concentration, measured in a sample from the North Saskatchewan River, was 0.005 µg/L.¹⁵

Aldrin and dieldrin were among the pesticide residues analysed in the Total Diet Study in Canada from 1976 to 1978.¹⁶ The minimum detectable concentration of both pesticides in composite samples was 1 ppb. The results showed that more than half of the daily intake of dieldrin was attributable to dairy products. Table 1 shows the average daily dietary intake of dieldrin by Canadians over the period 1969 to 1978.

Table 1. Average daily dietary intake of dieldrin by Canadians¹⁶

	Dietary intake of dieldrin ($\mu\text{g}/\text{kg}$ bw per day)
Ottawa (1969)	0.057
Vancouver (1970)	0.016
Halifax (1971)	0.032
Winnipeg (1972)	0.021
Toronto (1973)	0.025
Canada (1974)	0.009
Canada (1976–1978)	0.002

The exposure of infants to persistent organochlorine pesticides was determined during a recent (1992) Canada-wide survey.¹⁷ Samples of human milk ($n=497$) were analysed for 24 chlorinated pesticides and various industrial organic contaminants. Dieldrin was detected in 94% of the human milk samples (detection limit 0.04 ng/g). The median dieldrin concentration in the human milk samples was 0.26 ng/g, compared with 0.35 ng/g in 1986.

Analytical Methods and Treatment Technology

Aldrin and dieldrin can be extracted from water samples with pentane¹⁸ or methylene chloride⁵ and then analysed by gas chromatography with an electron capture detector. Detection limits of 0.001 $\mu\text{g}/\text{L}$ for aldrin and 0.002 $\mu\text{g}/\text{L}$ for dieldrin can be achieved for tap-water and river samples.¹⁸

Water treatment technologies such as granular activated carbon and reverse osmosis can reduce levels of aldrin and dieldrin in drinking water supplies.^{5,19}

Health Effects

Aldrin and dieldrin are readily absorbed by oral, inhalation and dermal routes. Absorption through the intact skin was about 7 to 8% of the applied dose in a human volunteer study.²⁰ Human volunteers exposed to an aldrin concentration of 1.31 $\mu\text{g}/\text{m}^3$ absorbed and retained about 20 to 50% of the inhaled aldrin vapours.^{21,22} Aldrin and dieldrin tend to accumulate in adipose tissue. When aldrin and dieldrin are mobilized from adipose tissue, their levels in blood increase, resulting in toxic manifestations. Mobilization has been found to be triggered by weight loss in dogs.²³

Following absorption, aldrin is quickly metabolized to dieldrin. The major metabolite resulting from dieldrin metabolism in the liver, 9-hydroxy dieldrin, is excreted via the bile into the faeces. Small amounts of other metabolites, such as trans-6,7-hydroxy dieldrin, dicarboxylic acids and bridged pentachloroketone, are excreted by laboratory animals.²⁰ The biological half-life of dieldrin in humans is about 266 days.²⁴

Aldrin and dieldrin are highly toxic to humans, affecting the central nervous system and the liver. Signs and symptoms related to ingestion of or dermal contact with toxic doses of aldrin and dieldrin include headache, dizziness, nausea, general malaise and vomiting, followed by muscle twitchings, myoclonic jerks and convulsions. These effects generally appear between 20 minutes and 24 hours after absorption of aldrin and dieldrin. Cerebral anoxaemia may lead to death.^{20,25–28} The lethal oral dose of aldrin or dieldrin in humans is estimated to be about 5 g.^{24,29}

Male volunteers were exposed to dieldrin doses of 0 to 3 $\mu\text{g}/\text{kg}$ bw per day for 18 months. The results showed no health effects and a proportional relationship between the daily intake of dieldrin and levels in blood and adipose tissue.²⁰ An epidemiological mortality study was carried out on a cohort of 1155 workers exposed to aldrin, dieldrin and endrin in a manufacturing plant. The results, consisting of almost 25 000 person-years of data, showed that there was no specific cancer risk associated with employment at this plant.³⁰

Oral LD₅₀ values for aldrin and dieldrin in the mouse and rat range from 50 to 70 mg/kg bw.²⁰ Laboratory studies on mice exposed orally to dieldrin showed an immunosuppressive effect, probably linked to hepatotoxicity.²³ Teratogenic effects of dieldrin were not reported in either humans or laboratory animals. Foetotoxicity in rodents occurred at levels of dosing causing maternal toxicity. Dieldrin crosses the placenta of humans and laboratory animals and might accumulate in the foetus.²³ Decreased fertility and pre-weaning pup mortality were reported in rats fed dieldrin. An evaluation of the genotoxic potential of aldrin and dieldrin indicated that they are not mutagenic.²³

Chronic feeding with aldrin or dieldrin led to hepatocellular carcinoma in mice and to non-neoplastic histological changes and increased liver to body weight ratios in rats, dogs and hamsters.²³ On the basis of a number of long-term carcinogenicity studies in rats fed aldrin or dieldrin, the no-observed-adverse-effect level (NOAEL) for both aldrin and dieldrin has been found generally to be 0.5 mg/kg diet (approximately equivalent to 0.025 mg/kg bw).²⁰ At higher concentrations, hepatomegaly and dose-related histological changes in the liver are commonly reported.

Classification and Assessment

The International Agency for Research on Cancer has determined that aldrin and dieldrin are not classifiable as to their carcinogenicity in humans,³¹ owing to inadequate evidence of carcinogenicity in humans and limited evidence of carcinogenicity in experimental animals.

For compounds that are not classifiable as to their carcinogenicity in humans, the acceptable daily intake

(ADI) is derived on the basis of division of a NOAEL in humans or in experimental animals by an appropriate uncertainty factor. The ADI for aldrin and dieldrin has been derived as follows:

$$\text{ADI} = \frac{0.025 \text{ mg/kg bw per day}}{250} = 0.0001 \text{ mg/kg bw per day}$$

where:

- 0.025 mg/kg bw per day is the NOAEL in rats for hepatotoxic effects²⁰
- 250 is the uncertainty factor, to take into account concern for the oncogenicity observed in the mouse.²⁰

Rationale

The maximum acceptable concentration (MAC) for aldrin and dieldrin in drinking water is derived from the ADI as follows:

$$\text{MAC} = \frac{0.0001 \text{ mg/kg bw per day} \times 70 \text{ kg bw} \times 0.20}{2 \text{ L/d}} = 0.0007 \text{ mg/L}$$

where:

- 0.0001 mg/kg bw per day is the ADI, as derived above
- 70 kg bw is the average body weight of an adult
- 0.20 is the proportion of daily intake of aldrin and dieldrin allocated to drinking water (available data are insufficient to estimate this value)
- 2 L/d is the average daily consumption of drinking water for an adult, as used by the Department of National Health and Welfare in 1978.*

References

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*Health Canada is now using 1.5 L as the average daily consumption of drinking water for an adult. Using this value in the calculations, the MAC becomes 0.9 µg/L.

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