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16. ABSTRACT

This report summarizes and evaluates information relevant to a preliminary interim assessment of adverse health effects associated with specific chemicals or compounds. The Office of Emergency and Remedial Response (Superfund) uses these documents in preparing cost-benefit analyses under Executive Order 12991 for decision-making under CERCLA. All estimates of acceptable intakes and carcinogenic potency presented in this document should be considered as preliminary and reflect limited resources allocated to this project. The intent in these assessments is to suggest acceptable exposure levels whenever sufficient data are available. The interim values presented reflect the relative degree of hazard associated with exposure or risk to the chemical(s) addressed. Whenever possible, two categories of values have been estimated for systemic toxicants (toxicants for which cancer is not the endpoint of concern). The first, RfD_S or subchronic reference dose, is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs during a limited time interval. The RfD is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs for a significant portion of the lifespan. For compounds for which there is sufficient evidence of carcinogenicity, q_1^*s have been computed, if appropriate, based on oral and inhalation data if available.

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HEALTH EFFECTS ASSESSMENT FOR DIBENZOFURAN

ENVIRONMENTAL CRITERIA AND ASSESSMENT OFFICE
OFFICE OF HEALTH AND ENVIRONMENTAL ASSESSMENT
OFFICE OF RESEARCH AND DEVELOPMENT
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PREFACE

This report summarizes and evaluates information relevant to a preliminary interim assessment of adverse health effects associated with dibenzofuran. All estimates of acceptable intakes and carcinogenic potency presented in this document should be considered as preliminary and reflect limited resources allocated to this project. Pertinent toxicologic and environmental data were located through on-line literature searches of the TOXLINE and the CHEMFATE/DATALOG data bases. The basic literature searched supporting this document is current up to May, 1986. Secondary sources of information have also been relied upon in the preparation of this report and represent large-scale health assessment efforts that entail extensive peer and Agency review. The following Office of Health and Environmental Assessment (OHEA) sources have been extensively utilized:

U.S. EPA. 1983. Health and Environmental Effects Profile for Tetra-, Penta- and Hexachlorodibenzofurans. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste, Washington, DC.

U.S. EPA. 1985. Health and Environmental Effects Profile for Brominated Dibenzofuran. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response, Washington, DC.

U.S. EPA. 1986c. Health Assessment Document for Polychlorinated Dibenzofurans. Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. EPA 600/8-86/018A. NTIS PB86-221256/AS. External Review Draft.

ABSTRACT

There are no pertinent subchronic, chronic, carcinogenicity, or reproductive toxicity data on dibenzofuran. Dibenzofuran was not found to be mutagenic in reverse mutation assays and a 2-year bioassay of a related compound (dibenzo-p-dioxin) did not result in treatment-related increases in tumor incidence. It is recommended that a pharmacokinetic profile of oral debenzofuran be developed and that subchronic and reproductive toxicity testing be initiated. Also, further testing for mutagenicity and clastogenicity in mammalian systems and short-term in vivo testing for carcinogenic potential should be performed.

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LIST OF ABBREVIATIONS

DWEL	Drinking water equivalent level
HA	Health advisory
K_{oc}	Soil sorption coefficient
ppm	Parts per million
RfD	Reference Dose
SNARL	Suggested-no-adverse-response level
UV	Ultraviolet

1. ENVIRONMENTAL CHEMISTRY AND FATE

The relevant physical and chemical properties and environmental fate of dibenzofuran are presented in Table 1-1.

In the atmosphere, dibenzofuran has the potential to undergo direct photolysis, which is due to UV absorption >290 nm, and it may react with photochemically generated hydroxyl radicals (estimated vapor phase ty_2 is ~7 hours). (U.S. EPA, 1982, 1986a). Since part of the dibenzofuran emitted to the atmosphere may exist in the particulate form, this part may, as a constituent of coke dust, grate ash, fly ash, flame soots, etc., transport over long distances because the particulate sorbed compound is less likely to undergo chemical reaction than the compound in the vapor state (U.S. EPA, 1982; Ligocki et al., 1985).

In water and soil systems, microbial degradation appears to be the dominant degradation mechanism of polynuclear aromatic compounds (Sims and Overcash, 1983). In aqueous systems, dibenzofuran should bioaccumulate moderately in aquatic organisms and adsorb to suspended solids and sediments (Lu et al., 1978, U.S. EPA, 1982; Bjoerseth et al., 1979). In the sorbed state the compound is likely to be persistent (Bjoerseth et al., 1979). Estimated K_{oc} values ranging from 1230-9940 indicate that dibenzofuran should strongly adsorb to soil; however, dibenzofuran has been detected in a shallow aquifer under a creosote facility (Bedient et al., 1984).

Pertinent data regarding human exposure to dibenzofuran could not be located in the available literature.

TABLE 1-1

Physical and Chemical Properties and Half-Lives for Dibenzofuran

Property	Value	Reference
CAS number	132-64-9	
Chemical class:	Polynuclear aromatic	
Molecular weight:	168.20	
Melting point:	82.8-83°C	U.S. EPA, 1982
Boiling Point:	276°C (760 mm Hg) 287°C (760 mm Hg)	U.S. EPA, 1982
Vapor pressure:	~1.75x10^{-2} mm Hg	U.S. EPA, 1982
Water solubility:	~3 mg/ℓ at 25°C	U.S. EPA, 1982
Log octanol/water partition coefficient:	4.12 3.18	Hansch and Leo, 1985 Lu et al., 1978
Bioconcentration factor:	83, alga (Oedogonium) 2860, snail (Physa) 947, fish (Gambusia)	Lu et al., 1978
Soil adsorption coefficient (K_{OC}):	1230-4150 (estimated) 9940 (estimated)	Lyman et al., 1982 Sabljic, 1984
Half-lives in Air:	<1 day (estimated) (vapor phase)	U.S. EPA, 1986a
Water:	years (adsorbed to sediments)	Bjoerseth et al., 1979

2. ABSORPTION FACTORS IN HUMANS AND EXPERIMENTAL ANIMALS

Pertinent data regarding the absorption of dibenzofuran after oral or inhalation exposure could not be located in the available literature.

3. TOXICITY IN HUMANS AND EXPERIMENTAL ANIMALS

The U.S. EPA (1986c) reviewed several studies that indicated that other than a few subchronic toxicity studies of PCDFs in rats, chicks and mice, there are no studies of chronic toxicity. Two of three monkeys died on a diet containing 5 μg/kg (food) administered over 6 months. It is highly probable that continuous exposure at low levels causes cumulative toxic effects especially in view of the lesions observed in monkeys at an acute dose that was <4% of the LD_{50} (McNulty et al., 1981). It appears likely that PCDFs make a substantial contribution to the toxicity of commercial PCBs and polychlorophenols.

Since the residence time of 2,3,7,8-T_4CDF is higher in guinea pigs than other animals tested and guinea pigs are most sensitive to the toxic effects of 2,3,7,8-T_4CDF, Ioannou et al. (1983) studied bioaccumulation and toxicity of 2,3,7,8-T_4CDF in male Hartley guinea pigs. Five- to six-week-old animals weighing 360-430 g and 16- to 18-week-old adult animals weighing ~800 g were treated by gavage with 2,3,7,8-T_4CDF dissolved in a mixture of emulphar:ethanol (1:1) diluted 8-fold in distilled water. Single oral treatments of 10 or 15 μg/kg bw 2,3,7,8-T_4CDF to adult animals resulted in immediate drastic weight loss and death within 2 4 weeks. In young animals neither single treatments of 4 μg/kg bw nor multiple treatments of 1 μg/kg bw/week for 4 weeks could produce any observable adverse effects by day 36, when the animals were sacrificed. However, multiple treatment of young animals with low doses totaling cumulative doses of between 4 and 12 μg/kg resulted in death of 75% of the animals.

The administration of PCDFs induces a number of hepatic enzymes, especially those using 3-methylcholanthrene as inducer (Poland and Glover, 1974).

Structure/activity relationships of PCDFs were studied for the induction of AHH and δ-aminolevulinic acid synthetase activities in chick embryo. The most acutely toxic isomers are also the most potent inducers of AHH activity. The ED_{50} of 2,3,7,8-T_4CDF on AHH induction in the rat is 0.5 μg/kg/day for 3 days (Poland et al., 1976).

Luster et al. (1979) exposed adult Hartley guinea pigs through injestion to 0.05-1 μg/kg/week of 2,3,7,8-T_4CDF in corn oil for 6 weeks to study its effects on immune function. Thymus-to-body weight ratios were slightly reduced in the 0.5 and 1.0 μg dosage groups. Cell-mediated immune functions were depressed for the 0.5 μg dosage group as indicated by lymphocyte blastogenesis, delayed hypersensitivity reactions and production of macrophage inhibitor factors.

Teratogenicity

Teratogenic activities of 2,3,7,8-T_4CDF have recently been reported by Weber et al. (1984) in mice. Pregnant C57Bl/6N mice were given either a single dose by gavage on gestation day 10 with 250, 500 and 1000 μg of 2,3,7,8-T_4CDF/kg bw or daily doses on gestation days 10 through 13 with 10, 30, 50 and 100 μg of 2,3,7,8-T_4CDF/kg bw. The chemical contained >98% pure 2,3,7,8-T_4CDF. The primary impurities were found to be P_5CDFs. Each animal was given a total of 10 mℓ/kg bw at each treatment. Though these doses were below toxic level to the dams, the animals receiving daily doses on four gestation days had mild changes in liver cells characterized by proliferation of the smooth endoplasmic reticulum.

In this experiment, the authors found that single administration on gestation day 10 with 250, 500 and 1000 μg/kg bw resulted in significant ($p<0.05$) increases in fetal mortality. In single as well as multiple dosed groups there were significant ($p<0.01$) dose-related increases in both isolated cleft palates, and hydronephrosis in the fetuses were observed.

In utero four exposures of 10 μg/kg/day resulted in significant ($p<0.01$) increases in hydronephrosis of the kidney. This was the lowest adverse effect level observed in this experiment. The right kidney of the fetuses seems to be affected more severely than the left kidney.

Teratogenic effects of 2,3,7,8-T_4CDF have also been reported recently in BXD recombinant inbred strains of mice by Hassoun et al. (1984). No teratogenic effects in humans have been observed.

Adverse Human Health Effects

The available information on adverse health effects in humans comes mostly from Japan where in 1968 many people ate rice oil (Yusho) that was later found to be contaminated with PCBs and PCDFs. In 1968, some 1200 Japanese people consumed high levels of PCDFs in rice oil (Yusho) contaminated with 800-1000 ppm of a Japanese PCB formulation, Kanechlor 400, which had leaked from a heat exchanger (Nagayama et al., 1975). This caused a severe toxic reaction known as Yusho episode. Symptoms of Yusho disease are described in Table 3-1. Several months before this incident, half a million chicks were killed by a crude bran oil from the same origin as Yusho oil but the warning went unheeded (Kohanawa et al., 1969). The composition of Kanechlor 400 resembled that of Aroclor 1248. However, reports of the total PCDF content varied greatly from investigator to investigator. Some general symptoms included the following: retarded growth, abnormal lipid metabolism, liver disturbances, acneform eruption, skin pigmentation and cutaneomucosal lesions. Liver and adipose tissue of a few patients examined 4 years after the contamination showed retention of PCBs at ppb-ppm levels and PCDFs at ppb levels. The 2,3,4,7,8-PCDF was found to be retained by the liver at higher concentrations than other isomers (Kuratsune et al., 1976; Kimbrough, 1974).

TABLE 3-1

Symptoms of Yusho Disease*

Symptoms	Males (%)	Females (%)
Blackening of nails	83.1	75.0
Black spots in all pores	64.0	56.0
Excessive sweating in palms	50.0	55.0
Acnelike skin eruptions	87.6	82.0
Red spots on limbs	20.2	16.0
Itching	42.7	52.0
Change in skin color	75.3	72.0
Swelling of hands and feet	20.2	41.0
Stiffened soles in feet and palms of hands	24.7	29.0
Pigmentation of mucous membranes	56.2	47.0
Increased eye discharge	88.8	83.0
Hyperemia of mucous membranes in eyes	70.8	71.0
Transient visual disturbance	56.2	55.0
Jaundice	11.2	11.0
Swelling of upper eyelids	71.9	74.0
Sense of weakness	58.4	52.0
Numbness of hands and feet	32.6	39.0
Fever	16.9	19.0
Hearing difficulty	18.0	19.0
Spasms of hands and feet	7.9	8.0
Headaches	30.3	39.0
Vomiting	23.6	28.0
Diarrhea	19.1	17.0

*Source: Kuratsune et al., 1972; U.S. EPA, 1986c

Mutagenicity

Dibenzofuran, 2,8-D_2CDF, 3,6,-D_2CDF, 2,3,7,8-T_4CDF and OCDF are not mutagenic in any of the standard Salmonella strains (Schoeny, 1982). A mixture of PCDFs containing 3-6 chlorines enhanced sister-chromatid exchanges in Chinese hamster lymphocyte cultures at 0.1-1.0 mg/ℓ (Inoue et al., 1979).

4. CARCINOGENICITY

Animal

Close structural similarity of PCDFs to PCDDs, especially 2,3,7,8-T_4CDD which is a proven animal carcinogen, raises concern as to the potential carcinogenicity of 2,3,7,8-T_4CDF. However, no animal carcinogenicity bioassay data on PCDFs are currently available in the literature.

The NCI (1979) conducted 2-year dibenzo-p-dioxin feeding studies using rats and mice. This compound is structurally similar to dibenzofuran. Rats and mice of both sexes received 0, 5000, or 10,000 ppm; survivors were sacrificed after 110-117 weeks (rats) or 91-97 weeks (mice) for histological analysis. Administration of the high concentration accelerated mortality rate in female rats and mice, and mean body weight gains were generally lower, which was due to treatment. Nonneoplastic hepatotoxic effects (primarily fatty metamorphosis and necrosis) were concentration-related in rats and female mice. There were also slight treatment-related increases in renal lesions, including renal tubular dilutations (female rats) and interstitial inflammation (female mice).

The U.S. EPA (1986c) noted that the biological activity of various chlorinated dibenzofurans varies greatly, so that risk assessment by analogy to any of these more widely studied compounds would not be recommended.

Human

Amano et al. (1984) recently completed a 16-year cohort mortality study of 1086 Yusho victims in Japan. The 581 males and 505 females sustained a total of 70 deaths (42 males vs. 45.81 expected and 28 females vs. 31.3 expected). These data are based upon Japanese national death rates

over age 40 through October 31, 1983. In this population, for persons over 40 years of age overall cancer mortality was greater than expected in men but no difference in women. In male Yusho victims 19 cancer deaths occurred vs. 11.50 expected and in female Yusho victims 7 cancer deaths occurred vs. 7.20 expected. However, by organ site, the risk of liver cancer was consistently found to be high in both men and women during the entire 16-year observation period. Even after a 9-year latent period, the risk of liver cancer in males was significant (observed = 5, expected = 0.75, $p<0.01$).

As a result of an in-depth review of this study, it has been suggested by CAG (U.S. EPA, 1986c) that a case could be made that PCBs as the components of the PCDFs that caused the Yusho incident in Japan may also have been responsible for the statistically significant liver cancers seen in these victims.

Dibenzofuran should be classified as an EPA Group D (U.S. EPA, 1986b) or an IARC Group 3 chemical. These categories are for compounds in which evidence of animal carcinogenicity is inadequate or limited and there are no human data.

5. REGULATORY STANDARDS AND CRITERIA

Pertinent guidelines and standards, including EPA ambient air quality criteria, drinking water standards, EPA or FAO/WHO ADIs (currently RfDs), EPA or FDA tolerances for new agricultural commodities or foods, SNARLS, HAs, DWELs, and ACGIH, NIOSH or OSHA occupational exposure limits could not be located in the available literature.

The U.S. EPA (1975) determined an odor threshold of 120 µg/ℓ for dibenzofuran. The U.S. EPA (1982) recommended that this level be considered an ambient water criteria, based exclusively on organoleptic properties.

6. RECOMMENDATIONS

Physical properties (vapor pressure at 25°C of 1.75×10^{-2} mm Hg) indicate that dibenzofuran may not constitute an inhalational health hazard. Dibenzofuran is likely to persist in soil sediments and may bioaccumulate in aquatic organisms. Oral exposure seems to be more environmentally relevant than inhalation exposure. Because of the sparsity of toxicological information, it is recommended that a complete pharmacokinetic profile of oral dibenzofuran be developed in experimental animals. Appropriate subchronic and reproductive toxicity testing using dietary dibenzofuran should then be initiated. Although dibenzofuran was negative for mutagenicity in bacterial reverse mutation assays (Schoeny, 1982) and the related dibenzo-p-dioxin was not carcinogenic in 2-year feeding studies (NCI, 1979), mutagenicity and clastogenicity assays in mammalian systems and short-term *in vivo* cancer biossays should be performed.

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