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Dibenz[a,h]anthracene (CASRN 53-70-3)

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Reference Dose for Chronic Oral Exposure (RfD)

0456

Dibenz[a,h]anthracene; CASRN 53-70-3

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Dibenz[a,h]anthracene

File First On-Line 12/01/1990

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	no data	
Carcinogenicity Assessment (II.)	on-line	03/01/1994

I. Chronic Health Hazard Assessments for Noncarcinogenic Effects

I.A. Reference Dose for Chronic Oral Exposure (RfD)

Substance Name -- Dibenz[a,h]anthracene
CASRN -- 53-70-3

Not available at this time.

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Chronic Health Hazard for Non-Carcinogenic Effects

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Carcinogenicity Assessment for Lifetime Exposure

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Quantitative Estimate of

__I.B. Reference Concentration for Chronic Inhalation Exposure (RfC)

Substance Name -- Dibenz[a,h]anthracene
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Not available at this time.

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__II. Carcinogenicity Assessment for Lifetime Exposure

Substance Name -- Dibenz[a,h]anthracene
CASRN -- 53-70-3

Last Revised -- 03/01/1994

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

__II.A. Evidence for Human Carcinogenicity**___II.A.1. Weight-of-Evidence Characterization**

Classification -- B2; probable human carcinogen

Basis -- Based on no human data and sufficient data from animal bioassays. Dibenz[a,h]anthracene produced carcinomas in mice following oral or dermal exposure and injection site tumors in several species following subcutaneous or intramuscular administration. Dibenz[a,h]anthracene has induced DNA damage and gene mutations in bacteria as well as gene mutations and transformation in several types of mammalian cell cultures.

___II.A.2. Human Carcinogenicity Data

None. Although there are no human data that specifically link exposure to dibenz[a,h]anthracene with human cancers, dibenz[a]anthracene is a component of mixtures that have been associated with human cancer. These include coal tar, soots, coke oven emissions and cigarette smoke (U.S. EPA, 1984, 1990; IARC, 1984).

___II.A.3. Animal Carcinogenicity Data

Sufficient. Dibenz[a,h]anthracene has been shown to be carcinogenic when administered to mice by the oral route (Snell and Stewart, 1962, 1963). Instead of drinking water DBA/2 mice (21/sex) were given a water-olive oil emulsion containing 0.2 mg/mL dibenz[a,h]anthracene ad libitum. Average exposure was estimated to be 0.85 mg/day for males and 0.76 mg/day for females. The control groups (25 male and 10 female) received the water-olive oil emulsion in place of water. The mice did not tolerate the olive oil vehicle well and all 4 groups lost weight

after a few weeks exposure and eventually became emaciated and dehydrated. Animals that died spontaneously or that became moribund were examined for tumors. The duration of the experiment was 279 and 237 days for males and females, respectively, in the dosed groups and 351 and 226 days for male and female controls. Mice developed pulmonary adenomas (treated males, 14/14; control males 1/23; treated females, 13/13; control females, 0/6), pulmonary carcinomas (treated males, 14/14; control males, 0/23; treated females, 10/13; control females, 0/6), mammary carcinoma (treated females, 12/13; control females, 0/6) and hemangioendothelioma (treated males, 10/14; control males, 0/23; treated females, 6/13; control females, 0/6). No statistical analyses appear to have been performed.

Mammary carcinomas were observed in two strains of female mice following gavage with dibenz[a,h]anthracene (Biancifiore and Caschera, 1962; Berenblum and Haran, 1955). Biancifiore and Caschera (1962) observed mammary carcinomas when female Balb/c (1/20) and pseudo-pregnant female (obtained by mating virgin females with vasectomized males) Balb/c (13/24) mice were treated for 15 weeks with a twice-weekly gavage containing 0.5% dibenz[a,h]anthracene (total dose was 15 mg/animal). Mammary carcinomas occurred in 2/30 pseudo-pregnant females not dosed with dibenz[a,h]anthracene. Previous studies indicated that mammary carcinomas did not occur in virgin Balb/c females (Biancifiore et al., 1959). A single 1.5-mg dose of dibenz[a,h]anthracene in polyethylene glycol [average molecular weight (a.m.u.) 400] (PEG-400) produced forestomach papillomas in 2/42 male Swiss mice after 30 weeks. In this short-term study no mice developed tumors when treated with PEG alone (1 time/week) for 30 weeks (0/20) (Berenblum and Haran, 1955).

Dibenz[a,h]anthracene has produced positive results in mouse skin painting assays for complete carcinogenicity. Swiss mice developed carcinomas following dermal exposure to dibenz[a,h]anthracene at concentrations of 0.001% or greater (Wynder and Hoffman, 1959; Van Duuren et al., 1967). Numerous studies that demonstrate complete carcinogenic activity and initiating activity are summarized in IARC (1973) and U.S. EPA (1990).

Subcutaneous injection of dibenz[a,h]anthracene induced sarcomas at the site of injection in several animal species. Groups (>19) of C3H mice received single subcutaneous injections of dibenz[a,h]anthracene in tricaprilyn at doses ranging from 0.0019-8 mg (approximately 0.09-360 mg/kg). No controls appear to have been used in this experiment (Bryan and Shimkin, 1943). Tumor latency appeared to decrease and the incidence of injection site sarcomas appeared to increase with dose (>76% at doses >0.06 mg or 2.8 mg/kg). A single subcutaneous injection of 2.4, 4.7, 9.3, 18.7, 37.5, or 75 ug dibenz[a,h]anthracene into groups of 100 NMRI mice was reported to produce a dose-related increase in tumor incidence (37/100, 39/100, 44/100, 56/100, 65/100, and 69/100, respectively) by the 114th week after injection (Pfeiffer, 1977). No concurrent controls were reported; however, a spontaneous tumor rate for NMRI mice was previously reported to be 0-2% (Pfeiffer, 1973). The development of fibrosarcomas from a single subcutaneous injection of 150 ug dibenz[a,h]anthracene was shown to be higher in AHH+ strains of mice than in AHH- strains.

Lubet et al. (1983) found that subcutaneous injections of dibenz[a,h]anthracene were associated with fibrosarcoma development in mice, but only for some strains. Four strains of mice used included two, C3H/HeJ and C57B1/6J, that respond to 3-methylcholanthrene treatment with increased levels and types of hepatic enzymes, including AHH. Two strains, AKR/J and DBA/2J were nonresponders. Groups of 30 animals were injected with a single dose of 150 mg dibenz[a,h]anthracene in 0.05 mL trioctanoin and observed for 9 months. A control group for each strain, consisting of 10 animals each, received a subcutaneous injection of 0.05 mL trioctanoin alone. The tumor incidence in the treated animals varied between 0 and 80%, depending on the strain. Tumor incidences were higher in the C3H and C57B1 mice but not in AKR or DBA mice. Likewise, the average latency period (in days) for fibrosarcoma development varied with the strain and tended to be inversely correlated with the tumor incidence rate. Numerous earlier studies that demonstrate the carcinogenicity of parenterally injected dibenz[a,h]anthracene in a variety of species are summarized in IARC (1973) and U.S. EPA (1990).

II.A.4. Supporting Data for Carcinogenicity

Dibenz[a,h]anthracene has produced positive results in bacterial DNA damage and mutagenicity assays and in mammalian cell DNA damage, mutagenicity and cell transformation assays. In bacterial DNA damage assays, positive results were obtained in *Escherichia coli* and *Bacillus subtilis* at exposure levels of 12-50 ug/well. Dibenz[a,h]anthracene tested positive for reverse mutation in *Salmonella typhimurium* strains TA100 and TA98 (3-5 ug/plate) and positive for forward mutation in strain TM677 (21 ug/mL) (McCann et al., 1975; Andrews et al., 1978; Baker et al., 1980; Hermann, 1981; Kaden et al., 1979). In mammalian cell DNA damage assays, positive results were obtained in human foreskin epithelial cells not activated with mixed-function oxidase (MFO) inducers (1-100 ug/mL) and in HeLa cells (28 ng/mL) activated with 3-methylcholanthrene (Lake et al., 1978; Martin et al., 1978). When Syrian hamster embryo cells and rat hepatocytes not activated with MFO inducers were exposed to 20-30 ug/mL the results were not positive (Casto, 1979; Probst et al., 1981). Dibenz[a,h]anthracene induced forward mutations in Chinese hamster embryo cells exposed to concentrations of 1 ug/mL or greater (Huberman and Sachs, 1976; Krahn and Heidelberger, 1977; Huberman, 1978). It transformed several types of mammalian cells exposed to concentrations of 10 ug/mL or greater; these cell types included: Syrian hamster embryo cells, mouse C3H10T 1/2 cells and mouse prostate C3H cells (DiPaolo et al., 1969; Chen and Heidelberger, 1969; Pienta et al., 1977; Casto et al., 1977; Casto, 1979; Reznikoff et al., 1973; Lubet et al., 1983).

Current theories on mechanisms of metabolic activation of polycyclic aromatic hydrocarbons are consistent with a carcinogenic potential for dibenz[a,h]anthracene. Dibenz[a,h]anthracene has a "bay-region" structure (Jerina et al., 1978). It is metabolized by mixed-function oxidases to dihydrodiols that are mutagenic in bacteria and tumorigenic in mouse skin painting assays and when injected into newborn mice (Wood et al., 1978; Nordqvist et al., 1979; Slaga et al., 1980; Buening et al., 1979).

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II.B. Quantitative Estimate of Carcinogenic Risk from Oral Exposure

Not available.

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II.C. Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

Not available.

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II.D. EPA Documentation, Review, and Contacts (Carcinogenicity Assessment)

II.D.1. EPA Documentation

Source Document -- U.S. EPA, 1984, 1990

The 1990 Drinking Water Criteria Document for Polycyclic Aromatic Hydrocarbons has received Agency and external review.

II.D.2. EPA Review (Carcinogenicity Assessment)

Agency Work Group Review -- 02/07/1990, 08/05/1993, 09/21/1993, 02/02/1994

Verification Date -- 02/07/1990

II.D.3. EPA Contacts (Carcinogenicity Assessment)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (301)345-2870 (phone), (301)345-2876 (FAX) or Hotline.IRIS@epamail.epa.gov (internet address).

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III. [reserved]

IV. [reserved]

V. [reserved]

VI. Bibliography

Substance Name -- Dibenz[a,h]anthracene

CASRN -- 53-70-3

Last Revised -- 12/01/1990

VI.A. Oral RfD References

None

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VI.B. Inhalation RfC References

None

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VI.C. Carcinogenicity Assessment References

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dibenzo[a,h]anthracene and its dihydrodiols to bacterial mutagens. Cancer Res. 38(7): 1967-1973.

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_VII. Revision History

Substance Name -- Dibenz[a,h]anthracene
CASRN -- 53-70-3

Date	Section	Description
12/01/1990	II.	Carcinogen assessment on-line
12/01/1990	VI.	Bibliography on-line
01/01/1992	IV.	Regulatory Action section on-line
09/01/1993	II.	Carcinogenicity assessment noted as pending change
09/01/1993	II.D.2.	Work group review date added
11/01/1993	II.D.2.	Work group review date added
03/01/1994	II.	Pending change note removed; no change
03/01/1994	II.D.2.	Work group review date added
08/01/1995	II.D.2.	EPA's RfD/RfC and CRAVE workgroups were discontinued in May, 1995. Chemical substance reviews that were not completed by September 1995 were taken out of IRIS review. The IRIS Pilot Program replaced the workgroup functions beginning in September, 1995.
04/01/1997	III., IV., V.	Drinking Water Health Advisories, EPA Regulatory Actions, and Supplementary Data were removed from IRIS on or before April 1997. IRIS users were directed to the appropriate EPA Program Offices for this information.

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_VIII. Synonyms

Substance Name -- Dibenz[a,h]anthracene
CASRN -- 53-70-3
Last Revised -- 12/01/1990

53-70-3

Dibenz(a,h)anthracene

DB(a,h)A

DBA

dibenz(a,h)anthracene

DIBENZO(a,h)ANTHRACENE

HSDB 5097

NSC 22433

RCRA WASTE NUMBER U063

1,2,5,6-DIBENZANTHRACEEN [Dutch]

1,2,5,6-dibenzanthracene

1,2:5,6-BENZANTHRACENE

1,2:5,6-DIBENZ(a)ANTHRACENE
1,2:5,6-Dibenzanthracene
1,2:5,6-DIBENZOANTHRACENE

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